Diagnosis and Clinical examination

Dr Hadeel Mohammed Abbood

- diagnosis is a process of investigations that helps identifying the exact condition or disease, and the cause. Diagnosis is the first step in the way of effective treatment.
- However, Clinicians might wrongly diagnose a condition. Therefore, in case of not sure about the diagnosis, a differential diagnosis might be given.
- Which is providing a list of the conditions and diseases ordered from the most to the least likely

Medical history:

- It is important to document the medical history of the patient. Most importantly, is to explain to the patient why this step is important as most of the patients would omit this information.
- Medical history has more than one important aspect. A current medical history must be taken before starting the clinical examination. The minimum that must be known is:
  - whether the patient is under the care of a physician,
  - is taking any medication
  - or has any medical condition that may affect the periodontal diagnosis or treatment.
- This permits the identification of patients who may:

1. • Need special care and management before receiving periodontal treatment.
2. • Pose an infection risk to the dental professional.
3. • Have conditions that provoke periodontal diseases.

Dental history:

- A complete history of the dental treatment for the patient is really important in diagnosis, management and identifying the causes of the condition the patient is complaining of. Such as referred pain, overhang, occlusion problem…etc.

Chief complaint:

Is a clear and concise statement that describes why the patient is coming to the clinic. It should be recorded using the patient's own words. This will help in preventing misinterpretation of the final diagnosis and stay within the patients need in treatment planning.

- We should take into account, the chief complaint should be recorded in each visit, even in case of follow-up. This will also help in following the changes in the main complaint and whether there is any improvement or not.
- Chronic inflammatory periodontal disease is usually painless, most of the patients do not have a periodontitis-related chief complaint. The gradual progression of the disease may make the patient accustomed to the symptoms, so they do not notice any signs and symptoms.
- In chronic gingivitis, the chief complaint of patient is usually bleeding during tooth brushing. Patients with periodontitis may complain of tooth migration, loose teeth, diastemas between teeth, or sometimes swelling and abscess.
- If there is no complaint, the reason for attendance should be noted. If there are other dental complaints these should also be noted.
- History of complaint:
- the onset, duration, severity and any triggers of the presenting complaint should be noted. In addition, family history of the periodontal problems or early tooth loss should be noted.

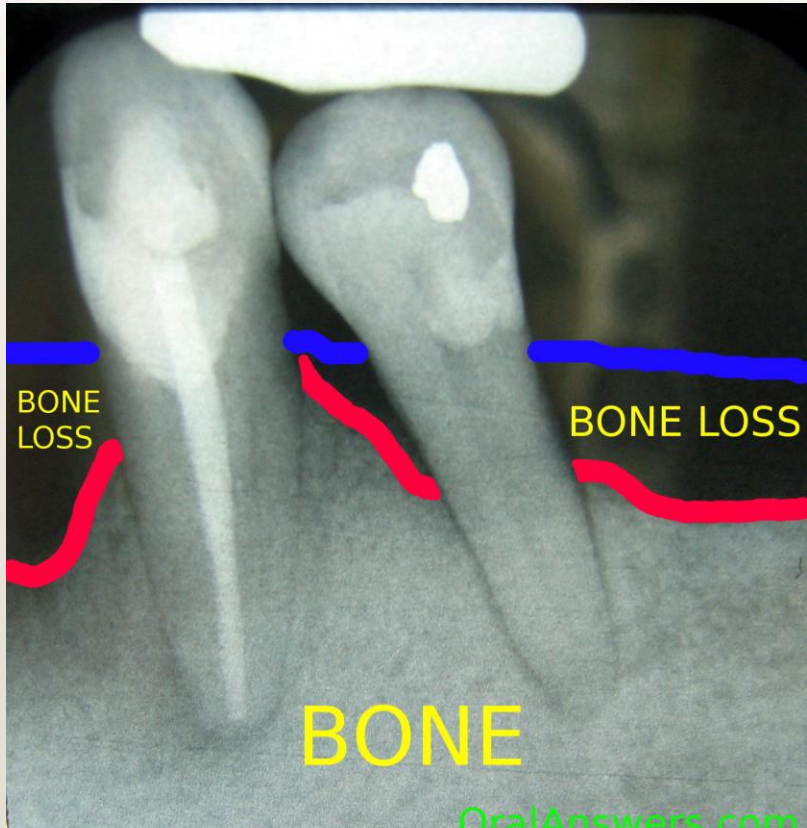
Radiographic examination

- Radiographs are used to provide information about the hard tissues and are recommended only when such data provides positive added value to that already held.
- The commonly used types are the periapical, bitewing and panoramic radiography. All of them can provide important information that aids in the diagnosis of periodontal diseases, but all of them have limitations:

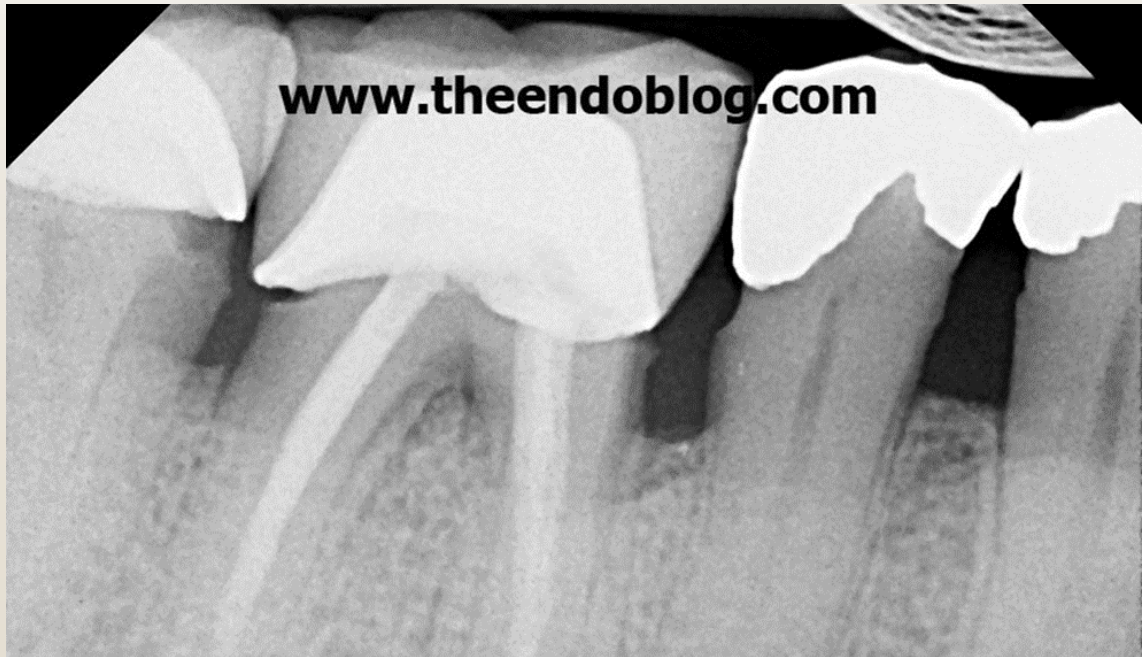
1. The presentation of three-dimensional object in two dimensions.
2. Important morphologic or pathologic aspects of the alveolar bone maybe masked by the overlapping of the teeth and other anatomic structures.
3. Only the interproximal alveolar bone levels can be assessed with some levels of certainty, because of the high density of the teeth that can cover the visualization of bone overlying them.
4. The detection and quantitative assessment of 2-wall and 3-wall defects remains a challenge even in interproximal areas.
5. Bone resorption detected only when there is a mineral loss of 30-50%.
6. Misdirection of the central beam of x-ray as well as the exposure and the processing errors further limit accuracy.

From radiograph, three aspects of bone loss can be considered:

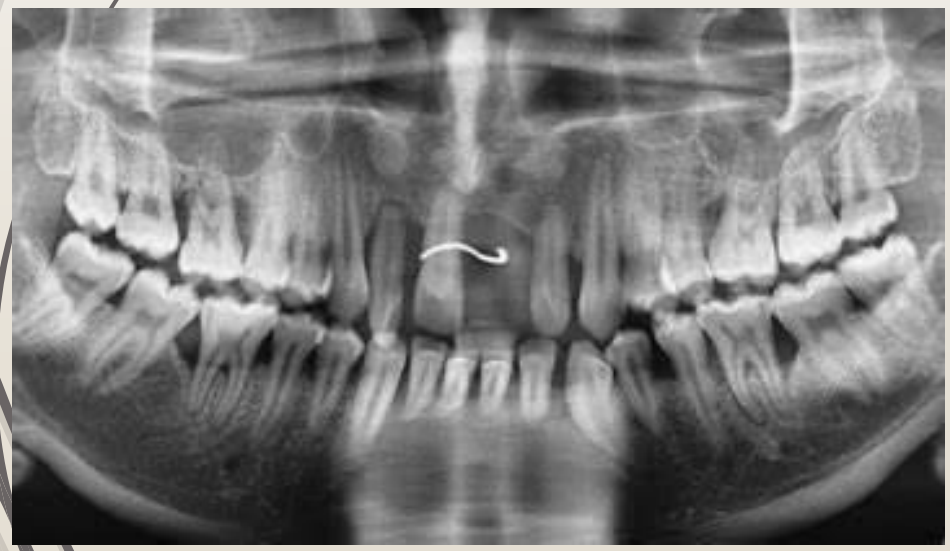
- 1- Severity of bone loss on an individual tooth: it can be measured by the percentage of the root that is no longer covered by a bone, by calculating the distance from the CEJ to the alveolar crest (AC) divided by the distance from the CEJ to the root apex (RA), multiplied by 100
- It should be noted that in 95% of the radiographic of healthy subjects (no bone loss), CEJ-AC distances range between 0.4 mm and 1.97 mm. Therefore, subjects with mean bone distance of < 2.0 mm are defined as having no detectable bone loss.

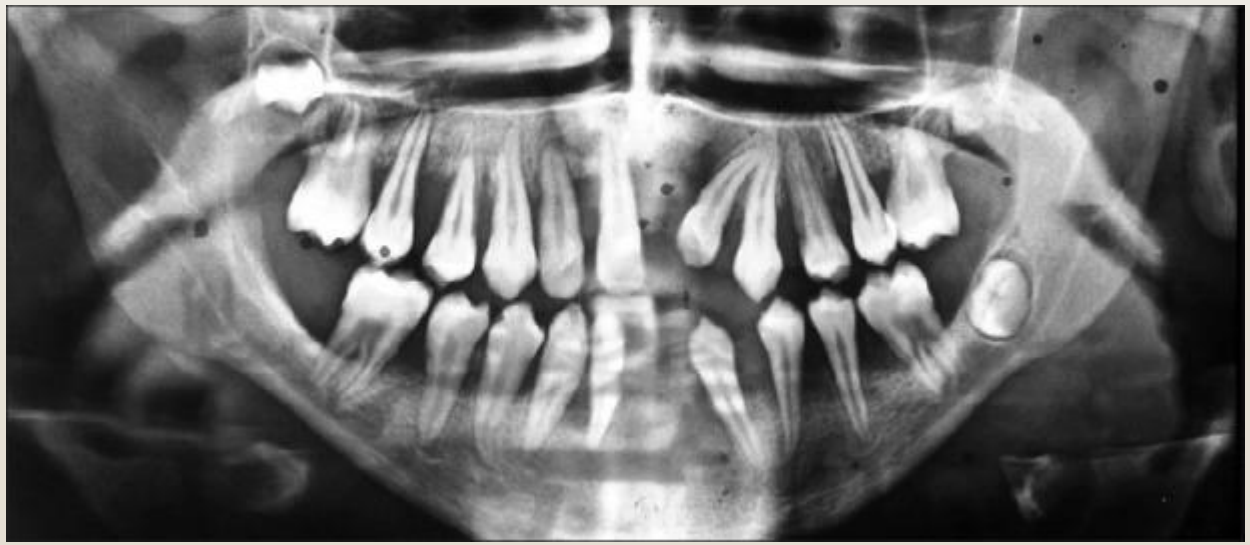


- 2-Pattern of bone loss: two main patterns of bone loss are described:
- a- Horizontal bone loss which occurs when the interproximal bone crest resorbed essentially evenly between teeth, this pattern of bone loss is associated with supra-bony true pocket where the base of the pocket lies coronal to AC.
- b- Vertical or angular pattern when more bone loss occurs on one side of the interproximal bone crest than the other side, which is associated with infra-bony true pocket. This means the base of the pocket lies within the bone defect.



- 3-Distribution: according to the international workshop for classification of periodontal diseases and conditions in 1999, the distribution can be defined by :
- • Localized: if the bone loss affect ≤30% of the sites
- • Generalized: if the bone loss affect > 30% of the site.





Clinical examination

Extra oral examination:

Extra-oral examination is a mandatory part of examination.

Head and neck lymph nodes should be examined for any enlargement or tenderness.

Any infection or neoplasm in the head intra-oral or extra oral area might lead to painful, swelling and warmth of the lymph nodes in the area.

However in periodontitis, it is rare to see any palpable lymph nodes unless there is periodontal abscess or any other acute infection.

Intraoral examination:

- Include examining the lips, oral mucosa, oropharyngial area

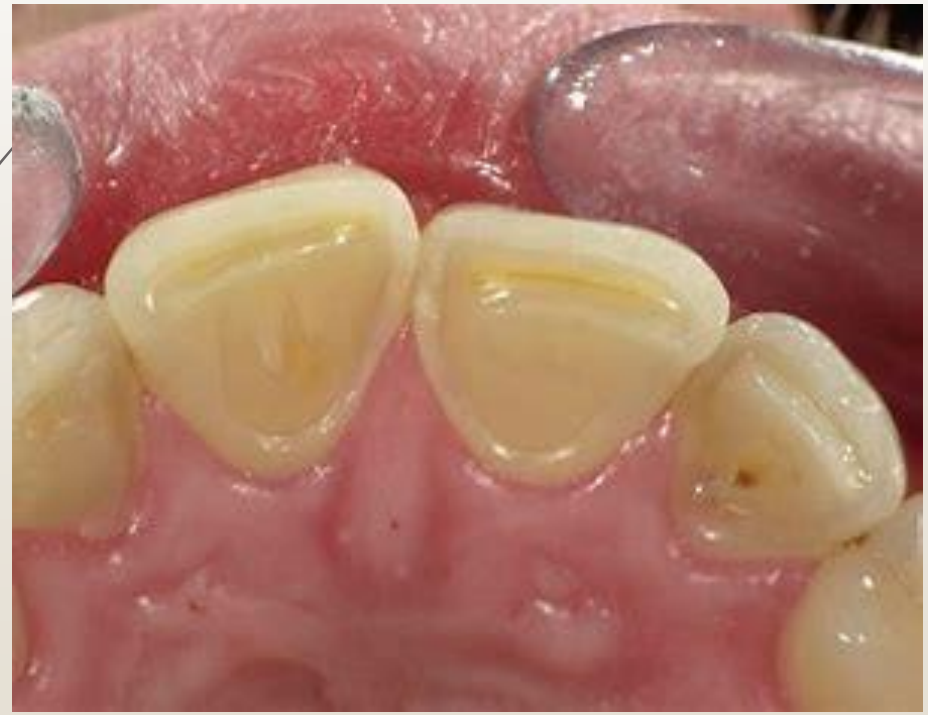
Teeth:

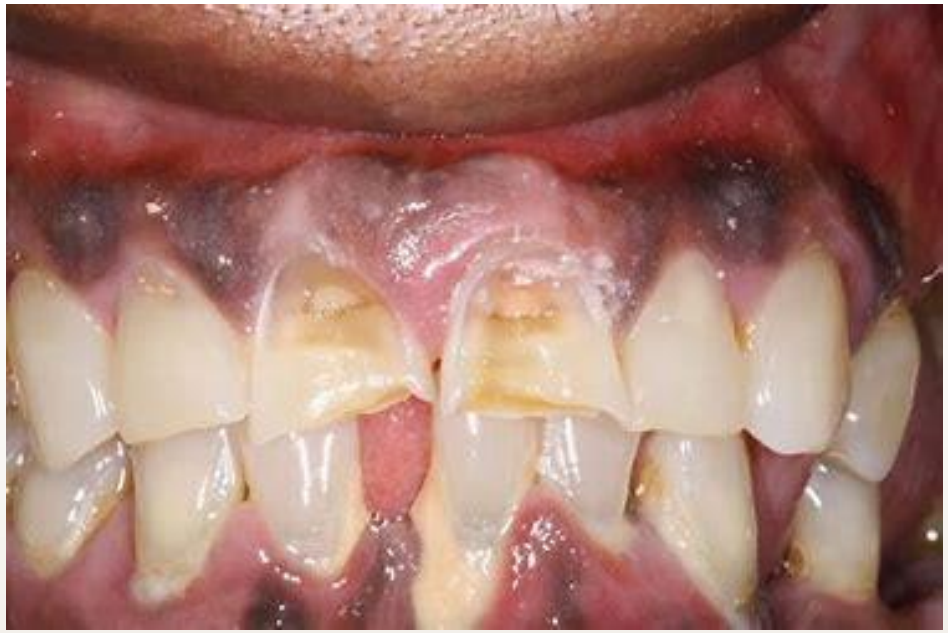
- Teeth should be examined for any caries, deformities and anomalies, wasting of teeth, staining, hypersensitivity.

Wasting of teeth is any gradual loss of tooth substance leaving a smooth

- surface, regardless of the cause of this wasting. It includes erosion,
- abrasion and attrition.

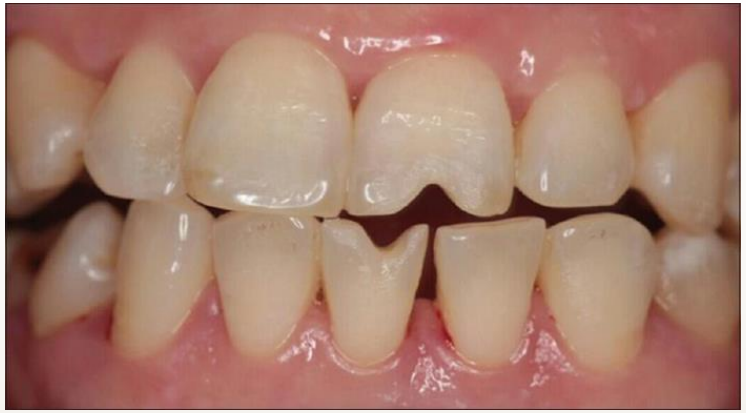
- Erosion: is a wedge shaped depression on the cervical area of the tooth
- surface. In early stages it affects enamel only but in late stages it might reach to the dentin.
- acidic beverages and citric acid
- intrinsic acidic regurgitation or vomiting such as in cases like bulimia nervosa or chronic gastro-oesophageal reflux . The pattern and the area affected might be important in the diagnosis. In cases of intrinsic acids the erosion will be more obvious in the lingual cervical area of the anterior mandibular teeth.

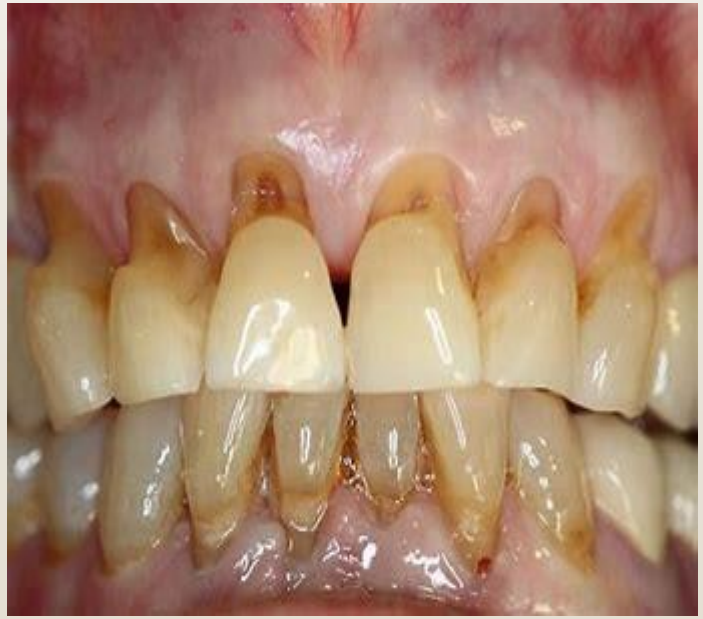




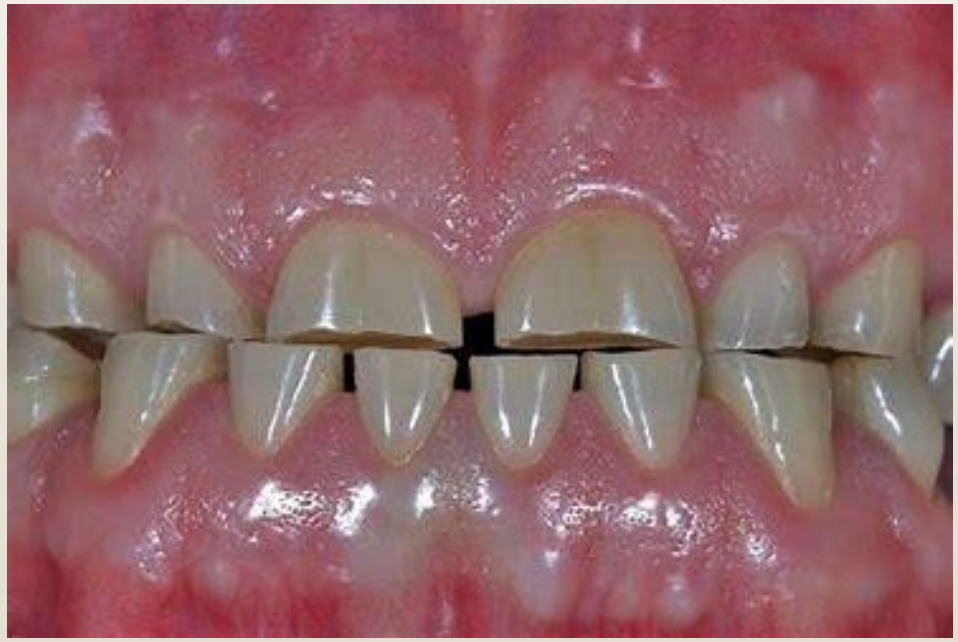
Abrasion:

- is a wear of tooth substance caused by mechanical action other than mastication and occlusion. It appears a saucer shape or wedge shape loss of tooth substance on the cervical area, more often on the cemental surface of the exposed root. Cementum is weaker than enamel.
- This defect may reach the dentin. If the cervical area affected, it is more likely because of tooth brushing using abrasive dentifrice or the presence of clasps of prosthesis.
- The way of tooth brushing might be an essential cause in abrasion, especially when using a horizontal brushing technique and the toothbrush is perpendicular to the vertical axis of tooth surface. In some cases we can see an abrasion on the incisal surface which could be as a result of bad habit, such as pencil biting or holding bobby pin.





- **Attrition:** loss of tooth substance from the occlusal surface due to functional contact. The surfaces of the affected teeth are flat and smooth.
- Functional tooth wear which is due to mastication is a slow and gradual process. Bruxism during sleeping is also a cause of attrition. Another cause of attrition is porceline crowns which can wear down natural teeth in contact.

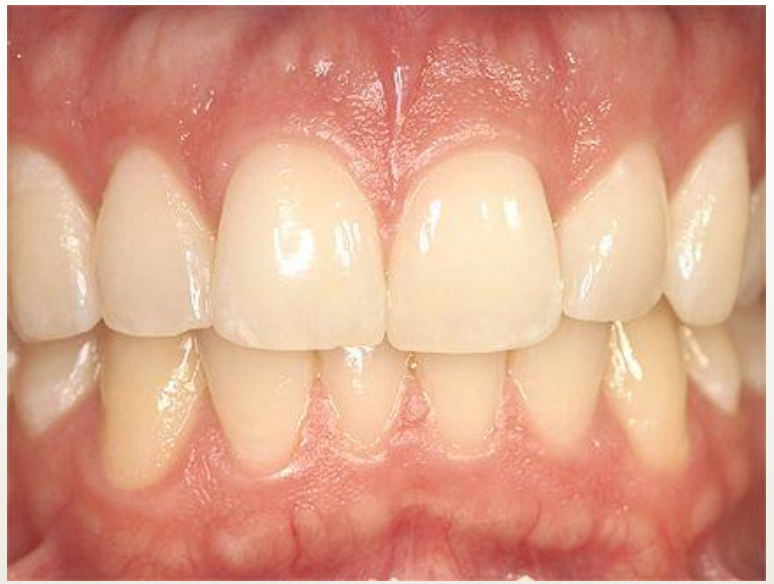


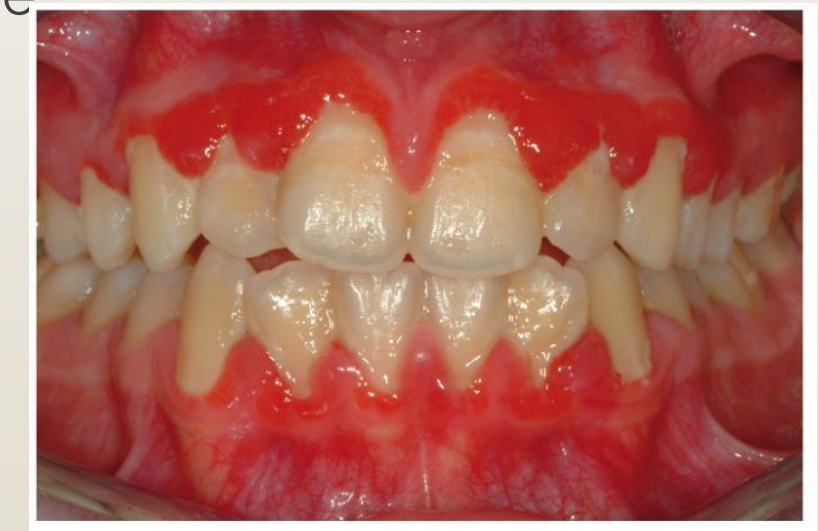
Clinical periodontal examination

- A part of the routine oral examination.
- Consists of;
  - Visual inspection of the gingiva,
  - Dental plaque and calculus,
  - Periodontal probing,
  - Tooth mobility.
- Periodontal chart should be used for recording periodontal findings
  - Act as a reference
  - Compare between the findings in each visit.

Gingival examination

- Visual inspection of inflammatory signs
- Healthy gingiva:
  - Light pink colour
  - Knife-edged appearance of the free gingival margin
- Inflamed gingiva;
  - Redness or erythema
  - Swelling or an enlarged appearance of the marginal gingiva

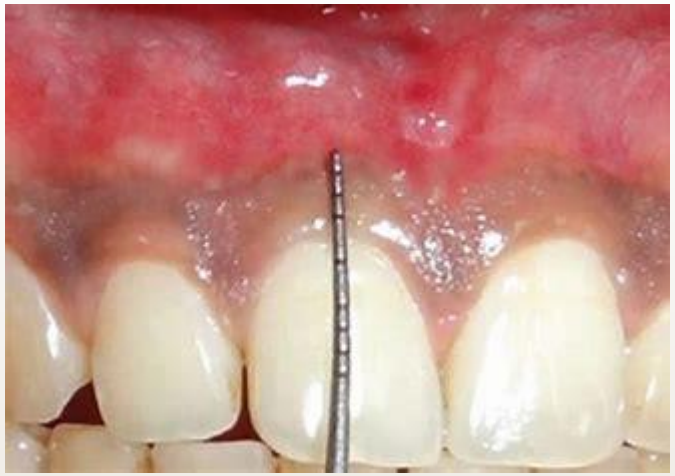




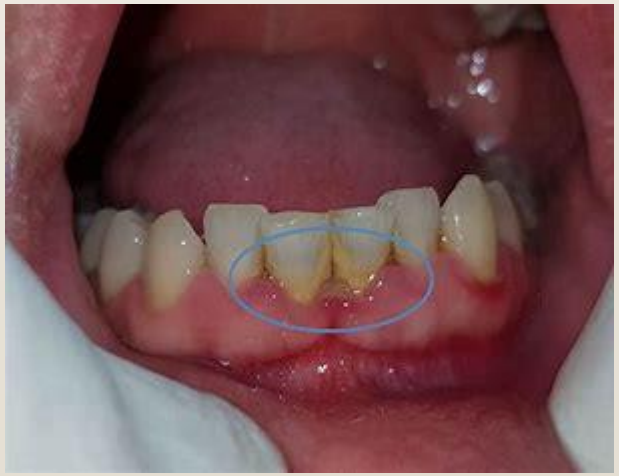
Gingival examination

- Inspect the consistency of gingival enlargement by pressing the side of a periodontal probe on the gingival swelling, to determine whether it is oedematous or fibrotic (an imprint will appear in case of the oedema).
- Bleeding from the gingival crevice when the inner aspect of the gingival sulcus is gently swept with the side of a periodontal probe
- Assess attached gingiva, in case of recession or a high fraenum attachment
- Interdental cratering with or without exposure of the underlying bone.
  - Necrotizing ulcerative PD
  - Immunocompromised patients with AIDS.
- Palpation of the gingiva is important to detect any swelling and abscess.

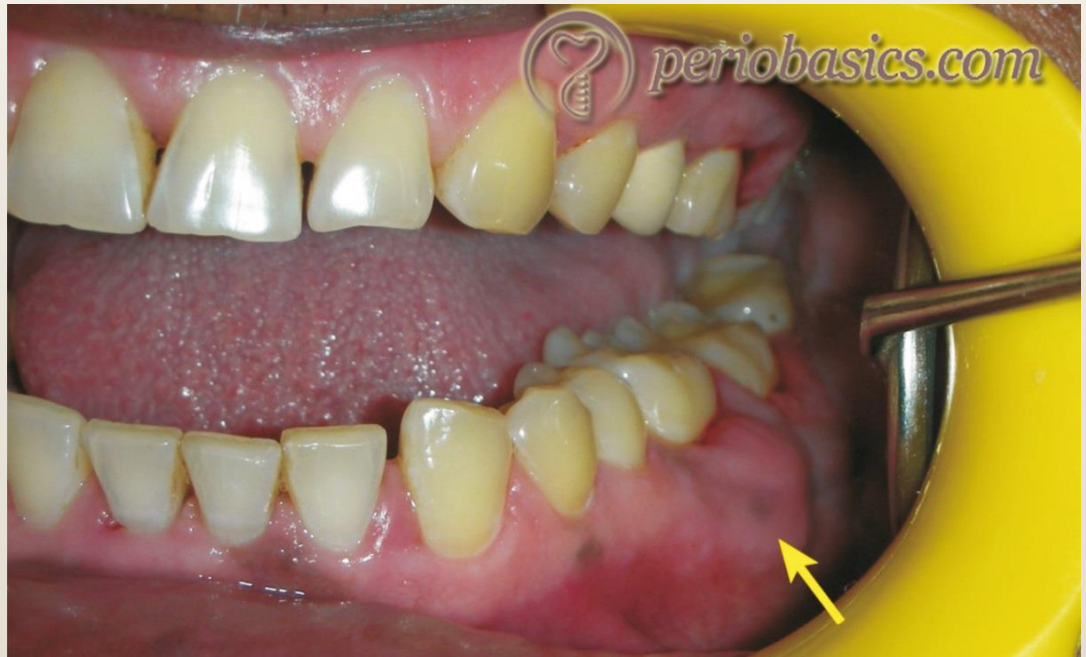
Attached gingiva



NUG



Periodontal abscess



Periodontal Indices

- There are several separate indices for clinical periodontal examination
- Precedes any radiological examination or other special tests.
- The information taken from them allow the clinician to decide on a logical basis whether additional tests are required.

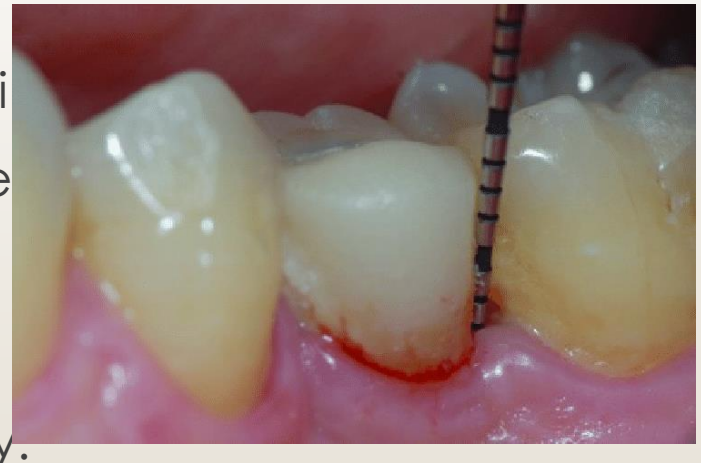
Plaque Index (PLI)

- Match the Gingival Index completely.
- Each of the four gingival areas of the tooth is given a score from 0-3. The scores from the four areas of the tooth may be added and divided by four to give the PLI for the tooth. Finally, by adding the indices for the teeth and dividing by the number of teeth examined, the PLI for the patient is obtained.
- PLI=0 no plaque
- PLI=1 film of soft deposit on the gingival margin, detected by running the periodontal probe along the tooth surface.
- PLI=2 moderate accumulation of dental plaque on the tooth surface, seen by naked eye.
- PLI=3 a large amount of soft deposits within the pocket, on the gingival margin and adjacer

Bleeding on Probing (BOP)

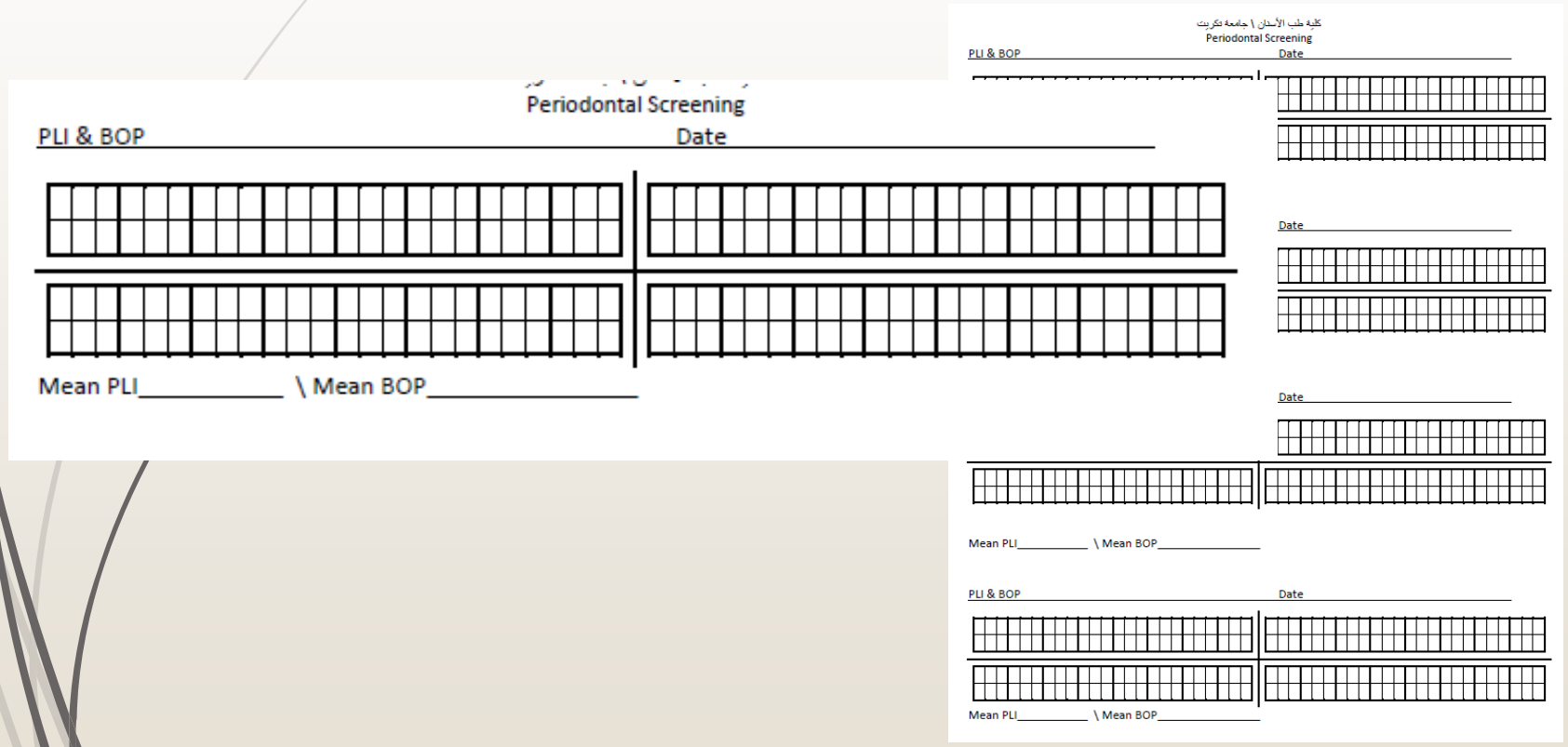
- The first thing to be examined
- Clinical indicator of disease activity.
- Running a periodontal probe gently along the bases of the gingival sulci
- Keep the probe tip moving, as applying poi

pressure may result in false positive results due to localized soft tissue penetration.



- Wait 30 seconds after probing.
- Absence of BOP means periodontal stability.
- Almost 100% of sites with lack of BOP in non-smokers will not progress to attachment loss.

PLI & BoP


Periodontal Screening
PLI & BOP
Date
Mean PLI_____ \ Mean BOP_____

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Periodontal Screening
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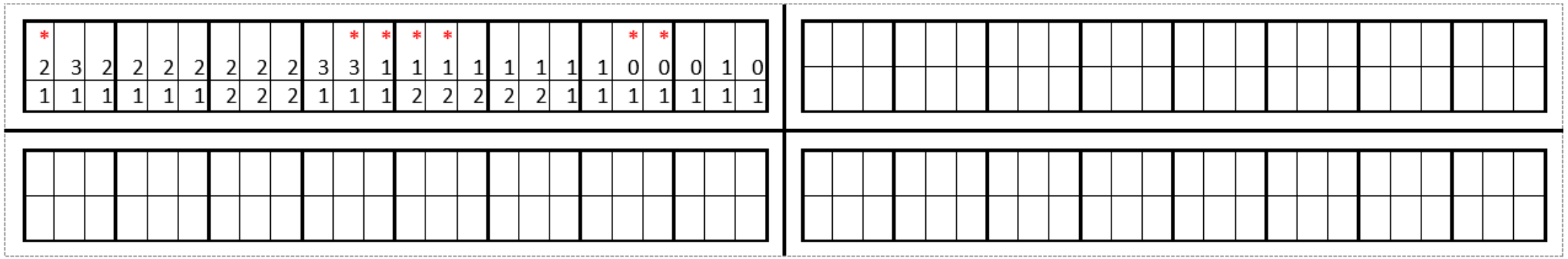
PLI & BOP Date



Mean PLI_____\ Mean BOP_____

Example:
Mean PLI= 250/192= 1.3
BoP= 27/192*100= 14%

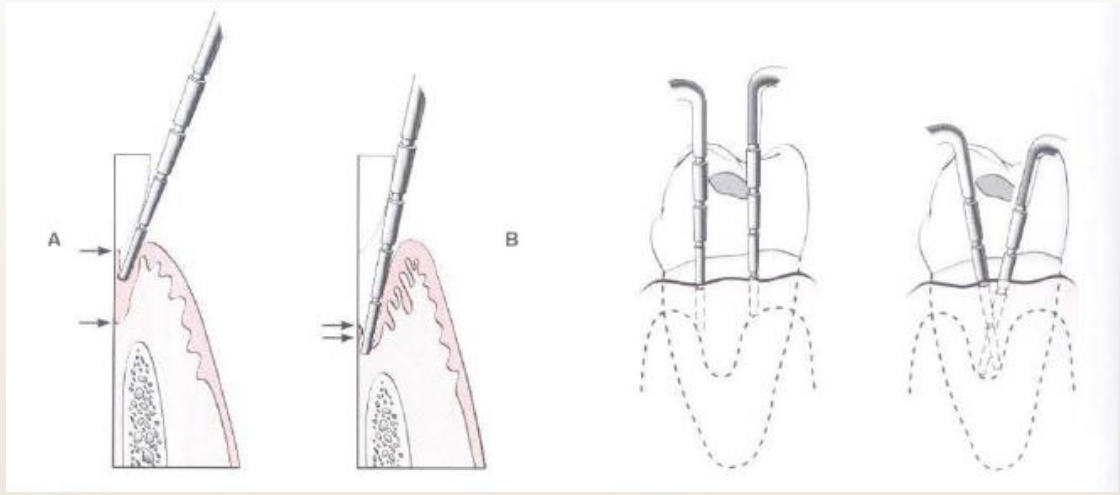
Diagnosis

Case definition of gingivitis in an intact periodontium

	Localized gingivitis	Generalized gingivitis
Probing attachment loss	No	No
Radiographic bone loss	No	No
BOP score	≥10%, ≤30%	>30%

Probing pocket depth (PPD)

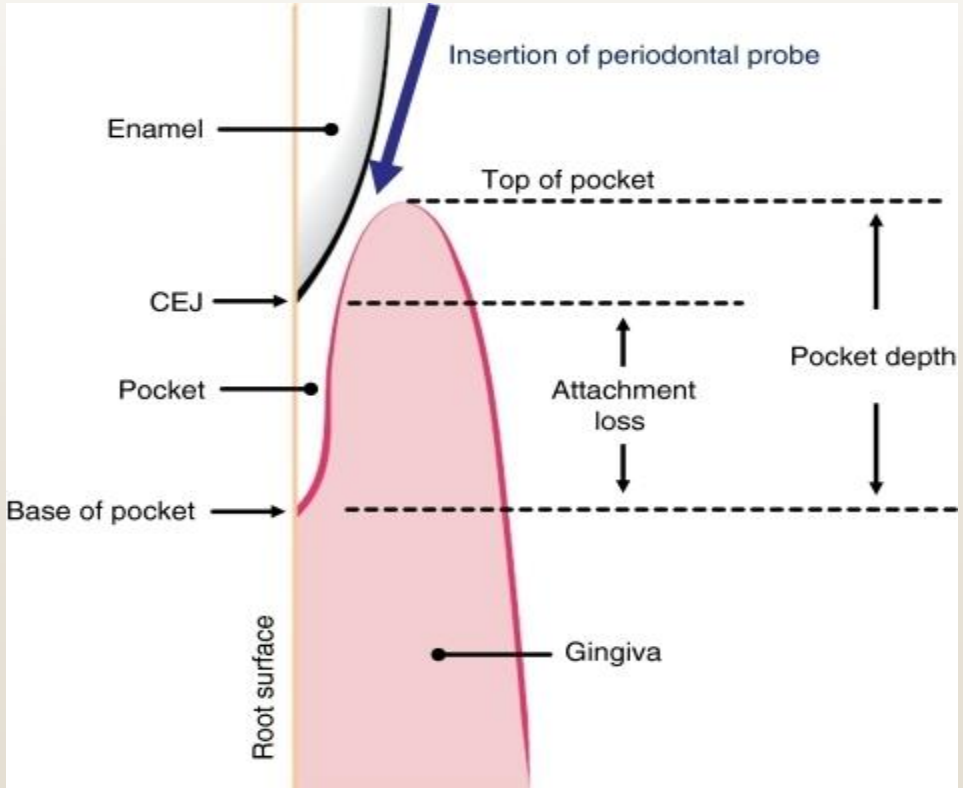
- Physical method to measure in mm, the distance from the base of the pocket to the gingival margins.
- This depth is not fixed
- Not reliable.
- Factors affecting probe penetration:
  - Probing force should not exceed 0.70N,
  - probe design,
  - probe angulation,
  - pocket depth
  - degree of inflammation
  - In inflamed tissue, probe might penetrate into the tissue to about 0.3mm.

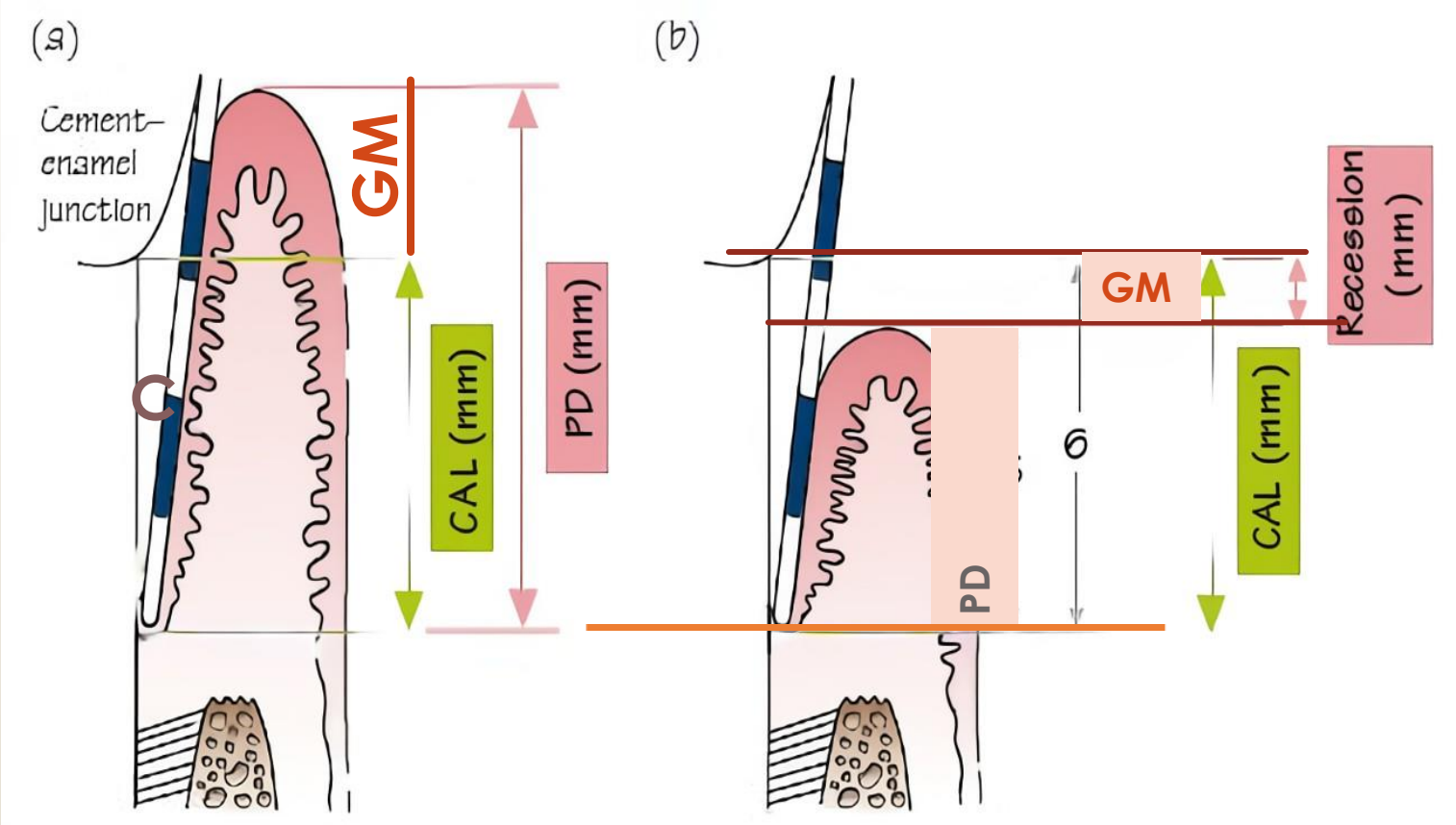


- The probe should be inserted parallel to the tooth vertical axis, and walked circumferentially around the tooth. The deepest score is recorded in each site.

Loss of attachment (LOA)

- The distance between the cementoenamel junction (CEJ) and the base of the sulcus
- Measured with periodontal instruments. Includes both pocket depth and recession measurements.
- LOA and the PPD are different
- If the gingival margin lies at CEJ junction then PD=LOA.



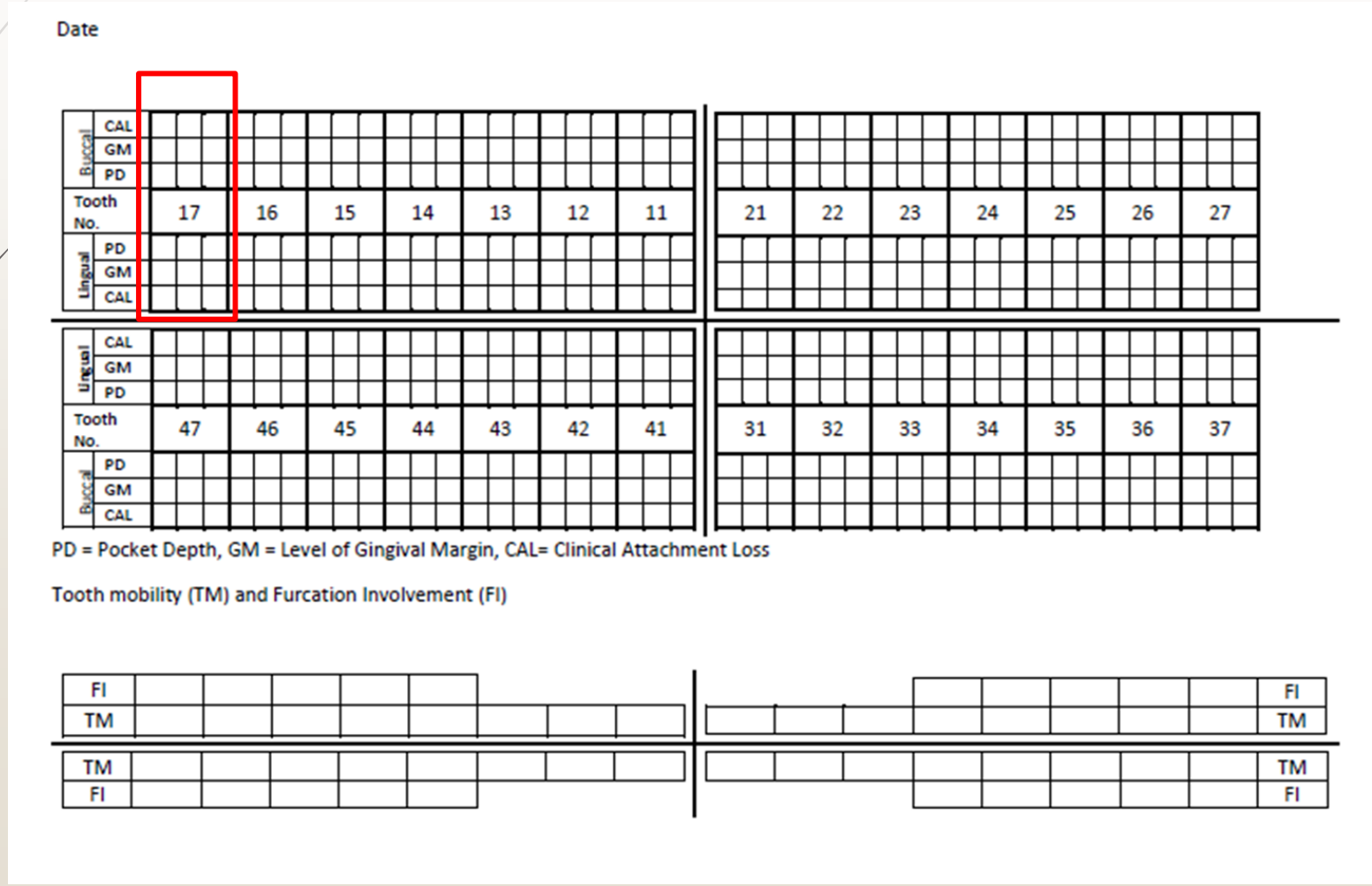


CAL=PD-GM

CAL=PD+GM

Distribution

- Localized: if the bone loss affect ≤30% of the sites
- Generalized: if the bone loss affect > 30% of the site.



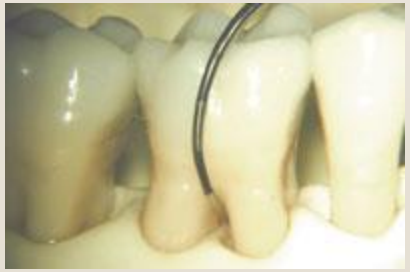
Furcation involvement

- Horizontal loss of support in the areas where the roots of multi-rooted teeth meet.
- Controlling disease and preventing recurrence at such sites is less likely.

Why?

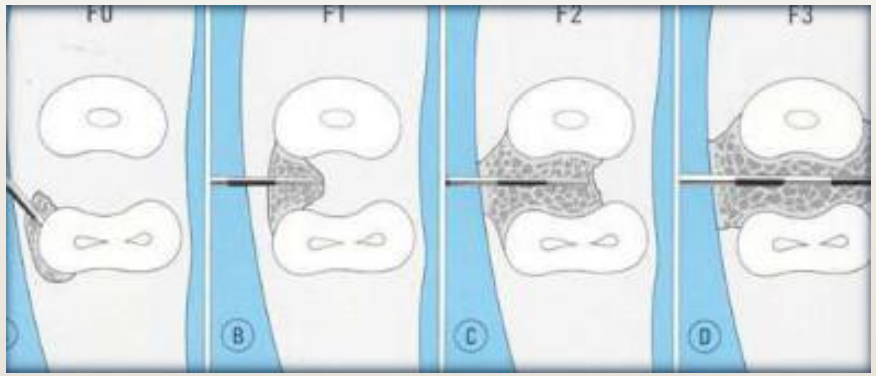
- Furcation area difficult for both the clinician and the patient to gain access and thus to clean.
- presence of the accessory canals in the furcation area

- Furcation involvements can be measured by the degree of horizontal penetration
- using a special probe with angulation called Nabers probe.



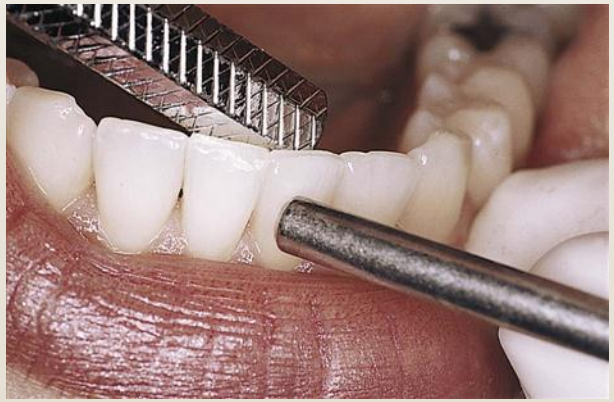
Furcation involvement

- Hamp index; the reference point an imaginary plane connecting the convexities of the roots forming the furcation
- Degree 1 less than 3 mm
- Degree 2 more than 3 mm.
- Degree 3 all the way through



Tooth mobility

- Teeth physiologically move within the socket.
- Any mobility beyond this physiological limit is called pathologic or abnormal.
- To examine tooth mobility put the tooth between two handles of metallic instruments or between one handle and a finger, and try to exert a force to move the tooth in a bucco-lingual direction.



Mobility

- 0: physiologic motion.
- 1: greater than normal
- 2: more than 1mm in a buccolingual direction
- 3: more than 1mm movement in a buccolingual direction with vertical direction.
- Vertical movement can be detected by pressing the tooth into its socket and observing if there is vertical movement.

Thank you

Epidemiology of periodontal disease

Dr Hadeel Mohammed Abbood

The need for epidemiology

- Epidemiology is the study of the distribution and the determinants of health-related status or event in a population.

- It represents the basic science of public health. In public health clinics they look for the determinants of the disease in the whole population. This might be different from the determinants of the same disease in individuals that clinicians look for in clinical practices. For example, the determinants of periodontitis in individuals are dental plaque and calculus, whereas, public health practitioners might determine other causes for periodontitis in population, such as low socioeconomic status or lack of access to dental clinics.

- In epidemiology, the distribution of a given disease is usually described by percentage and the number of persons affected among subgroup, such as gender, socioeconomic status, ethnicity, education level or other characteristics in the population. The difference in distribution among subgroups is not random. It is because of physical, biological, behavioural, cultural, social or other factors that make this subgroup more susceptible to disease than other subgroups.

Why is it important to study epidemiology?

1. From studying the epidemiology of the disease, we can know the prevalence of the disease, and whether it is rare or prevalent. It can provide us information about the natural history of the disease too.
2. Epidemiologic studies can identify the causes and the risk factors of the disease
3. It can provide guidance and recommendations for prevention and control of the disease.
4. The new molecular epidemiologic studies can help to identify the perfect time of intervention in the continuum process of the disease
5. It can give us an idea about the prognosis of the disease under specific treatment.

Measuring the occurrence of conditions or diseases:

- Prevalence
- Risk
- The odds
- Incidence rate

Prevalence

- is the sum of all examined individuals or sites that exhibit the condition or disease of interest divided by the sum of the number of individuals or sites examined. The prevalence can range from 0 to 100%.
- As an example of prevalence, the CDCP reported about the prevalence of individuals with at least one periodontal pocket depth of 4 mm or deeper:
  - from 1988 to 1994, a little more than 1 in 5 Americans had such a condition, for a prevalence of a little more than 20%;
  - from 1999 to 2004, only 1 in 10 Americans fell into this category, for a prevalence of around 10%.
  - These findings suggested a 50% decline in the prevalence of ≥4 mm PD for adults aged 20 and 64 years, which occurred over approximately a decade.
  - Many countries do not have prevalence surveillance systems, which makes it difficult to determine whether these trends observed in the United States are isolated events or part of a more general trend.

Risk

- The *risk* is the probability that an individual or a site will develop a particular condition or disease during follow-up.
- The risk for a condition or a disease is a number that ranges between 0% and 100%.
- The simplest way to estimate risk is to have a fixed number of persons or sites at risk at some defining moment (i.e., time zero [t0]). After a follow-up period (i.e., from t0 to tn), the risk can be calculated as the proportion of persons or sites in which the clinical outcome of interest develops during the follow-up period.
- When a risk is reported, it should be accompanied by a specific time period to which it is applied. A 5% risk for death may be considered small when it refers to a 20-year period but large when it refers to a 3-month period.
- As an example, consider concerns about occupational human immunodeficiency virus (HIV) infection among dentists. It has been reported that the risk for developing an HIV infection within the year subsequent to an accidental needle stick with HIV-contaminated blood is 0.3%. Such a statistic has an intuitive appeal and can be related to patients or colleagues.
    - A risk of 0.003 (0.3%) indicates that for every 1000 individuals who have an accidental HIV contaminated needle stick, 3 are expected to develop an HIV infection within a year of the event.

The odds

- The odds for an event is the probability that an event occurred divided by the probability that an event did not occur.
- Whereas probability is a value that has to range between 0 and 1, odds values range from 0 to infinity. If the probability for observing an event is small, then the odds and the probability are almost identical. For example, if the probability for a vertical root fracture after an endodontic procedure is 0.001, then the odds are 0.001/0.999 or 0.001001.
- Odds are commonly reported in studies because they are often easier to estimate with statistical models than probabilities. For example, the odds for developing an HIV infection after an accidental needle stick with HIV-contaminated blood are 0.003 (0.003/0.997).

Incidence rates

- are an alternative measure to describe disease occurrence. In clinical trials or epidemiology, the rate reflects the number of disease occurrences per person-time or site-time. the disease rate is a ratio in which the numerator is the number of subjects or sites diagnosed with the disease of interest and the denominator is the sum of the time at risk overall subjects or sites in the population.

- Incidence rates—as opposed to the previously introduced measures of disease occurrence—imply an element of time. The denominator in the incidence rate has time as the dimension.

- Thus, the dimension of incidence rate is 1/time. This dimension is often referred to as “person-time” or “site-time” to distinguish the time summation from ordinary clock-time. The magnitude of the incidence rate can vary between 0 and infinity. When there are no new disease onsets during the study period, the incidence rate is 0. When every person observed dies instantaneously at the start of the study (and thus the sum of the time periods is 0), then the incidence rate is infinity.

- An example of the application of rates is provided in Fig. 6.1, in which the number of teeth lost per 1000 tooth-years is plotted as a function of the maximum probing depth at the start of follow-up. The plot suggests a nonlinear relationship between maximum pocket depth and tooth loss, with a substantial increase in tooth loss rate for teeth that have periodontal pockets of 7 mm or deeper.

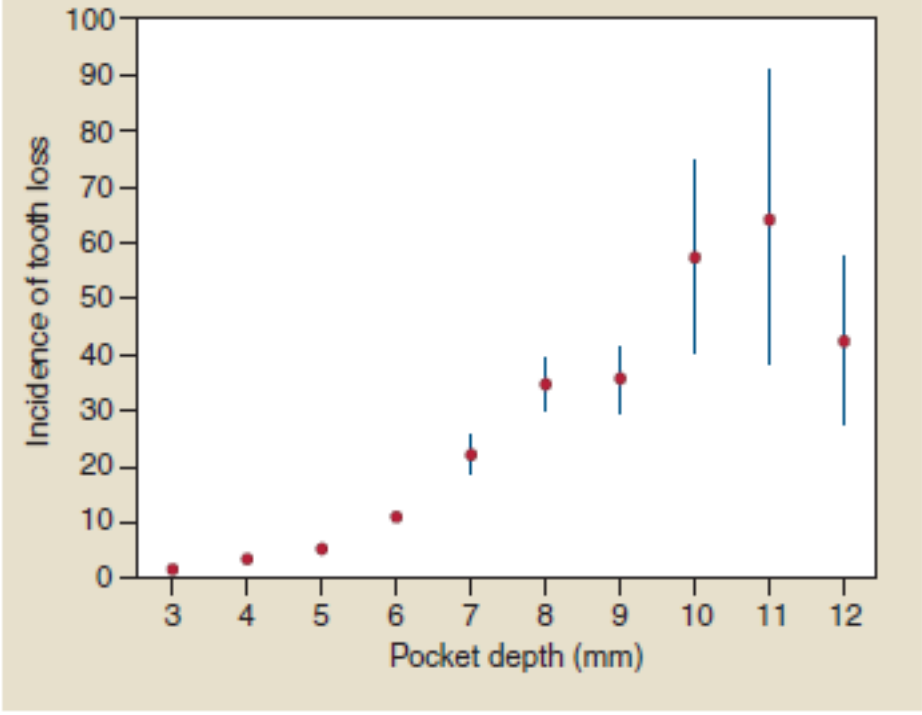


Fig. 6.1 Rate of tooth loss per 1000 tooth-years as a function of maximum probing depth per tooth in a cohort of 1021 patients between the ages of 40 and 65 years under periodontal specialist care for destructive periodontal disease. *(Data from Hujoel PP, Cunha-Cruz J, Selipsky H, et al: Abnormal pocket depth and gingival recession as distinct phenotypes.* Periodontol 2000 *39:22–29, 2005.)*

Typical measurement of periodontal disease

- A periodontal examination can measure various characteristics of the periodontium. Dental records of periodontal patients typically contain information about:
  - Present, missing or impacted teeth
  - clinical probing depth,
  - bleeding on probing,
  - gingival recession,
  - mobility of teeth,
  - furcation involvements .
  - Some clinicians may collect information about the presence of gingivitis by evaluating the color and form of the gingival tissues.
  - These measures can be complemented with radiographic examinations that may provide information about marginal bone levels.
- In research settings or in some selected private practices, additional periodontal measures may be collected, such as clinical attachment levels, microbiologic measures, gingival crevicular fluid volume, biomarkers in the GCF, and indices that measure the amount of gingival inflammation or dental plaque or debris accumulation.

Two common measures of gingival inflammation are:

1. Gingival Index (GI) and
2. Bleeding on probing.

- The GI was proposed in 1963 as a method for assessing the severity and quantity of gingival inflammation. With this particular index, only gingival tissues are assessed. Each of the four gingival areas of the tooth (i.e., facial, mesial, distal, and lingual) are assessed for inflammation and rated as normal gingiva (a score of 0) to severely inflamed gingiva with a tendency to spontaneously bleed (a score of 3). Gingiva that is mildly inflamed but without bleeding on probing is given a score of 1, whereas moderately inflamed gingiva with bleeding is given a score of 2. The scores can be averaged for each patient to provide patient means.

BoP

- The specific approach to obtain a bleeding measure can be done as follows, the examiner dried a quadrant of teeth with air. Then, starting with the most posterior tooth in the quadrant (excluding the third molar), the examiner placed a periodontal probe 2 mm into the gingival sulcus at the facial site and carefully swept the probe. After probing the sites in the quadrant, the examiner assesses the presence or absence of bleeding at each probed site. The same procedure was repeated for the remaining quadrant.

- Commonly used measures of periodontal tissue destruction include
  - mean probing depth,
  - mean attachment loss,
  - and mean recession level.
- The clinical protocols regarding how such mean values are collected and calculated can vary considerably.

True and Surrogate Measures of the Periodontal Condition

- *True endpoints* are tangible outcomes that directly measure how a patient feels, functions, or survives.

True endpoints include oral health–related quality-of-life measurements and self-reported problems, such as a positive answer to the following question: “When you brush or floss your teeth, do you notice bleeding that is both regular and that involves spitting blood-stained saliva?”.

- *Surrogate endpoints* are intangible to the patient. Surrogate endpoints in periodontal research include anatomic measures (e.g., probing depth), measures of inflammation (e.g., bleeding), microbiologic measures, and immunologic measures. Surrogate endpoints are often objective, because they can be measured by the clinician (rather than relying on self-report by patients) or by laboratory methods. Surrogate endpoints can be misleading when the goal is to provide reliable information about clinical decisions related to diagnosis, etiology, treatment, or prognosis.

TABLE 2.1 Examples of Potentially Misleading Surrogates[a]

Disease or Condition	Experimental Treatment	Control Treatment	Effect on Surrogate Endpoint	Effect on True Endpoint	Misleading Conclusion	Reference
AIDS	Immediate zidovudine	Delayed zidovudine	Significant increase of 30–35 CD4 cells/mm^3	No change in incidence of AIDS, AIDS-related complex, or survival	False-positive	80
Osteoporosis	Fluoride	Placebo	Significant increase of 16% in bone mineral density of lumbar spine	Nonvertebral fracture rates increased by 85%	False-positive	
Lung cancer	ZD1839 (Iressa)	Placebo	Dramatic tumor shrinkage in 10% of patients	No effect	False-positive	82
Aphthous ulcers	Thalidomide	Placebo	Although thalidomide expected to decrease TNF-α production, significant increase of 4.4 pg/mL in TNF-α production occurred, suggesting harm	Pain diminished and ability to eat improved	False-negative	32
Edentulism dentures	Implant-supported	Conventional dentures	No impact on chewing cycles	Improved oral health–related quality of life	False-negative	5
Prostate cancer	Radical prostatectomy	Watchful waiting	Substantial elimination of tumor mass	No effect on overall mortality risk	False-positive	78
Advanced colorectal cancer	5-FU + LV	5-FU	23% of patients had 50% or greater reduction in tumor volume	No effect on overall survival	False-positive	40
Periodontitis	Surgery	Scaling	Mean pocket depth reduced by 0.5 mm	Effect on tooth loss or quality of life unknown	?	30a

[a]For some examples, the experimental treatment led to improvements in surrogate endpoints, whereas the true endpoint was either unaffected or worsened (a false-positive conclusion). For other examples, the experimental treatment had no impact or worsened the surrogate endpoint, whereas the true endpoint improved (a false-negative conclusion).
AIDS, Acquired immunodeficiency syndrome; *5-FU*, 5-fluorouracil; *LV*, leucovorin; *TNF-α*, tumor necrosis factor-alpha.

Epidemiologic Study Designs

Randomized Controlled Trials (RCT)

- RCT in periodontics typically assign patients or some teeth within a patient randomly to a treatment. Patients are then monitored, and subsequent outcomes are assessed.
- Requirement of designing an RCT;

1. there needs to be a pretrial hypothesis that specifies the endpoint,
2. the treatments to be compared,
3. the patient population,
4. the degree of required precision.
5. A secure randomization process,
6. the masking of patients and clinicians,
7. the presence of an independent data and safety monitoring board,
8. Strict adherence to the pretrial hypothesis, which must include an intent-to-treat analysis.

- It is important to strictly follow the up mentioned factors in RCTs to reach to a reliable result. Otherwise, false positive results might be obtained.

Cohort Studies

Cohort studies can also be referred to as exposure-based study designs. Subjects who are free of the disease of interest are classified with respect to an exposure (e.g., cigarette smoking, diabetes) and followed longitudinally for the assessment of periodontal outcomes.

TABLE 6.4 Examples of Periodontal Cohort Studies

Periodontal Exposure	Outcome	Sample Size
Periodontal disease and tooth loss[35]	Coronary heart disease	51,529
Gingivitis[9]	Tooth loss	>500

Cohorts can be defined by a geographic area. Some natural disease history studies of destructive periodontal disease have been conducted on the basis of geographic location.

Examples include the Norwegian Longitudinal Study.

A cohort can be defined by records (e.g., schools, health insurance plans, unions, industries, professional organizations).

Many cohort studies of periodontal disease outcomes are performed in patients who belong to a particular dental insurance company or to a professional group.

Case–Control Studies

- Persons with a condition or outcome of interest (i.e., cases) are compared with persons without a condition of interest (i.e., controls) with respect to the history of the suspected causal factors.

- The primary goal of a case–control study is to find out what past exposures or factors are different between patients with a disease versus those without the disease.

- The case–control study is a challenging type of study to conduct. Trying to minimize the role of bias in case–control studies require careful planning, conduct, and analysis. Two important elements of the case–control study design are:

1. the definitions of the terms case and control. A case is a person in the population or study group who has been identified as having a particular disease, health disorder, or condition. The case definition should be rigorous to minimize bias and misclassification; it can be based on symptoms, signs, or the results of diagnostic tests.

- In a case–control study, the controls should be at risk for developing the investigated disease and come from the same population that generated the cases. For example, if the investigated disease is root caries, the controls should be at risk for developing root caries (i.e., have exposed root surfaces) and originate from the same population that generated the cases that have root caries.

Suspected Modifiable Causative Factors for Periodontal Disease

- **Tobacco Smoking**
- Tobacco smoking is recognized by several organizations as one of the primary drivers of periodontal disease epidemiology. Many criteria for causality have been satisfied, and smoking cessation has been shown to slow the progression of periodontal disease.
- The strong impact of tobacco smoking on periodontal disease has the potential to induce spurious causal associations in other suspected risk factors for periodontal disease. For example, smoking is a risk factor for both type 2 diabetes and periodontal disease, thereby making associations between type 2 diabetes and periodontal disease susceptible to biases.
- To obtain reliable inferences about causal factors other than smoking, studies of periodontal disease epidemiology may need to be restricted to those who have never smoked.

• Nutrition

• Several studies have demonstrated relationships between periodontal disease and a variety of medical conditions that center on carbohydrate metabolism, including intake of dietary carbohydrates, exercise, obesity, prediabetes, and diabetes.

• A systematic review of randomized controlled trials involving carbohydrates suggested that the increased intake of fermentable carbohydrates may cause an increase in gingivitis.

• Two systematic reviews suggested that diabetes was a risk factor for destructive periodontal disease.

- **Dental Plaque**
- Several systematic reviews have provided evidence that chemotherapeutic and mechanical plaque control will reduce gingival inflammation.
- Essential oils and cetylpyridinium-chloride–containing mouth rinses may reduce gingival inflammation. Interdental brushes may reduce dental plaque, bleeding, and probing pocket depth. Power-driven toothbrushes may be more effective than manual toothbrushes for removing plaque and reducing inflammation.

Thank you

Immunology

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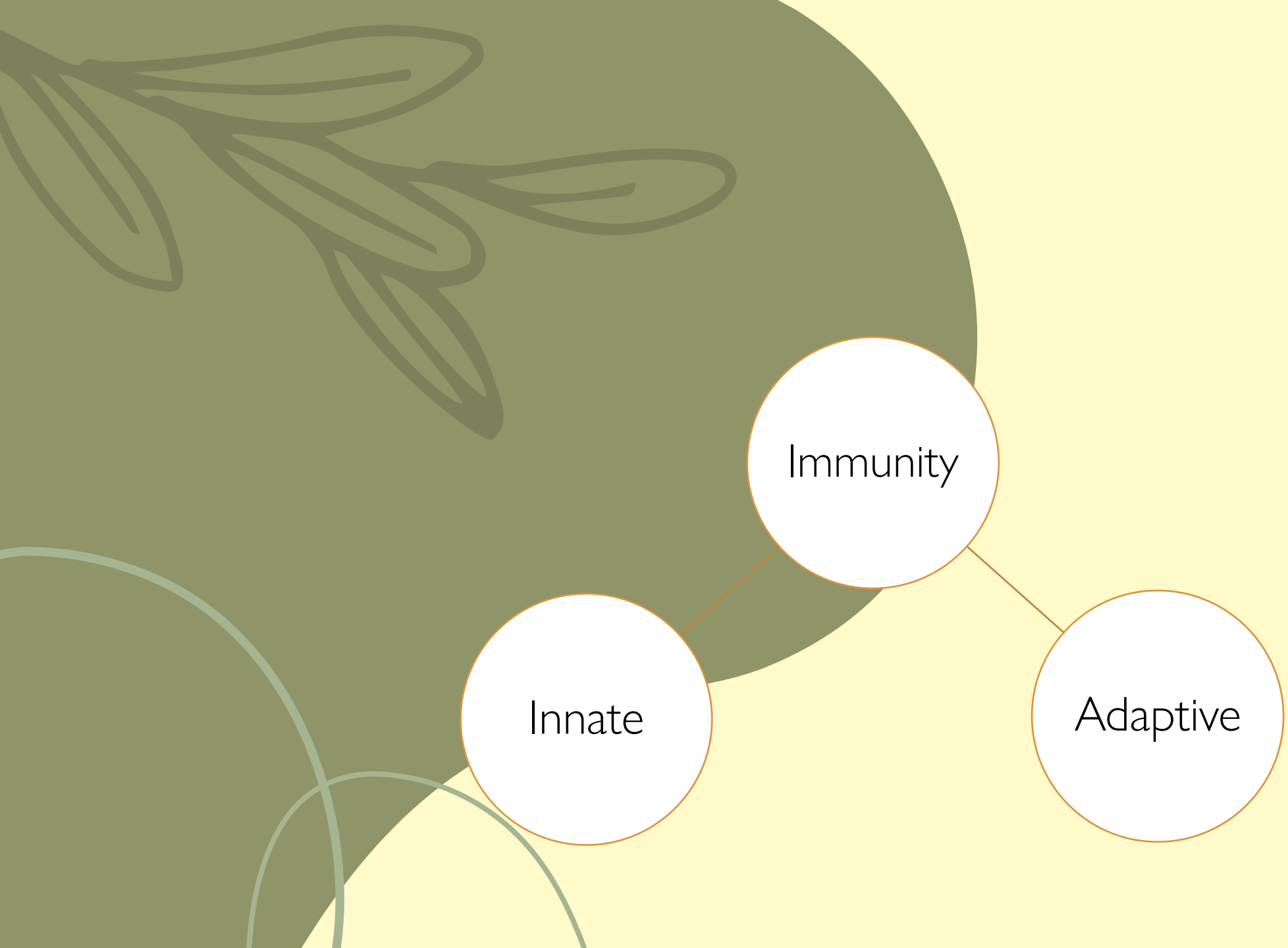
Immune System

- The immune system is essential for the maintenance of periodontal health,
- It is central to the host response to periodontal pathogens.
- If the immune response is dysregulated, inappropriate, persistent, or excessive in some way, then damaging chronic inflammatory responses
- The immune response to plaque bacteria involves the integration at the molecular, cellular, and organ level of elements


Immunity
Innate
Adaptive

Innate Immunity

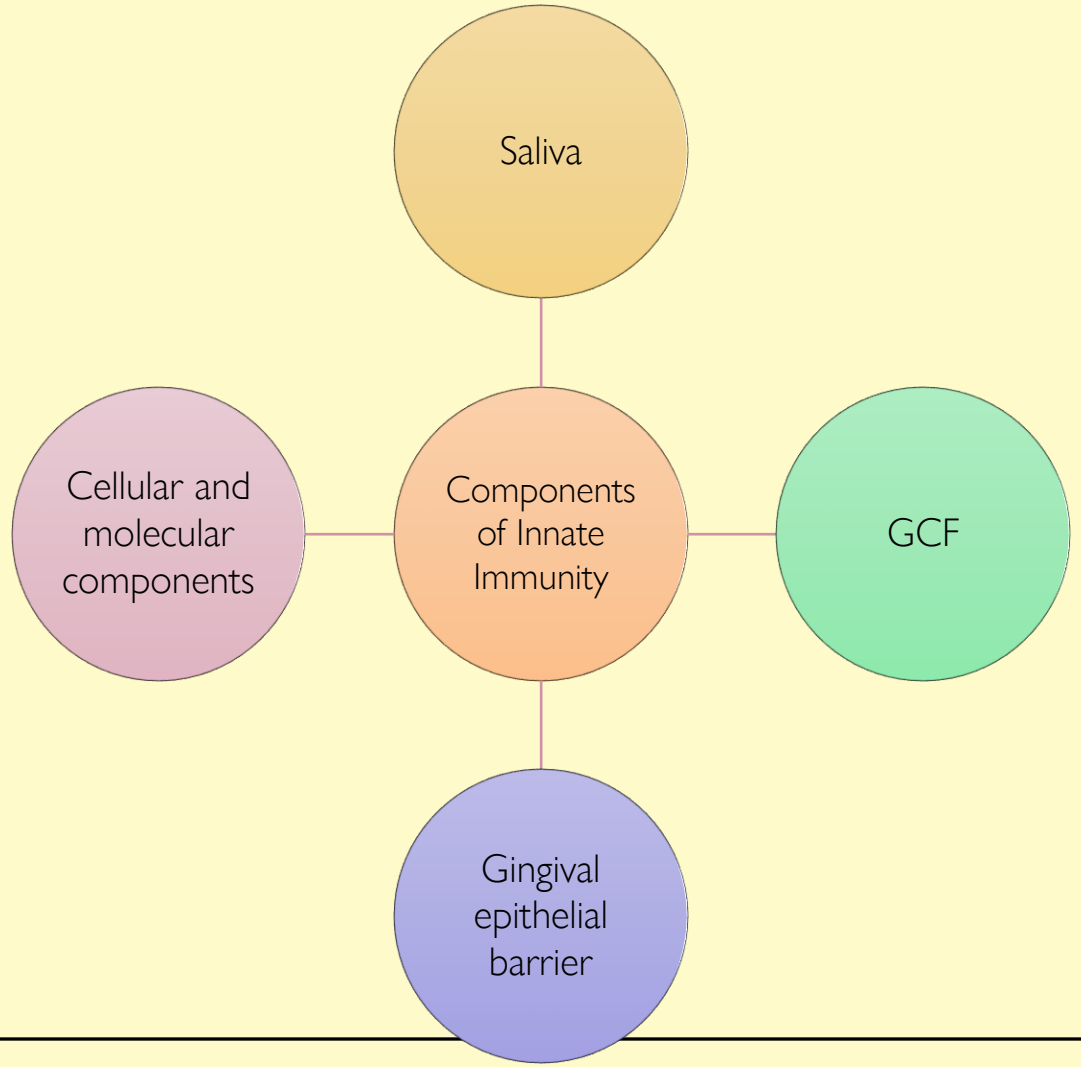
- Defenses against infection include a wide range of mechanical, chemical, and microbiologic barriers that prevent pathogens from invading the cells and tissues of the body.
- Saliva, GCF, and the epithelial keratinocytes of the oral mucosa all protect the underlying tissues of the oral cavity and the periodontium.
- The commensal microbiota (e.g., In dental biofilm) may also be important for providing protection against infection by pathogenic microorganisms through effective competition for resources and ecologic niches and also by stimulating protective immune responses.
- If bacterial products enter the tissues, then the cellular and molecular elements of the innate immune response are activated.

Characteristics of Innate Immunity

- The term innate immunity refers to the elements of the immune response that has 3 main characteristics:
- 1- these elements are determined by inherited factors (and therefore “innate”),
- 2- they have limited specificity,
- 3- they are “fixed” in that they do not change or improve during an immune response or as a result of previous exposure to a pathogen.

- The recognition of pathogenic microorganisms and the recruitment of effector cells (e.g., Neutrophils) and molecules (e.g., The complement system) are central to effective innate immunity.
- Innate immune responses are orchestrated by a broad range of cytokines, chemokines, and cell surface receptors,
- The stimulation of innate immunity leads to a state of inflammation.
- If innate immune responses fail to eliminate infection, then the effector cells of adaptive immune responses (lymphocytes) are activated.
- Immune response functions as a network of interacting molecular and cellular elements in which innate immunity and adaptive (antigen-specific) immunity work together toward a common purpose.

Components of Innate Immunity



Saliva

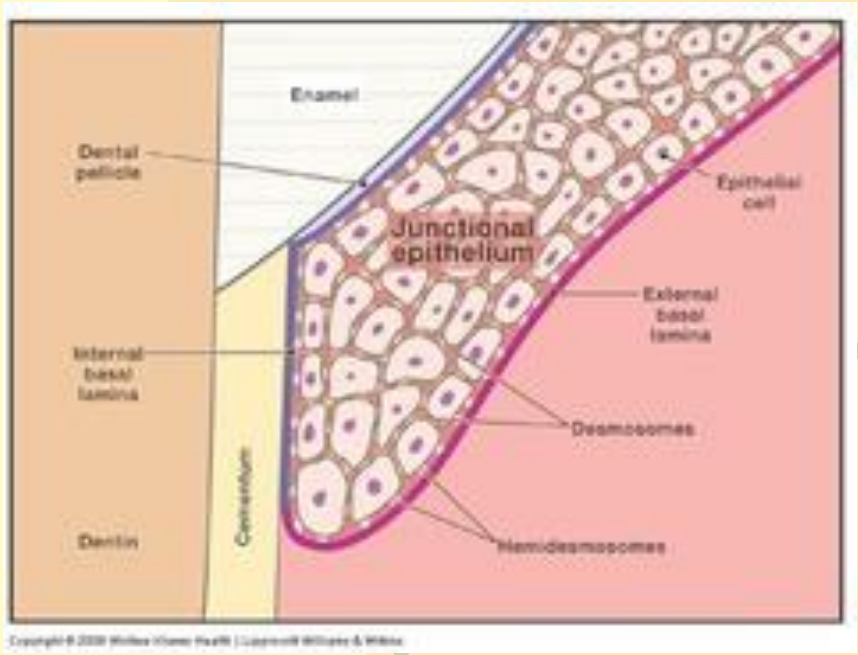
- Saliva that is secreted from the three major salivary glands (i.e., parotid, submandibular, and sublingual), as well as from the numerous minor salivary glands,
- It has an important role in the maintenance of oral and dental health.
- The action of shear forces associated with saliva flow is important for preventing the attachment of bacteria to the dentition and oral mucosal surfaces.
- Human saliva also contains numerous molecular components that contribute to host defenses against bacterial colonization and periodontal disease.

Saliva Constituent	Host Defense Function
Antibodies (e.g., immunoglobulin A)	Inhibit bacterial adherence, promote agglutination
Histatins	Neutralize lipopolysaccharides, inhibit destructive enzymes
Cystatins	Inhibit bacterial growth
Lactoferrin	Inhibits bacterial growth
Lysozyme	Lyses bacterial cell walls
Mucins	Inhibits bacterial adherence, promotes agglutination
Peroxidase	Neutralizes bacterial hydrogen peroxide

Gingival epithelial barrier

- The epithelial tissues play a key role in host defense because:
  - They are the main site of the initial interactions between plaque bacteria and the host,
  - They are also the site of the invasion of microbial pathogens.
- The keratinized epithelium of the sulcular and gingival epithelial tissues provides protection for the underlying periodontal tissue,
- Acting as a barrier against bacteria and their products.

Junctional epithelium



- The junctional epithelium has significant

intercellular spaces,

- It is not keratinized, and
- It exhibits a higher cellular turnover rate.
- These properties render the junctional epithelium permeable, thereby allowing for the inward movement of microbes and their products and the outward movement of GCF and the cells and molecules of innate immunity.
- The spaces between the cells of the junctional epithelium widen with inflammation, which results in increased GCF flow.

Stimulated epithelial cells can produce:

- Matrix metalloproteinases (MMPs), which contribute to a loss of connective tissue.
- Epithelial cells also secrete a range of cytokines in response to periodontal bacteria, which signal immune responses. These include:
  - Proinflammatory cytokines: IL-1β, TNF-α, and IL-6,
  - Chemokine IL-8 and the monocyte chemoattractant protein-1 (MCP-1), which serve to signal neutrophil and monocyte migration from the vasculature into the periodontal tissue.

Gingival crevicular fluid (GCF)

- GCF originates from the postcapillary venules of the gingival plexus.
- It has a flushing action in the gingival crevice,
- It also likely functions to bring the blood components (e.g., neutrophils, antibodies, complement components) of the host defenses into the sulcus.
- The flow of GCF increases in inflammation, and neutrophils are an especially important component of GCF in periodontal health and disease.

Pathogen recognition and activation of cellular innate responses:

Plaque bacteria and their products penetrate the periodontal tissues,

Specialized “sentinel cells” macrophages and dendritic cells recognize their presence and signal protective immune responses.

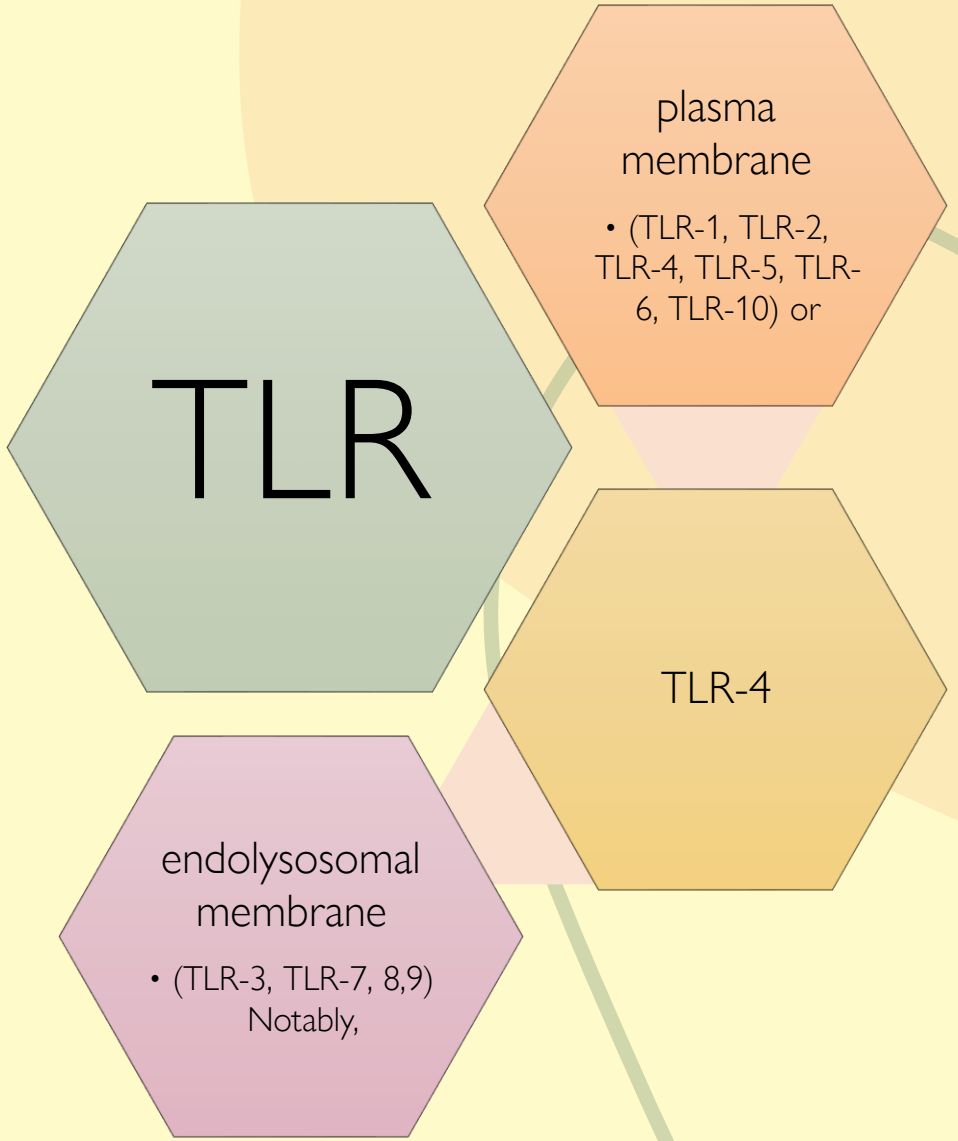
These cells express a range of pattern recognition receptors (PRRs) that interact with microbe-associated molecular patterns (MAMPs).

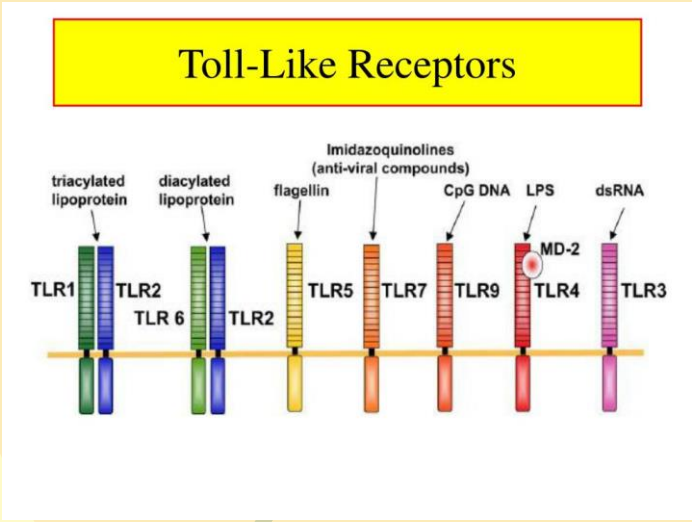
The activation of PRRs activates innate immune responses to provide immediate protection, and adaptive immunity is also activated with the aim of establishing a sustained antigen-specific defense.

- Excessive and inappropriate or dysregulated immune responses lead to chronic inflammation and the concomitant tissue destruction associated with periodontal disease.

Toll like receptors (TLR):

- The TLR family currently consists of 10 known functional TLRs in humans, of which TLR-10 is the only member having an unclear biologic role.
- Bacterial lipopolysaccharides (LPS) with TLRs:
- P. gingivalis, A. actinomycetemcomitans, and F. nucleatum all possess LPS molecules that interact with TLR-4 to activate myeloid immune cells.

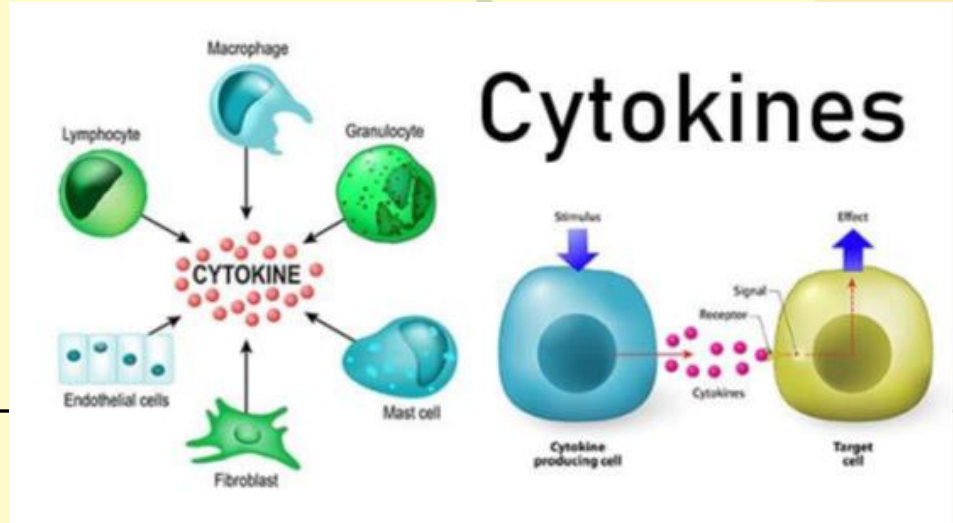




- Individual species of plaque bacteria have a wide variety of MAMPs, which may interact with PRRs.
- P. gingivalis:
  - LPS signals via TLRs (predominantly TLR-2),
  - Fimbriae, proteases, and DNA from P. gingivalis are all recognized by host cells through interaction with specific PRRs.
- Certain nonimmune cells in the periodontium (e.g., epithelial cells, fibroblasts) also express PRRs and may recognize and respond to MAMPs from plaque bacteria.

Pro inflammatory cytokines

- Although the signaling pathways activated by PRRs may be diverse, in general terms, they converge to elicit similar host cell responses in the form of:
  - The up-regulation of cytokine secretion
  - Cell differentiation that leads to enhanced signaling of the adaptive immune response.



The signaling of cytokine responses by PRRs influences:

- innate immunity (e.g., neutrophil activity),
- adaptive immunity (e.g., T-cell effector phenotype),
- The development of destructive inflammation (e.g., the activation of fibroblasts and osteoclasts).

IL-1β

- Activating other cells that express the IL-1R1 receptor (e.g., endothelial cells)
- Stimulating the synthesis and secretion of other, secondary mediators such as PGE2.
- IL-1β also stimulates the secretion of the chemokine IL-8, which stimulates neutrophil chemotaxis.

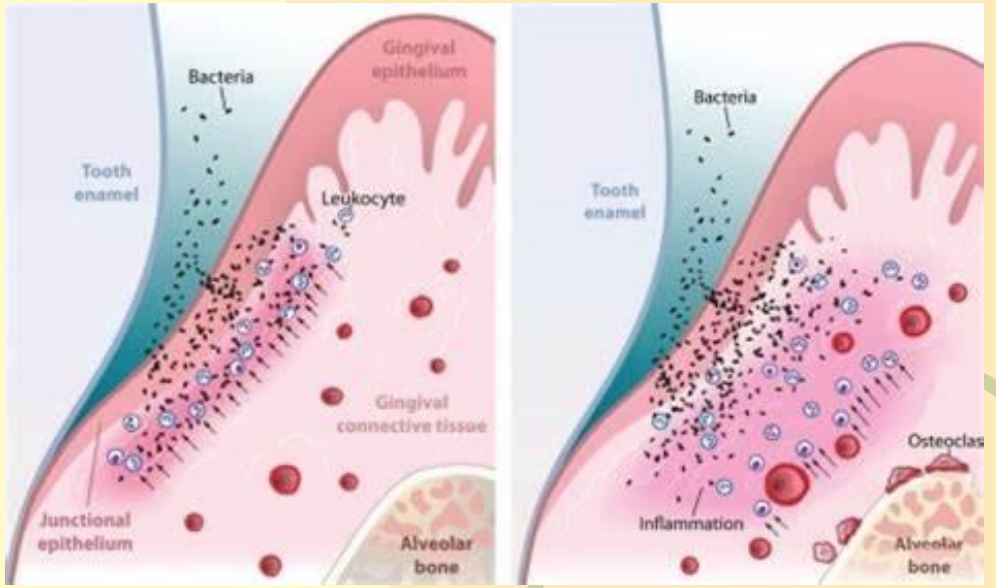
- IL-1β acts synergistically with TNF- α and stimulate migration of neutrophils to the periodontium.

- IL-1β and TNF-α also activate MMP secretion from fibroblasts and osteoclasts; this facilitates the movement of neutrophils through the connective tissues (and thus protective innate responses), but it also contributes to the tissue destruction associated with periodontal disease, along with MMPs from neutrophils.

Other cytokines

- Up-regulated as a result of the activation of PRRs include IL-6, which influences the development of a number of immune cells (e.g., B cells, dendritic cells) and stimulates osteoclast differentiation and thus bone turnover.
- Cytokines from T-cell subsets feedback to and modify innate immune responses; for example, IFN-γ from Th1 cells activates macrophages, and IL-10 and TGF-β suppress immune responses.
- Cytokines are pleiotropic (i.e., they have multiple effects).

Cells of innate immunity:

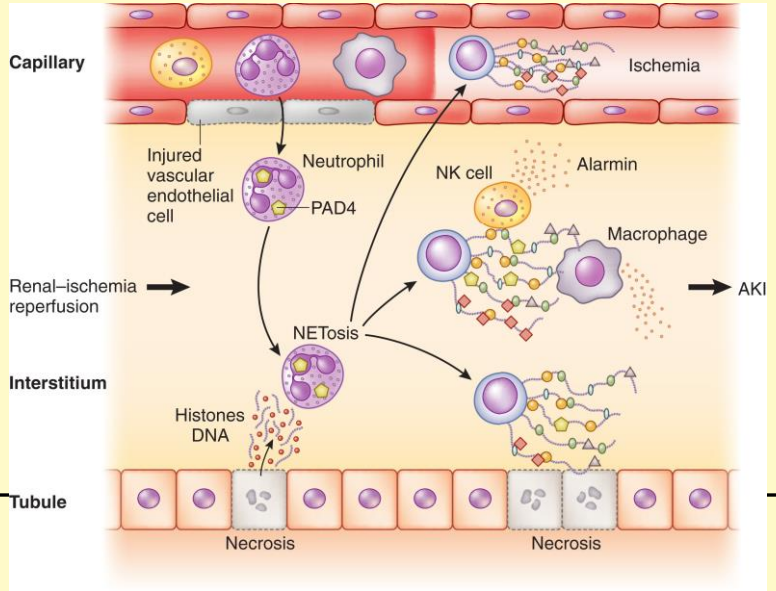


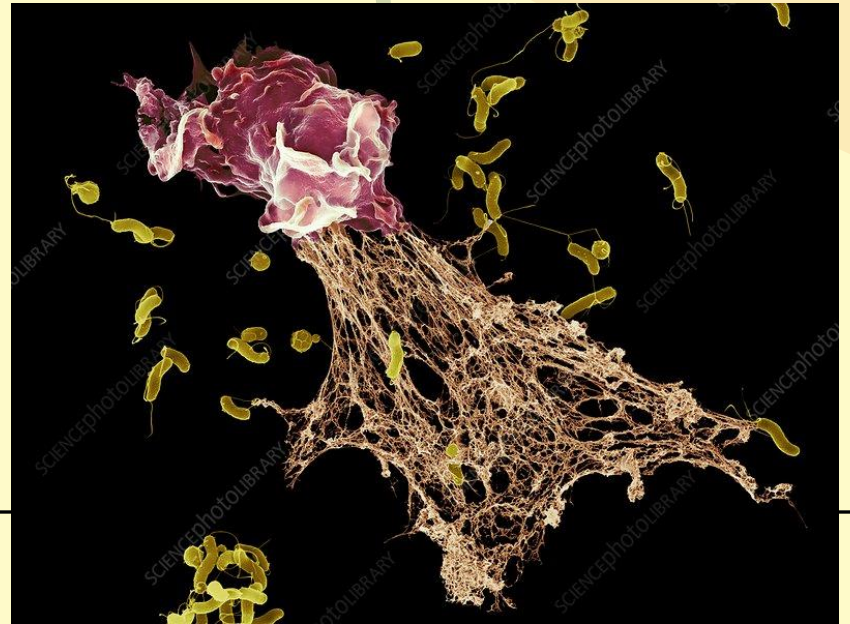
- **Neutrophils**
- Neutrophils are the “professional” phagocytes that are critical to the clearance of bacteria that invade host tissues.
- Present in clinically healthy gingival tissues, and they migrate through the intercellular spaces of the junctional epithelium into the sulcus. This is part of a “low-grade defense” against plaque bacteria, and it is necessary to prevent infection and periodontal tissue damage.
- The importance of neutrophils to the maintenance of periodontal health is demonstrated clinically by the observations of severe periodontitis in patients with neutrophil defects.

- A small proportion (1% to 2%) of the intercellular spaces in healthy junctional epithelium is occupied by neutrophils (and other leukocytes at various stages of differentiation),
- This can increase to 30% with even modest inflammation.
- In the inflammatory state, changes to the local vasculature occur in the gingiva: high endothelial venules develop from the postcapillary venules of the gingival plexus, which facilitates leukocyte emigration and increases the flow of GCF into the pocket.
- Neutrophils migrate from the gingival plexus to the extravascular connective tissue and then into the junctional epithelium through the basement membrane.
- The presence of a layer of neutrophils in the junctional epithelium forms a host defense barrier between subgingival biofilm and the gingival tissue.
- The migration of neutrophils contributes to the disruption of the junctional epithelium by the degradation of the basement membrane through protease release and the action of reactive oxygen species (ROS).

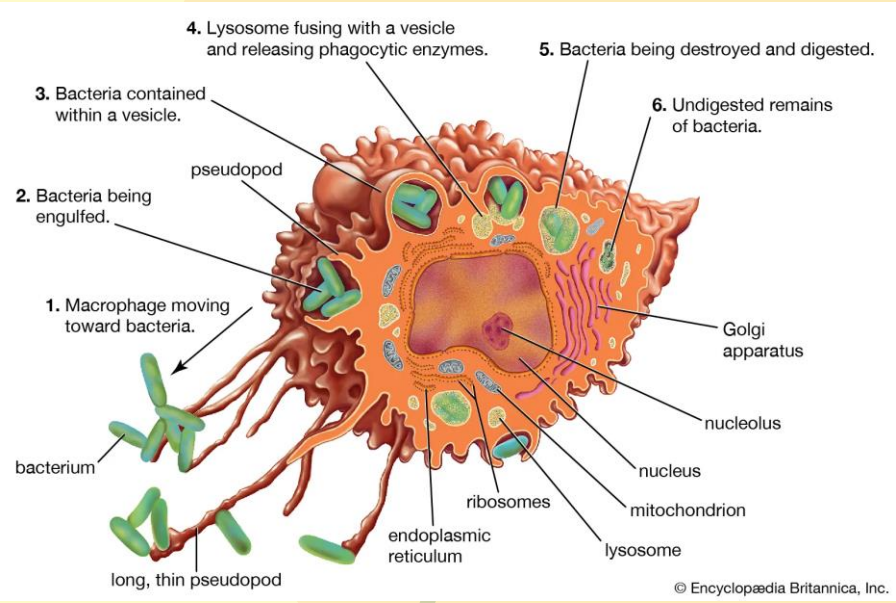
neutrophil extracellular traps (NETs).

- An aspect of neutrophil-mediated immunity
- NETs constitute a highly conserved antimicrobial strategy in which decondensed nuclear DNA and associated histones are extruded from the neutrophil, thus forming weblike strands of DNA in the extracellular environment.
- These strands, in conjunction with antimicrobial peptides (AMPs), facilitate the extracellular killing of microorganisms that become trapped within the NETs.





Macrophages



- One of the key elements of the innate immune system and are involved in the initiation, development, and resolution of inflammatory diseases, including periodontitis.
- In addition to immunostimulatory roles, they play pivotal roles in immunoregulation and tissue repair.
- Macrophages can be differentiated into osteoclast cells.
- They possess PRRs that can recognize invading bacteria and signals the immune response.
- They are important cells in innate and adaptive immune response. In the stage of repair and resolution of inflammation, Lipoxins signal macrophages to phagocytose the remnants of apoptotic cells at sites of inflammation without generating an inflammatory response.

Adaptive Immunity

Adaptive immunity

- Adaptive immunity has evolved to provide a focused and intense defense against infections that overwhelm innate immune responses.
- Adaptive immunity is particularly important as ecologic, social, and demographic changes—which alter susceptibility to existing and emerging infective microorganisms—outpace the natural evolution of biologic systems.
- The development of effective vaccination is, along with the identification of antibiotics, perhaps one of the greatest triumphs of medical science; this success is based on knowledge of the elements and principles of adaptive immunity.

Characteristics

- Adaptive immunity contrasts with innate immunity with regard to the dynamic of the underlying cellular and molecular responses:

1. adaptive immunity is slower
2. relies on complex interactions between APCs and T and B lymphocytes.
3. The antigen specificity of the responses of a diverse range of effector elements, including cytotoxic T cells and antibodies.
4. The ability of adaptive immune responses to improve during exposure to antigen and on subsequent reinfection events.

- Our current understanding suggests that the cellular and molecular elements of adaptive immunity are more diverse than those of innate immunity,
- Although a role for many of these factors in periodontal disease has been identified, our knowledge is far from complete.
- The importance of adaptive immune responses in periodontal pathogenesis is endorsed by histologic studies of established lesions in periodontal disease.

gingivitis and stable periodontal lesions *VS.* active (progressing) periodontitis

- Dominated by T cells, and these cells are clustered mainly around blood vessels.
- These cells are activated but not proliferating.
- A predominance of the helper T-cell subset (i.e., CD4-expressing T cells) over the cytotoxic T-cell subset (i.e., CD8-expressing T cells) is observed.
- These T cells are considered to be proactively maintaining tissue homeostasis in the presence of the microbial challenge of the plaque biofilm.

- B cells and plasma cells predominate and are associated with pocket formation and the progression of disease.

Cellular elements:
Antigen-Presenting Cells (APCs)

- Central elements of the activation and function of T cells and B cells are the presentation of antigen by specialized APCs to T cells and the development of a specific cytokine milieu that influences the development of T cells with particular effector functions.
- APCs detect and take up microorganisms and their antigens, after which they may migrate to lymph nodes and interact with T cells to present antigen.
- In periodontium: APCs are B cells, macrophages, and at least two types of dendritic cells (i.e., dermal dendritic cells and Langerhans cells).
- It is increasingly recognized that the engagement of PRRs (and in particular TLRs) by MAMPs from pathogenic microorganisms is not only central to signaling innate immunity in the form of cytokine up-regulation but also a critical element of the activation of APCs and the elaboration of T-cell effector function.
- Thus, TLR activation increases the expression of molecules on APCs, which are critical to the interaction of these cells with T cells. In addition, TLR activation enhances antigen uptake and processing.

T Cells

- Several different subsets of thymic lymphocytes (i.e., T cells) develop in the bone marrow and thymus and migrate to the peripheral tissues to participate in adaptive immune responses.
- The expression of the cell surface molecules (CD4 or CD8) or particular T-cell antigen receptors (αβ or γδ) broadly defines functional T-cell subsets that emerge from the thymus.
- CD4+ helper T cells are the predominant phenotype in the stable periodontal lesion, and it is thought that alterations in the balance of effector T-cell subsets within the CD4+ population may lead to progression toward a destructive, B-cell–dominated lesion.
- The best-defined functional subsets of CD4+ T cells are the Th1 and Th2 cells, and a dynamic interaction between Th1 and Th2 cells may provide, in part, an explanation for fluctuations in disease activity and the progression of periodontal disease.

stable periodontal lesion *vs.* destructive lesion

strong innate response results in IL-12 synthesis (e.g., by tissue macrophages)

That leads to a Th1 response that provides protective cell-mediated immunity.

Th1 cells secrete IFN-γ, which activates cell-mediated immunity (i.e., macrophages, NK cells, and CD8+ cytotoxic T cells) against pathogenic microorganisms.

Stable periodontal lesion

A poor innate response would lead to reduced IL-12

Permit the development of Th2 responses

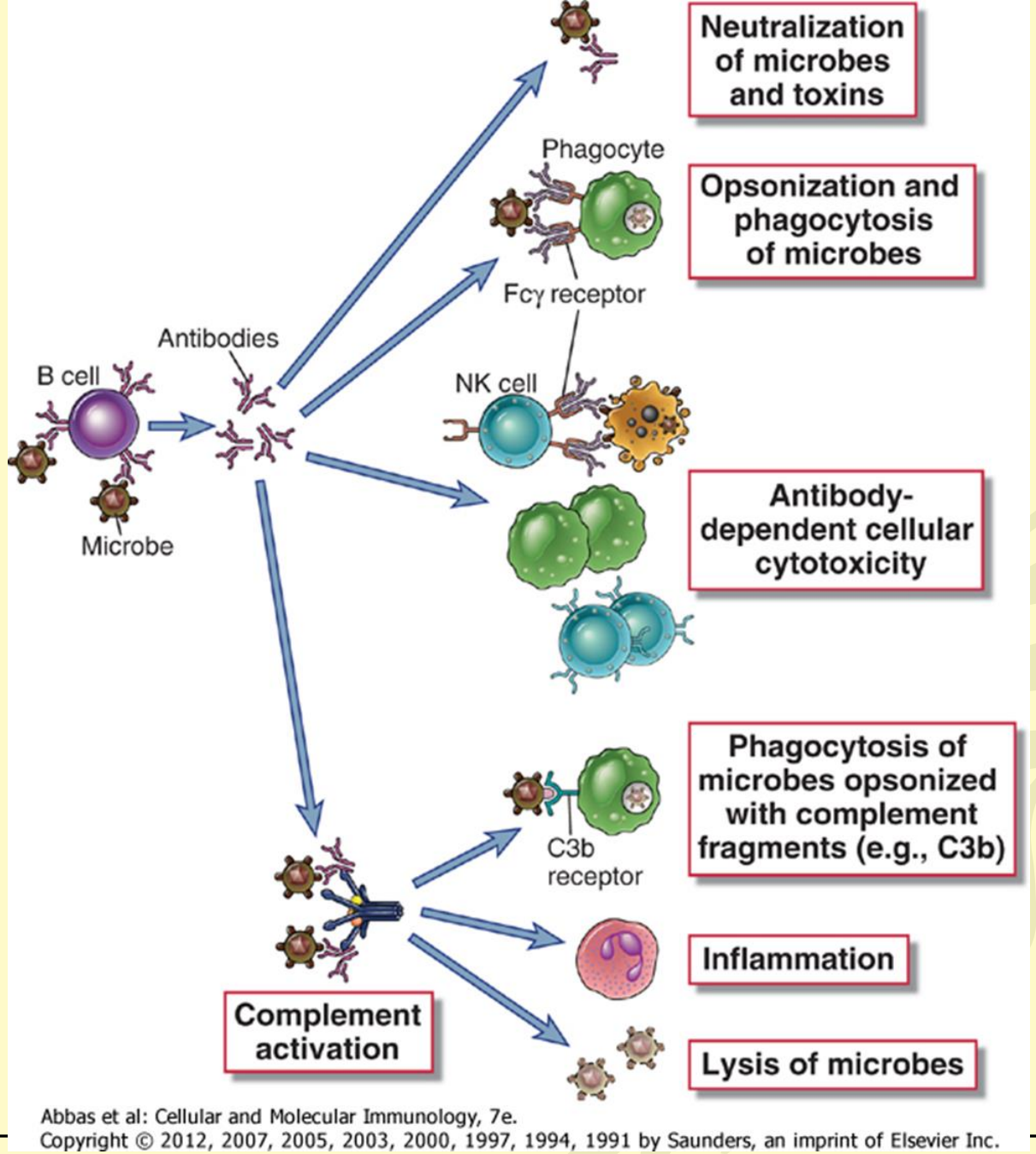
Lead to the activation of B cells;

enhanced B-cell– derived IL-1β.

Destructive lesion

B-cells:

- The production of specific antibodies that would serve to clear tissue infections through:
  - Interaction with the complement system
  - Enhancing neutrophil phagocytosis.
  - Neutralization of microbes and toxins
- B cells are also a source of proinflammatory cytokines that contribute to tissue destruction.

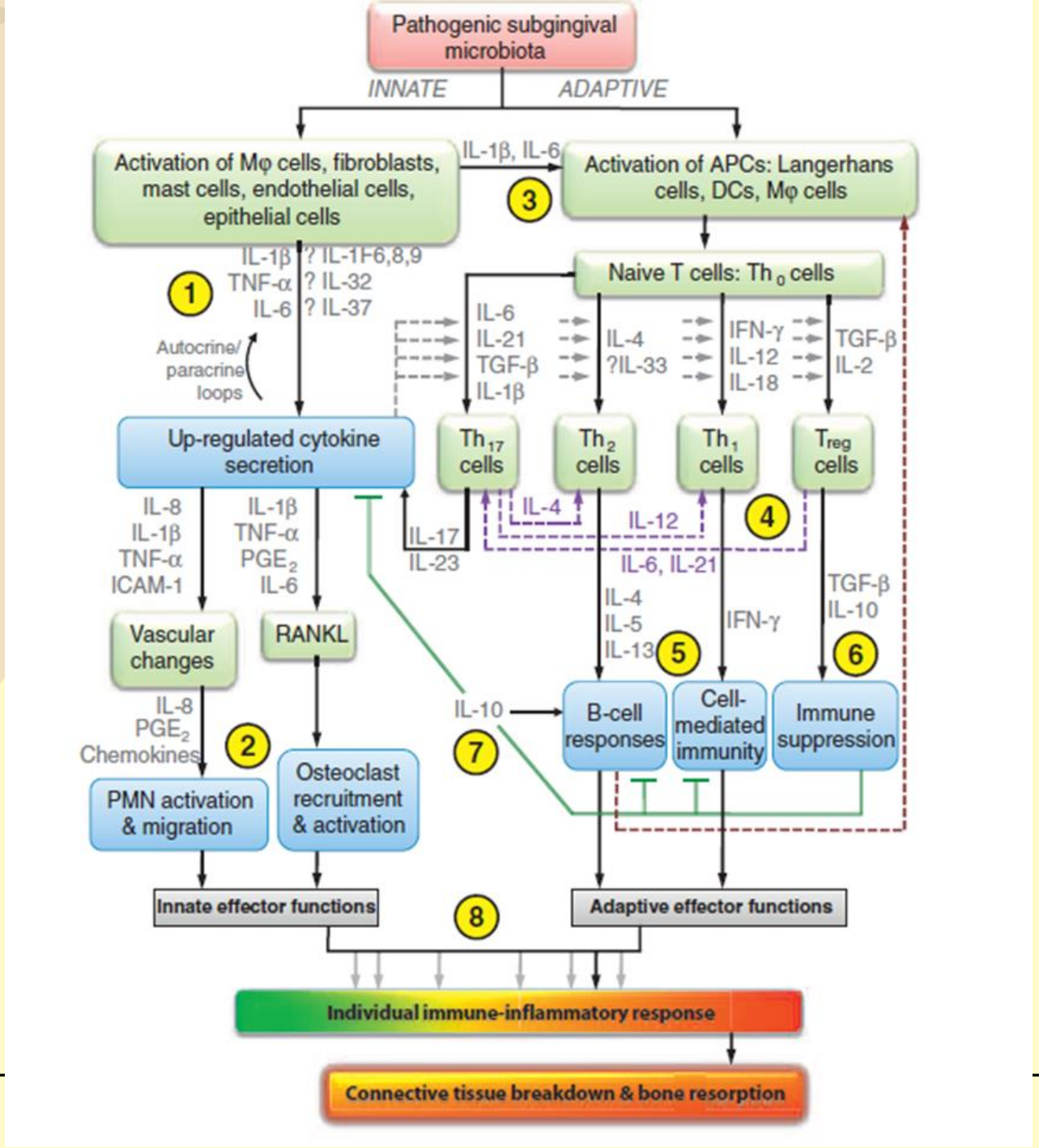


The humoral response to plaque

- Specific antibodies are produced in response to the bacterial challenge in periodontal disease and are the endpoint of B-cell activation.
- Differentiated plasma cells.
- High levels of antibodies appear in GCF (in addition to those in the circulation), and these are produced locally by plasma cells in periodontal tissues.
- Antibodies to periodontal pathogens are primarily IgG, with few IgM or IgA types produced.
- Many species of oral bacteria elicit a polyclonal B-cell response that augment responses against nonoral bacteria and may lead to the production of autoantibodies (e.g., antibodies against collagen and connective tissue proteins), which may contribute to tissue destruction in periodontal disease.

- Variations in the levels of specific antibodies to different species in different clinical presentations suggest differences in pathogenesis.
  - For example, antibodies to A. actinomycetemcomitans of the IgG2 subclass predominate in aggressive periodontitis.
  - Other P. gingivalis molecules (i.e., fimbriae and hemagglutinin) also act as antigens. Specific antibodies are also generated by serotype-specific carbohydrate antigens (e.g., capsular polysaccharide of P. gingivalis, carbohydrate of A. actinomycetemcomitans LPS).

- Individuals with aggressive periodontitis have monocytes that are hyperresponsive to LPS and that produce elevated quantities of PGE2. A. actinomycetemcomitans is commonly associated with aggressive periodontitis, these bacteria trigger cytokines release from monocytes and as a consequence, increase IgG2
- The significance of antibodies in periodontitis is not clear. It is not known whether these antibodies have a protective function or whether they participate in disease pathogenesis.


Pathogenic subgingival microbiota
INNATE
ADAPTIVE
Activation of Mφ cells, fibroblasts, mast cells, endothelial cells, epithelial cells
IL-1β, IL-6
3
Activation of APCs: Langerhans cells, DCs, Mφ cells
IL-1β
TNF-α
IL-6
? IL-1F6,8,9
? IL-32
? IL-37
1
Naive T cells: Th0 cells
Autocrine/ paracrine loops
IL-6
IL-21
TGF-β
IL-1β
IL-4
?IL-33
IFN-γ
IL-12
IL-18
TGF-β
IL-2
Up-regulated cytokine secretion
Th17 cells
Th2 cells
Th1 cells
Treg cells
IL-8
IL-1β
TNF-α
ICAM-1
IL-1β
TNF-α
PGE2
IL-6
IL-17
IL-23
IL-4
IL-12
4
IL-6, IL-21
IL-4
IL-5
IL-13
IFN-γ
TGF-β
IL-10
5
6
Vascular changes
RANKL
IL-8
PGE2
Chemokines
2
IL-10
7
B-cell responses
Cell-mediated immunity
Immune suppression
PMN activation & migration
Osteoclast recruitment & activation
Innate effector functions
8
Adaptive effector functions
Individual immune-inflammatory response
Connective tissue breakdown & bone resorption

Osteo-immunology in periodontal diseases

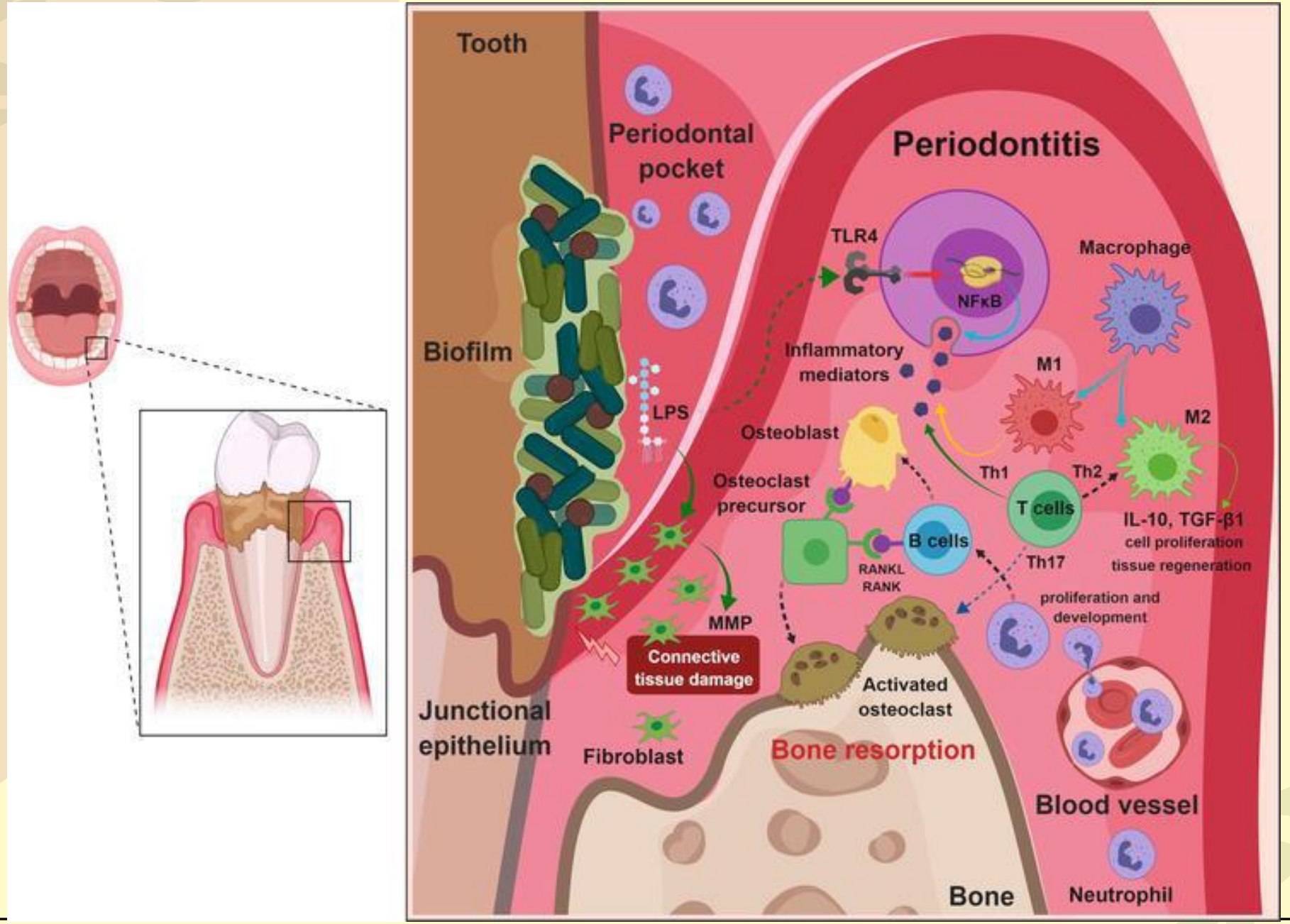
- As the advancing inflammatory front approaches the alveolar bone, osteoclastic bone resorption commences. This is a protective mechanism to prevent bacterial invasion of the bone, but it ultimately leads to tooth mobility and even tooth loss. The resorption of alveolar bone occurs simultaneously with the breakdown of the periodontal ligament in the inflamed periodontal tissues. Two critical factors determine whether bone loss occurs:
- (1) the concentration of inflammatory mediators in the gingival tissues must be sufficient to activate the pathways that lead to bone resorption.
- (2) the inflammatory mediators must penetrate to within a critical distance of the alveolar bone.

- Histologic studies have confirmed that the bone resorbs so that a width of non-infiltrated connective tissue of about 0.5 to 1.0 mm overlying the bone is always present. It has also been demonstrated that bone resorption ceases when at least a 2.5-mm distance is present between the site of bacteria in the pocket and the bone.
- Osteoclasts are stimulated by proinflammatory cytokines and other mediators of inflammation to resorb the bone, and the alveolar bone “retreats” from the advancing inflammatory front.
- Osteoclasts are multinucleated cells that are formed from osteoclast progenitor cells and macrophages. Osteoclastic bone resorption is activated by a variety of mediators (e.g., IL-1β, TNF-α, IL-6, PGE2).

Receptor Activator of Nuclear factor-Kappa beta (RANK)

- Osteoclast can be also activated by RANK which is expressed by osteoclast progenitor cells. This receptor is activated by RANK Legand (RANKL) which is a member of the TNF family.
- RANKL is produced by osteoblasts and stromal cells of the bone marrow. It is also produced by T-cells and other inflammatory cells. When RANKL is attached to RANK the process of differentiation of progenitor cells into active osteoclast will be initiated.
- Osteoprotegrin (OPG) is another member of the TNF family, however, when it binds to RANKL; it inhibits its activity.
- In patients with periodontitis, it is detected that the GCF contains reduced OPG levels and elevated levels of RANKL.
- The process of bone destruction is the imbalance between osteoclast activating and inhibiting cytokines

- TNF-α and IL-1 trigger bone resorption by controlling the osteoclast formation and keeping it in a continuous increase.
- IL-1 also affects the osteoclastogenic effect of TNF-α by reinforcing the differentiation of osteoclast precursors and the expression of RANKL.
- Cytokines like IL-1β play a vital role as it induces RANKL expression in various cells which include osteoblasts, periodontal ligament fibroblasts, and gingival fibroblasts.
- In the same way, IL-6 is produced by different cells such as osteoblasts and fibroblasts. It plays an extremely important role in inducing osteoclast formation and promoting bone resorption.
- In addition, the inflammatory mediator prostaglandin E2 is very necessary for this process. It triggers bone resorption by the upregulation of RANKL expression and the inhibition of OPG expression in osteoblastic cells.


Tooth
Periodontal pocket
Periodontitis
TLR4
NFκB
Macrophage
Biofilm
Inflammatory mediators
M1
LPS
Osteoblast
M2
Th1
Th2
Osteoclast precursor
T cells
IL-10, TGF-β1
cell proliferation
tissue regeneration
B cells
RANKL
RANK
Th17
proliferation and development
MMP
Connective tissue damage
Activated osteoclast
Junctional epithelium
Fibroblast
Bone resorption
Blood vessel
Bone
Neutrophil

How does a pocket develop?

- The bacterial biofilm causes inflammation in the gingival tissues that causes swelling, and therefore the sulcus deepens slightly (false pocket).
- The inflammatory response may subsequently spread to the deeper tissues and is characterized by infiltration by defense cells and breakdown of collagen in the connective tissues.
- The junctional epithelium migrates apically to maintain an intact epithelial barrier, and thus the sulcus becomes deeper again and is now referred to as a pocket.
- Bacteria in the biofilm proliferate apically, exploiting and perpetuating this environmental niche.
- The bacteria are never completely eradicated by the host response, and thus they continue to provoke an immune–inflammatory response, leading to progressing tissue breakdown, continued apical migration of the junctional epithelium, resorption of alveolar bone, and gradual deepening of the pocket

Resolution of Inflammation

- Inflammation is an important defense mechanism to combat the threat of bacterial infection, but inflammation also results in tissue damage associated with the development and progression of most chronic diseases associated with aging, including periodontal disease.
- Resolution of inflammation (i.e., “turning off” inflammation) is an active process that is regulated by specific mechanisms that restore homeostasis.
- It is possible that controlling or augmenting these mechanisms may lead to the development of new treatment strategies for managing chronic diseases such as periodontitis.
- It is mediated by specific molecules, including a class of endogenous, proresolving lipid mediators that includes the lipoxins, resolvins, and protectins. These molecules are actively synthesized during the resolution phases of acute inflammation;
    - they are antiinflammatory, and they inhibit neutrophil infiltration. They are also chemoattractants, but they do not cause inflammation. For example, lipoxins stimulate infiltration by monocytes but without stimulating the release of inflammatory cytokines.

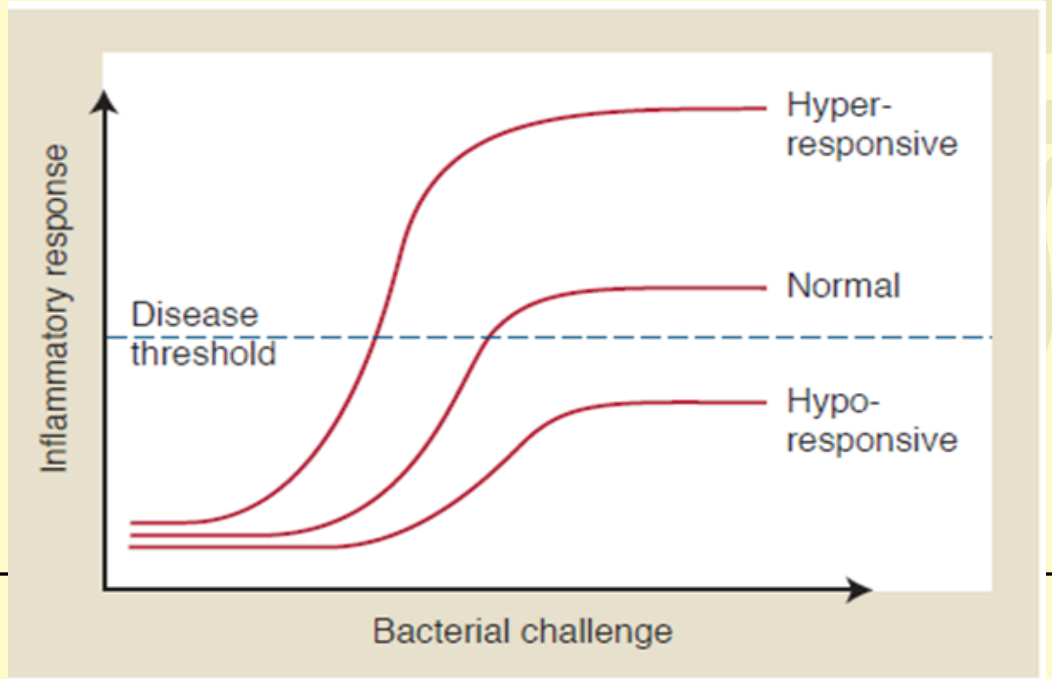
Individual variations in response to bacterial plaque

- Immune responses to the bacterial challenge do not occur in isolation, but rather take place in the context of other host and environmental factors that influence these responses and thereby determine the progression of disease.
- Certain risk factors increase susceptibility to periodontal disease, particularly smoking and diabetes.
- Quantitative and qualitative differences exist in immune responses among individuals. This could be due to infectious agents (e.g., bacteria) exert evolutionary selection pressures on the species that they infect.

“hyperinflammatory” or “hyporesponsive” trait

- Some studies have confirmed that immune cells from patients with periodontal disease secrete higher quantities of proinflammatory cytokines than do cells from persons who are periodontally healthy.
- Cytokine profiles are also different in those individuals with immune-mediated diseases as compared with healthy control subjects.
- These observations have led to the trait concept in which certain individuals possess a hyperinflammatory phenotype that accounts for their increased susceptibility to chronic inflammatory conditions such as periodontitis.
- Such a trait may also underpin shared susceptibility between conditions such as periodontitis and cardiovascular disease or diabetes.
- at present, it is not possible to identify with certainty those patients who are hyper-responders.

- It is likely that many reasons contribute to disease variations among individuals, such as variations in immune responses, pathogenesis, and the plaque biofilm
- This situation results in an uneven disease experience in the population.
- although plaque bacteria initiate the inflammatory response, most of the tissue damage results from the host response, which is influenced by genetic factors, as well as environmental and acquired risk factors.
- Risk factors such as smoking alter the progression of the immune–inflammatory response and shift the balance toward increased periodontal breakdown



Therapeutic Strategies

- Various treatment strategies have been developed to target the host response.
- Matrix metalloproteinase (MMP) inhibitors (e.g., low-dose formulations of doxycycline) have been used in combination with scaling and root planing or surgical therapy.
- High-risk patient populations (e.g., diabetic patients, patients with refractory periodontal disease) have benefited from the systemic administration of MMP inhibitors.
- Soluble antagonists of TNF-α and IL-1 can be delivered locally to periodontal tissues in nonhuman primates.
- Other therapeutic strategies are aimed at inhibiting the signal transduction pathways involved in inflammation. Inflammatory mediators including proinflammatory cytokines (e.g., IL-1, TNF, IL-6), MMPs, and others would be inhibited at the level of the cell-signalling pathways required for the transcription factor activation necessary for inflammatory gene expression or mRNA stability.
- Novel analogues of defensins have shown even higher antibacterial activity than the endogenous -defensins 1 and 3, without any cytotoxic effects on host cells, thus indicating the promise of this approach.

Thank you

Interrelationship between Periodontics and other parts of dentistry

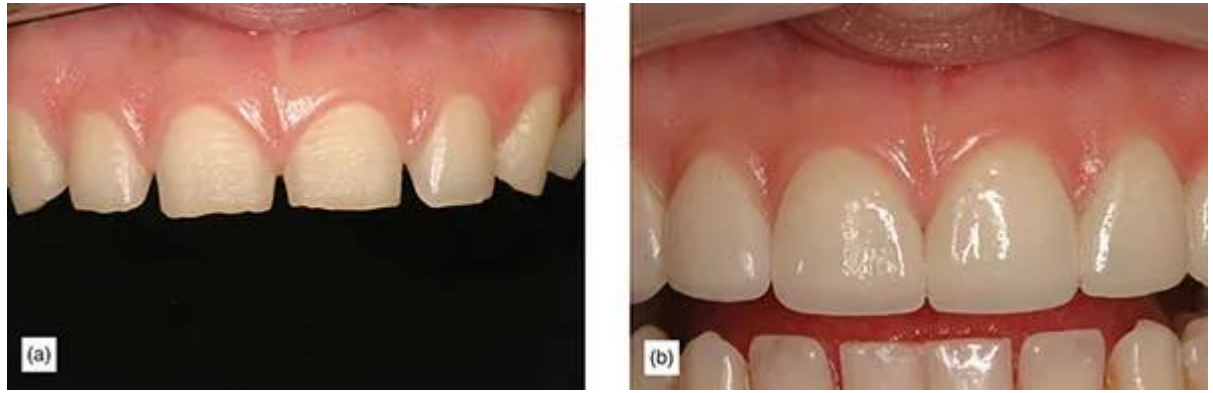
Dr Hadeel Mohammed Abbood

Restorative interrelationship

Biologic Considerations
Margin Placement and Biologic Width

- One of the most important aspects of understanding the periodontal–restorative relationship is the location of the restorative margin to the adjacent gingival tissue.
- Restorative clinicians must understand the role of biologic width:
  - in preserving healthy gingival tissues
  - controlling the gingival form around restorations.
- They must also apply this information in the positioning of restoration margins, especially in the aesthetic zone, where a primary treatment goal is to mask the junction of the margin with the tooth.
- A clinician is presented with three options for margin placement:
  - Supragingival,
  - Equigingival (even with the tissue),
  - And subgingival.

- The **supragingival margin** has the least impact on the periodontium. Classically,
- This margin location can be applied in unaesthetic areas because of the marked contrast in color and opacity of traditional restorative materials against the tooth.
- With the advent of more translucent restorative materials, adhesive dentistry, and resin cements, the ability to place supragingival margins in aesthetic areas is now a reality. Therefore, whenever possible, these restorations should be chosen not only for their aesthetic advantages but also for their favorable periodontal impact.



- The use of **equigingival margins** traditionally was not desirable because:
  - They were thought to retain more plaque than supragingival or subgingival margins and therefore resulted in greater gingival inflammation.
  - There was also the concern that any minor gingival recession would create an unsightly margin display.
- These concerns are not valid today, not only because the restoration margins can be aesthetically blended with the tooth, but also because restorations can be finished easily to provide a smooth, polished interface at the gingival margin.

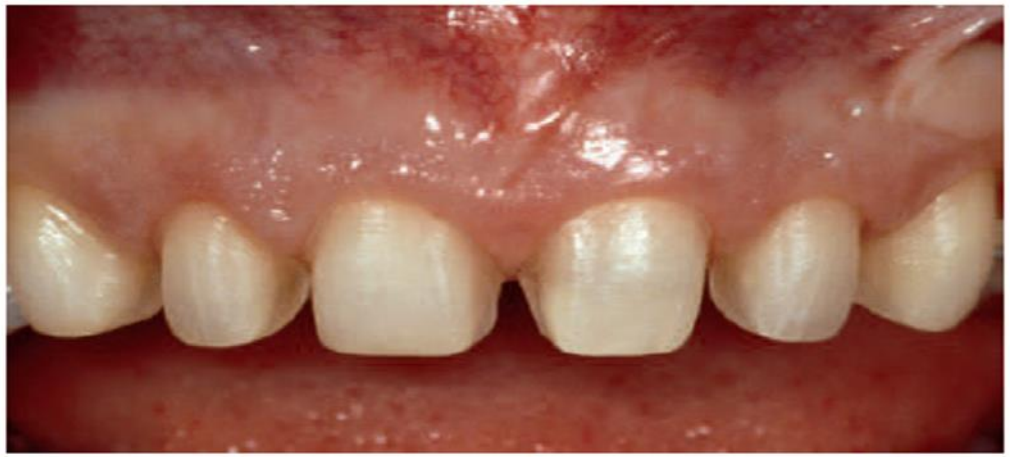


Fig. 70.1 With the advent of adhesive dentistry and ultrathin ceramic veneers, it now is possible to prepare restorations equigingival without visible margins. The preparations for six porcelain veneers with the margins placed at the level of tissue are shown.

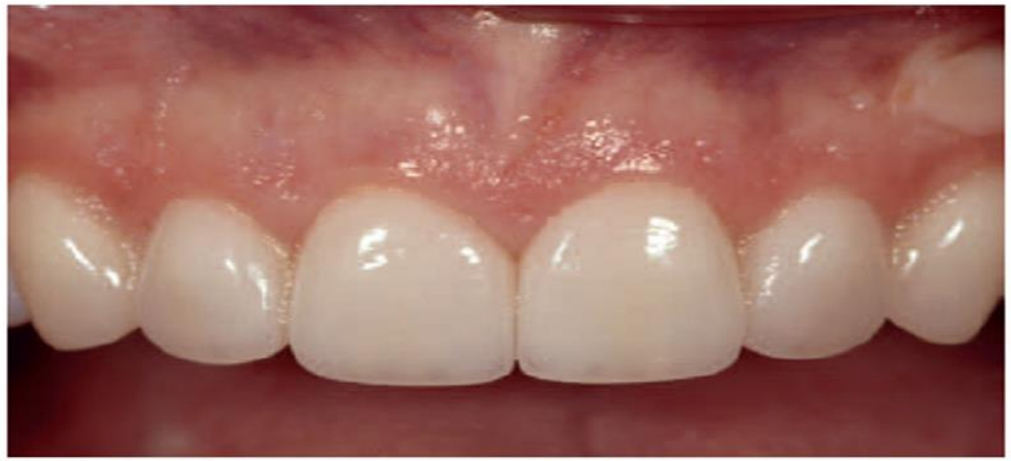


Fig. 70.2 The completed veneers from Fig. 70.1. Note the invisible gingival finish line, even though the margin has not been carried below tissue.

subgingival margins

- From a periodontal viewpoint, both supragingival and equigingival margins are well tolerated.
- The greatest biologic risk occurs when placing *subgingival margins.*

  Why?

  - These margins are not as accessible as supragingival or equigingival margins for finishing procedures.
  - In addition, if the margin is placed too far below the gingival tissue crest, it violates the gingival attachment apparatus.

Restorations may need to be extended gingivally:

- (1) to create adequate resistance and retentive form in the preparation,
- (2) to make significant contour alterations because of caries or other tooth deficiencies,
- (3) to mask the tooth–restoration interface by locating it subgingivaly, or
- (4) to lengthen the tooth for aesthetic reasons.

biologic width

- The dimension of space that the healthy gingival tissues occupy between the base of the sulcus and the underlying alveolar bone is composed of the junctional epithelial attachment and the connective tissue attachment. The combined attachment width is now identified as t*he biologic width.*
  - In the average human, the connective tissue attachment occupies 1.07 mm of space above the crest of the alveolar bone
  - The junctional epithelial attachment below the base of the gingival sulcus occupies another 0.97 mm of space above the connective tissue attachment.
  - The combination of these two measurements, averaging approximately 1 mm each, constitutes the biologic width.
- Clinically, this information is applied to diagnose biologic width violations when the restoration margin is placed 2 mm or less away from the alveolar bone and the gingival tissues are inflamed with no other etiologic factors evident.

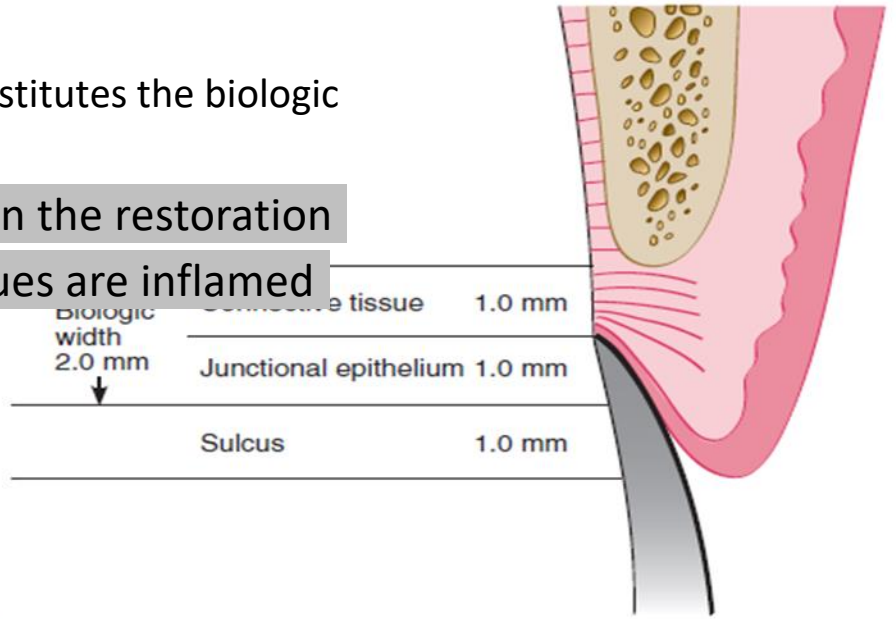
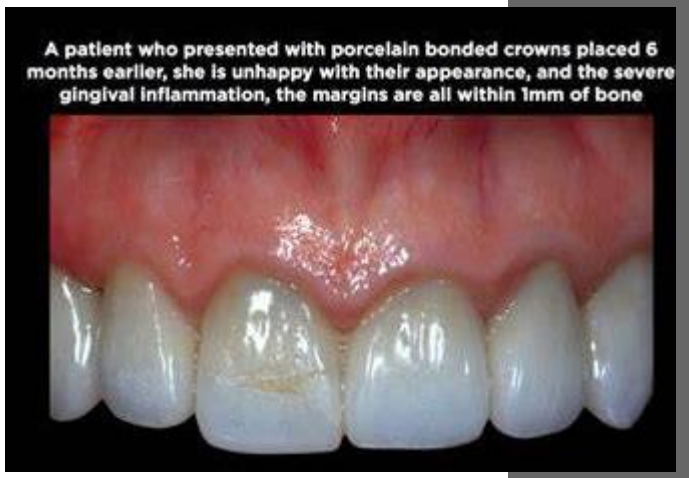
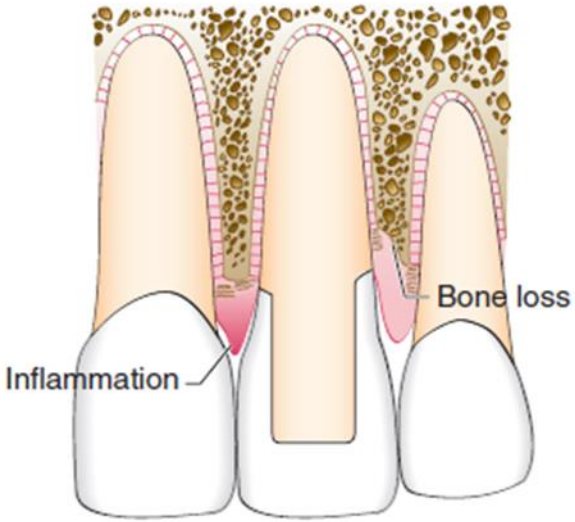


Fig. 70.3 Average human biologic width: connective tissue attachment 1 mm in height; junctional epithelial attachment 1 mm in height; sulcus depth of approximately 1 mm. The combined connective tissue attachment and junctional epithelial attachment, or biologic width, equals 2 mm.

violation of the biologic width

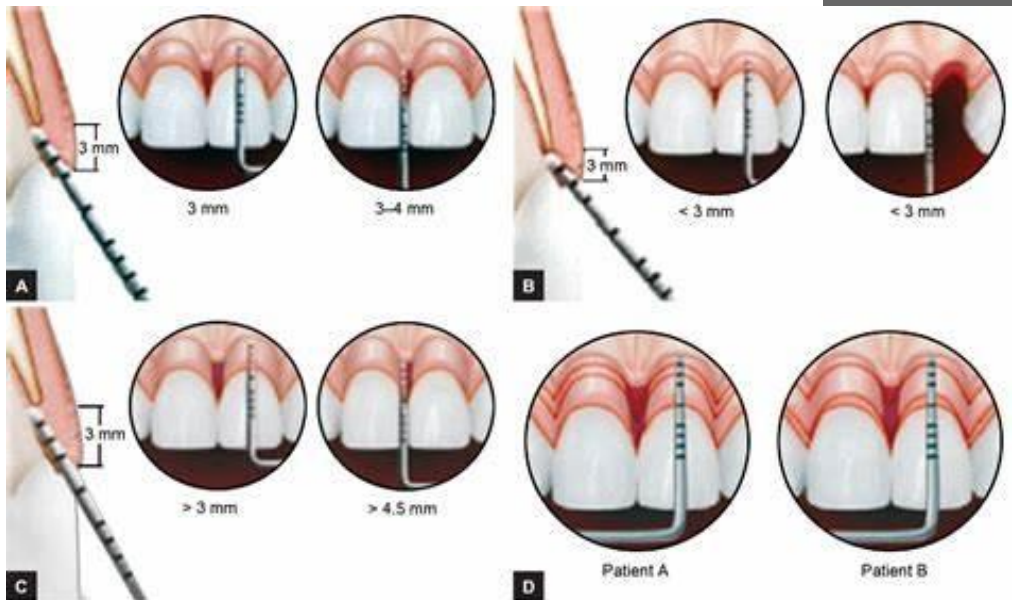


- When the restoration margin is placed too far below the gingival tissue crest, it impinges on the gingival attachment apparatus and creates a violation of biologic width.
- Two different responses can be observed from the involved gingival tissues.
  A. One possibility is that bone loss of an unpredictable nature and gingival tissue recession occurs as the body attempts to re-create room between the alveolar bone and the margin to allow space for tissue reattachment.
  B. Inflammation of the gingiva
- Factors that may impact the likelihood of recession include:
  I. The alveolar bone surrounding the tooth is very thin in width.
  II. whether the gingiva is thin and fragile
  III. whether the periodontium is highly scalloped



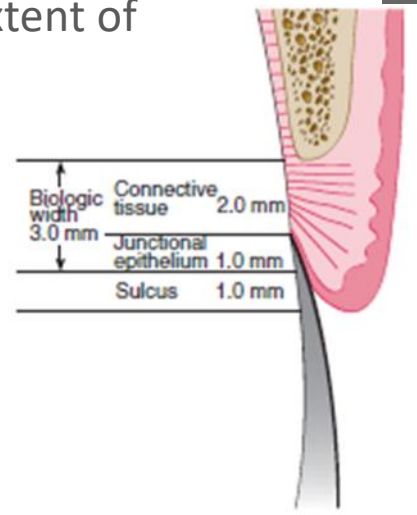
Biologic width evaluation

- **Radiographic** interpretation can identify interproximal violations of biologic width.
  - superimposition.
- **Clinical evaluation**
- If a patient experiences tissue discomfort when the restoration margin levels are being assessed with a periodontal probe, it is a good indication that the margin extends into the attachment and that a biologic width violation has occurred.
- A more positive assessment can be made clinically by measuring the distance between the bone and the restoration margin using a sterile periodontal probe. The probe is pushed through the anesthetized attachment tissues from the sulcus to the underlying bone. If this distance is less than 2 mm at one or more locations, a diagnosis of biologic width violation can be confirmed.
- Circumferentially around the tooth to evaluate the extent of the problem.



Normal variation

- Biologic width violations can occur in some patients in whom the margins are located more than 2 mm above the alveolar bone level.
- A range of different biologic width was reported; 0.75 mm to 4.3 mm.
- This information dictates that specific biologic width assessment should be performed for each patient to determine if the patient needs additional biologic width, in excess of 2 mm, for restorations to be in harmony with the gingival tissues.
- The biologic width can be identified for the individual patient by probing to the bone level (referred to as “sounding to bone”) and subtracting the sulcus depth from the resulting measurement.
- This measurement must be done on teeth with healthy gingival tissues and should be repeated on more than one tooth to ensure an accurate assessment.
- The information obtained is then used for definitive diagnosis of biologic width violations, the extent of correction needed, and the parameters for placement of future restorations.

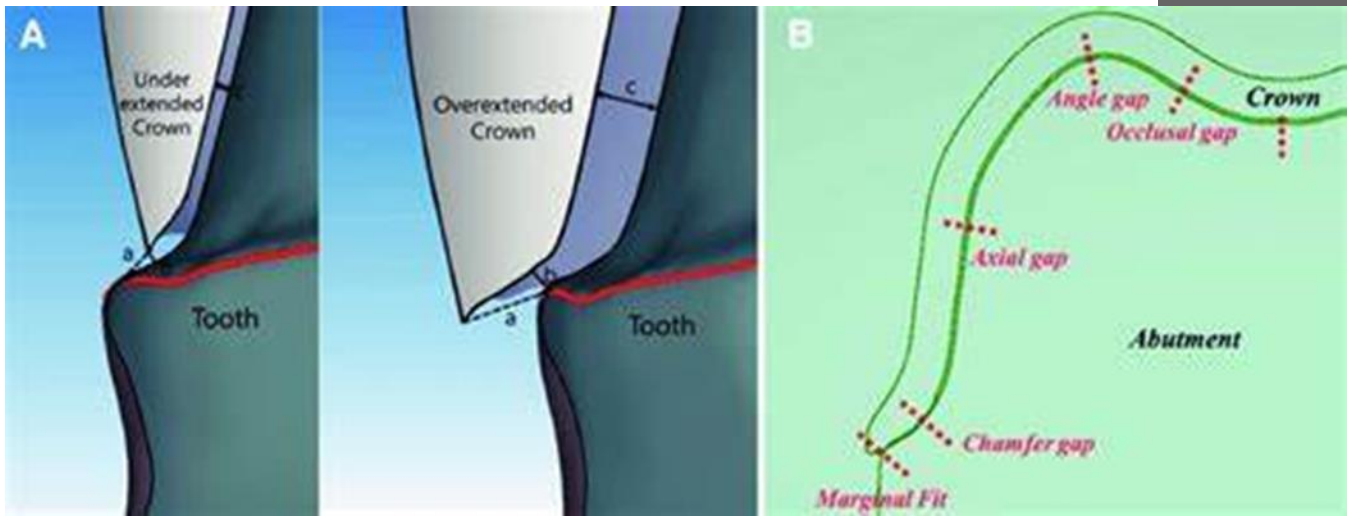


Margin Placement Guidelines

- It is recommended that the patient's existing sulcular depth be used as a guideline in assessing the biologic width requirement for that patient.
- The base of the sulcus can be viewed as the top of the attachment,
- ensuring that the margin is placed in the sulcus and not in the attachment. The variations in sulcular probing depth are then used to predict how deep the margin can safely be placed below the gingival crest.
- With shallow probing depths (1 to 1.5 mm), extending >0.5 mm subgingivally risks violating the attachment..
- With shallow probing depths, future **recession is unlikely** because the free gingival margin is located close to the top of the attachment.
- Deeper sulcular probing depths provide more freedom in locating restoration margins farther below the gingival crest. In most circumstances, however, **the deeper the gingival sulcus, the greater is the risk of gingival recession.**
- Locating the restorative margin deep subgingivally should be avoided:
  a. It increases the difficulty in making an accurate impression,
  b. finishing the restoration margins,
  c. Increases the likelihood of inflammation and recession.

Marginal Fit

- Marginal fit has clearly been implicated in producing an inflammatory response in the periodontium.
- It has been shown that the level of gingival inflammation can increase corresponding with the level of marginal opening. Margins that are significantly open (several tenths of a millimeter) are capable of harboring large numbers of bacteria and may be responsible for the inflammatory response seen.
- However, the quality of **marginal finish and the margin location** relative to the attachment are much more critical to the periodontium than the difference between a 20-µm fit and a 100-µm fit.



Crown Contour

- Restoration contour has been described as extremely important to the maintenance of periodontal health.
- Ideal contour provides access for hygiene, has the fullness to create the desired gingival form, and has a pleasing visual tooth contour in aesthetic areas.(Describe ideal crown contour?)
- overcontouring crowns leads to and gingival inflammation, whereas undercontouring produces no adverse periodontal effect.
- The most frequent cause of overcontoured restorations is inadequate tooth preparation by the dentist, which forces the technician to produce a bulky restoration to provide room for the restorative material.
- In areas of the mouth in which aesthetic considerations are not critical, a flatter contour is always acceptable.

Aesthetic Tissue Management

Managing Interproximal Embrasures

- Current restorative and periodontal therapy must consider a good aesthetic result, especially in the “aesthetic zone.” the interproximal papilla is an important part in creating this aesthetic result.
- The interproximal embrasure created by restorations and the form of the interdental papilla have a unique and intimate relationship.
- **The ideal interproximal embrasure** should house the gingival papilla without impinging on it and should also extend the interproximal tooth contact to the top of the papilla so that no excess space exists to trap food and to be aesthetically displeasing.
- **Papillary height** is established by the level of the bone, the biologic width, and the form of the gingival embrasure. Changes in the shape of the embrasure can impact the height and form of the papilla. The tip of the papilla behaves differently than the free gingival margin on the facial aspect of the tooth. Whereas the **free gingival margin averages 3 mm** above the underlying facial bone, **the tip of the papilla averages 4.5 to 5 mm** above the interproximal bone. This means that if the papilla is farther above the bone than the facial tissue but has the same biologic width, the interproximal area will have a sulcus 1 to 1.5 mm deeper than that found on the facial surface.

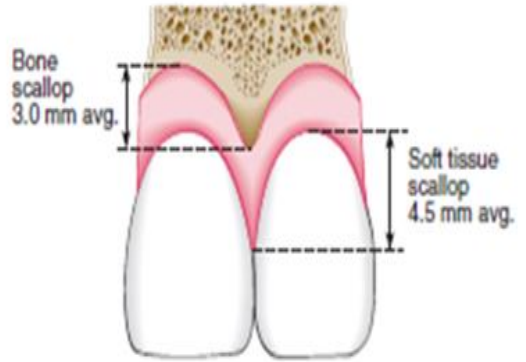


Fig. 70.6 Comparison of the behavior of the interproximal papilla relative to bone and the free gingival margin relative to bone in the average human. There is a 3-mm scallop from the facial bone to the interproximal bone. However, on average, a 4.5- to 5-mm gingival scallop exists between the facial tissue height and the interproximal papilla height. This extra scallop of 1.5 to 2 mm of gingiva compared with bone is the result of the extra soft-tissue height above the attachment interproximally.

CLINICAL CORRELATION

- If you create restorations with less than 5 mm from the contact to the bone, open gingival embrasures can be avoided. The downside to this approach is that the teeth will look square and blocky.
- Complete removal of healthy papillae to the bone level can routinely regenerated 4 to 4.5 mm of total tissue above bone, with an average sulcus depth of 2 to 2.5 mm.

Chance of complete papilla fill

- When the distance from the interproximal bone to the interproximal contact of the teeth measured 5 mm or less, 98% of these sites had complete papilla fill.
- When the distance was 6 mm, only 56% of the sites had complete papilla fill.
- When the distance was 7 mm, only 27% of the sites had complete papilla fill.

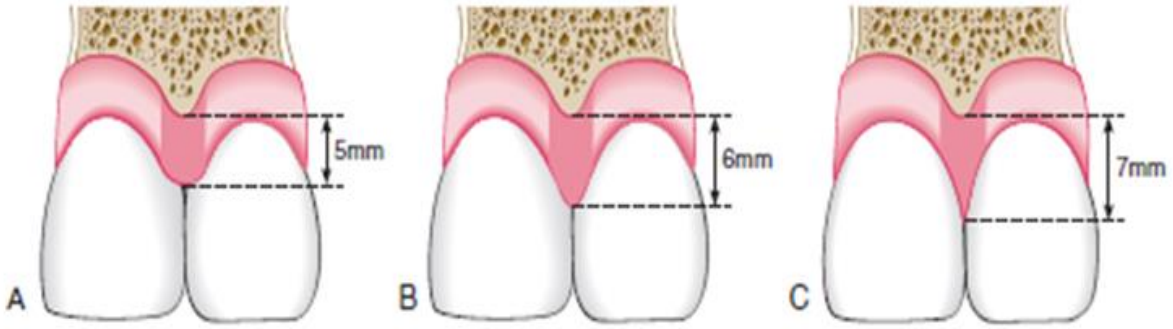


Fig. 70.7 The probability of complete fill of gingival embrasure by papilla. (A) With 5 mm from crest of bone to the apical contact point, there is a 98% chance of complete fill of the space. (B) At 6 mm from crest to contact, the chance of filled embrasure drops to 56%. (C) At 7 mm from crest to contact, the chance of complete fill drops to 27%.

Assessing open embrasure

- it is imperative that tissue be healthy and mature. Performing the analysis on inflamed or immature tissues will result in supragingival margins when the tissues heal.
- If the papillary sulcus measures greater than 3 mm, there is some risk of recession with restorative procedures.

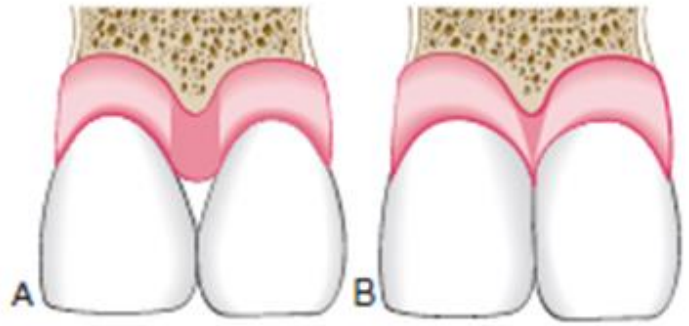
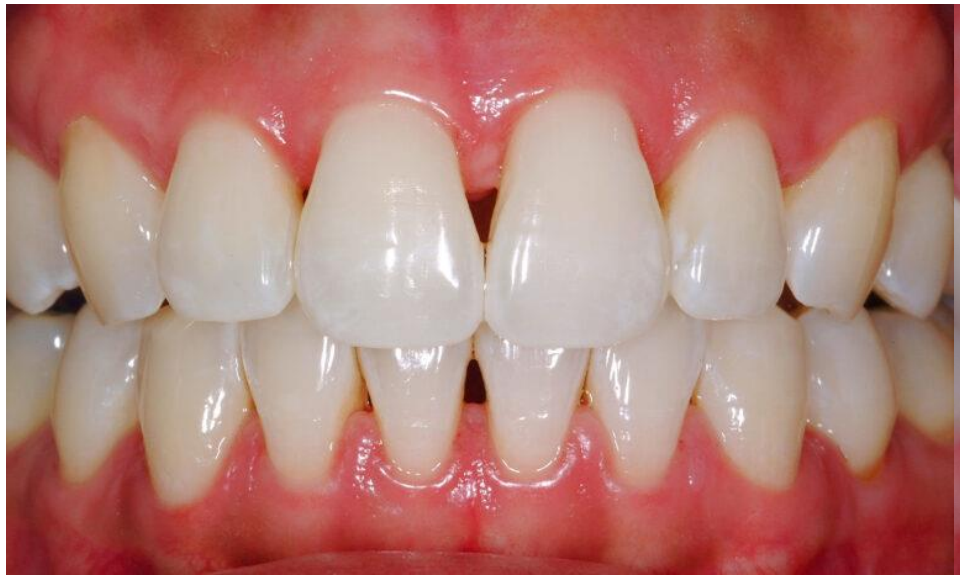
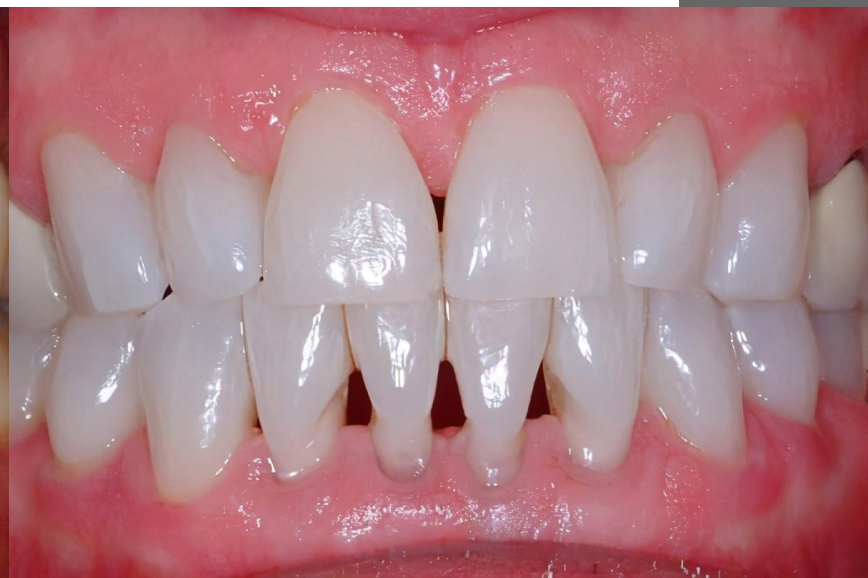


Fig. 70.8 Relationship between gingival embrasure volume and papillary form. (A) Gingival embrasure of the teeth is excessively large as the result of a tapered tooth form. Because of the large embrasure form, the volume of tissue sitting on top of the attachment is not molded to the shape of a normal papilla but rather has a blunted form and a shallower sulcus. (B) Ideal tooth form in which the same volume of tissue sits on top of the attachment as in part A. Because of the more closed embrasure form from the teeth in part B, however, the papilla completely fills the embrasure and has a deeper sulcus, averaging 2.5 to 3 mm. Note that the ideal contact position is 3 mm coronal to the attachment.

- The papilla in question is compared with the adjacent papillae. If the papillae are all on the same level, and if the other areas do not have open embrasures, the problem is one of gingival embrasure form. Restorative correction
- If the papilla in the area of concern is apical to the adjacent papillae, however, the clinician should evaluate the interproximal bone levels.
  - If the bone under that papilla is apical to the adjacent bone levels, the problem is caused by bone loss. Periodontal treatment
  - If the bone is at the same level, the open embrasure is caused by the embrasure form of the teeth and not a periodontal problem with the papilla. Restorative correction





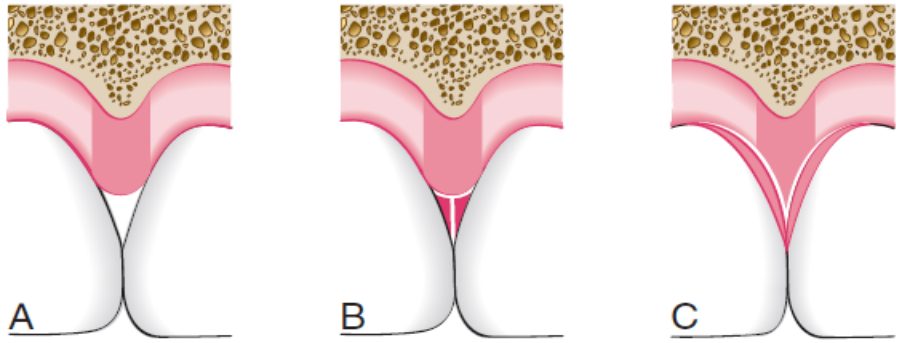
Open Gingival Embrasures

- This open space is usually caused by:
- (1) tooth shape,
- (2) root angulation, or
- (3) periodontal bone loss.
- The first step in the diagnosis of this problem is to evaluate a periapical radiograph of the central incisors. Correction of open gingival embrasure can be in different ways. Periodontal surgery, orthodontic treatment or restorations.

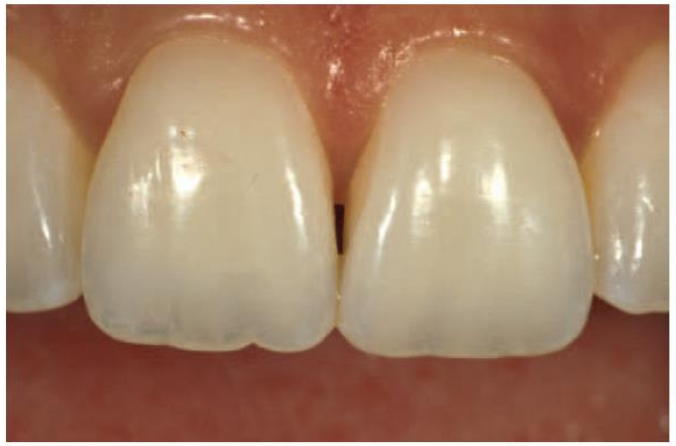
Correcting Open Gingival Embrasures **Restoratively**

Restorative dentistry can correct this problem by moving the contact point to the tip of the papilla. To accomplish this, the margins of the restoration must be carried subgingivally 1 to 1.5 mm, and the emergence profile of the restoration is designed to move the contact point toward the papilla while blending the contour into the tooth below the tissue.

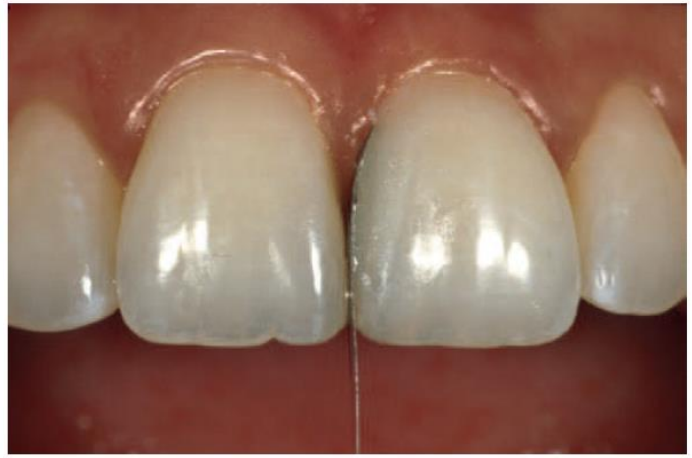
This can be accomplished easily with direct bonded restorations



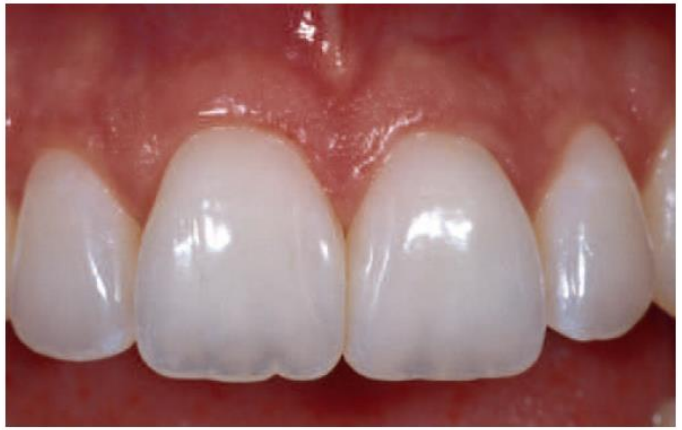
eFig. 70.26 Methods of altering gingival embrasure form. (A) Typical open gingival embrasure caused by excessively tapered tooth form. (B) Common method employed by restorative dentists to correct the embrasure, in which material is added supragingivally. This closes the embrasure by moving the contact to the tip of the papilla but results in overhangs that cannot be cleaned using dental floss. Removing these overhangs restoratively reopens the embrasure. (C) Correct method of closing the gingival embrasure, in which the margins of the restoration are carried 1 to 1.5 mm below the tip of the papilla. Note that this does not encroach on the attachment because the average interproximal sulcus probes 2.5 to 3 mm. This allows easy cleaning because of the convex profile. It also reshapes the papilla to a more pleasing profile aesthetically.



eFig. 70.27 This patient has parallel roots, has recently completed orthodontic therapy, and is unhappy with the open gingival embrasure between her central incisors. An evaluation of papillary height reveals that all are at an equal level. This can only mean that the open embrasure is the result of an overly tapered tooth form (see eFigs. 70.28 and 70.29).



eFig. 70.28 One method of correctly altering tooth form of the patient in eFig. 70.27. A metal matrix band has been shaped to the desired tooth form and placed 1 to 1.5 mm below the tip of the papilla. Restorative material then was added to the tooth against the matrix band, forming the new mesial surface of the left central incisor.



eFig. 70.29 One-year recall photograph after restoring the mesial surfaces of the right and left central incisors, moving the proximal contact to the tip of the papilla and extending the restorations 1 to 1.5 mm below the papilla, blending them into the tooth and making an easily cleaned area (see eFigs. 70.27 and 70.28).

Pontic Design

Classically, there are four options to consider in evaluating pontic design: hygienic, ridge lap, modified ridge lap, and ovate designs.

Regardless of design, the pontic should provide:

I. An occlusal surface that stabilizes the opposing teeth,

II. Allows for normal mastication

III. Does not overload the abutment teeth.

The area of the pontic interfacing with the gingiva can be porcelain, metal, zirconia, lithium disilicate, or some other material with no variation in the biologic response of the tissue provided it has a smooth surface finish.

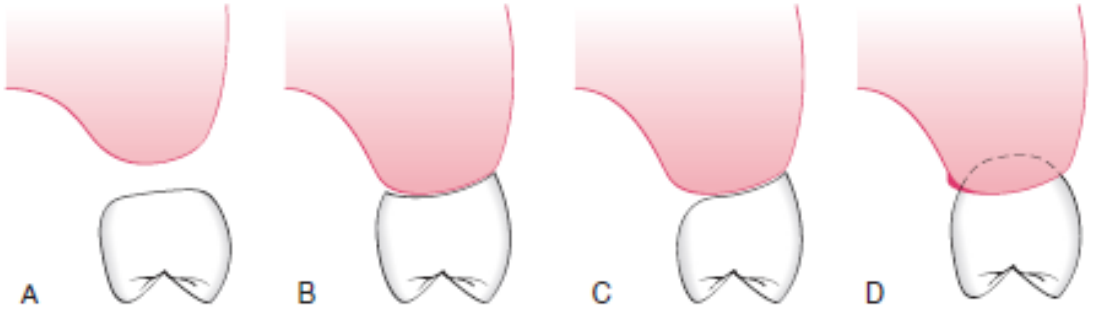


Fig. 70.9 Four options to designing the shape of a pontic. (A) Hygienic pontic. Tissue surface of the pontic is 3 mm from the underlying ridge. (B) Ridge-lap pontic. Tissue surface of the pontic straddles the ridge in saddle-like fashion. The entire tissue surface of the ridge-lap pontic is convex and very difficult to clean. (C) Modified ridge-lap pontic. Tissue surface on the facial is concave, following the ridge. However, the lingual saddle has been removed to allow access for oral hygiene. (D) Ovate pontic. The pontic form fits into a receptor site within the ridge. This allows the tissue surface of the pontic to be convex and also optimizes aesthetics.

- The key differences between the four pontic designs relate to the aesthetics and access for hygiene procedures.
- The primary method for cleaning the undersurface of pontics is to draw dental loss mesiodistally along the undersurface. The shape of this undersurface determines the ease with which plaque and food debris can be removed in the process. The hygienic and ovate pontics have convex undersurfaces, which makes them easiest to clean. The ridge lap and modified ridge-lap designs have concave surfaces, which are more difficult to access with the dental floss. though the hygienic pontic design provides the easiest access for hygiene procedures, it is much less aesthetic and objectionable by some patients.

The ovate pontic

- is the ideal pontic form, particularly in areas of aesthetic concern. It is created by forming a receptor site in the edentulous ridge with a diamond bur, electrosurgery, pressure, or wound healing.
- The site is shaped to create either a flat or a concave contour so that when the pontic is created to adapt to the site, it will have a flat or convex outline. The depth of the receptor site depends on the aesthetic requirements of the pontic.

- In highly aesthetic areas such as the maxillary anterior region, it is necessary to create a receptor area that is 1 to 1.5 mm below the tissue on the facial aspect.
- This creates the appearance of a free gingival margin and produces optimal aesthetics. This site can then be tapered to the height of the palatal tissue to facilitate hygiene access from the palatal side.
- In the posterior areas, a deep receptor site can complicate hygiene access. In these situations, the ideal site has the facial portion of the pontic at the same level as the ridge, and then the site is created as a straight line to the lingual side of the pontic.
- This removes the convexity of the ridge and produces a flat, easily cleanable tissue surface on the pontic

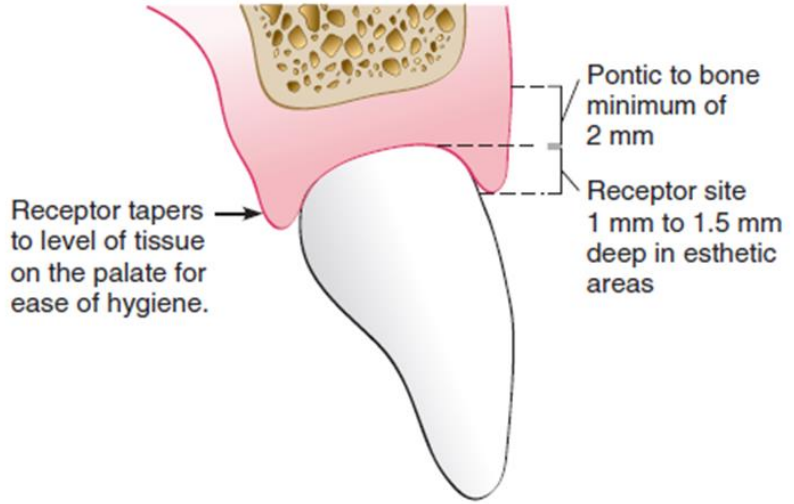


Fig. 70.10 Ideal shape and form of an ovate pontic in the aesthetic area. The receptor site has been created 1 to 1.5 mm apical to the free gingival margin on the facial aspect. This creates the illusion of the pontic erupting from the tissue. On the palatal side, the pontic is tapered so that the receptor site is not extended below tissue; this allows easier access for oral hygiene. Note that when the receptor site is created, the bone must be a minimum of 2 mm from the most apical portion of the pontic.

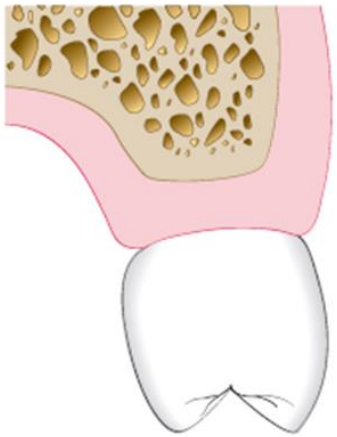
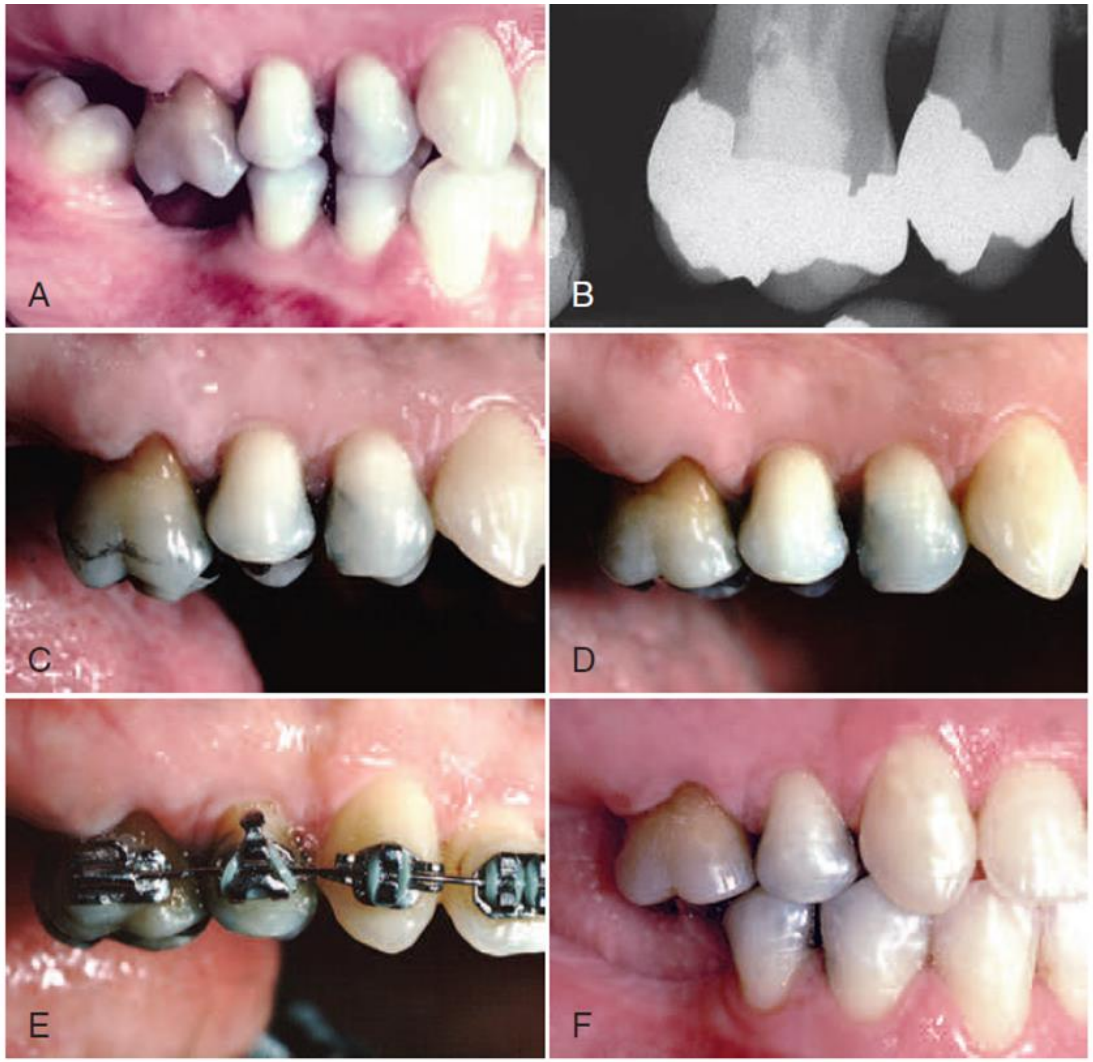


Fig. 70.11 Option for creating an ovate pontic receptor site in less aesthetic areas of the mouth. Rather than creating the receptor site so that the pontic extends into the ridge, it is possible to create a flattened receptor site in which the pontic sits flush with the ridge. This facilitates oral hygiene.

Periodontal-orthodontic interrelationship

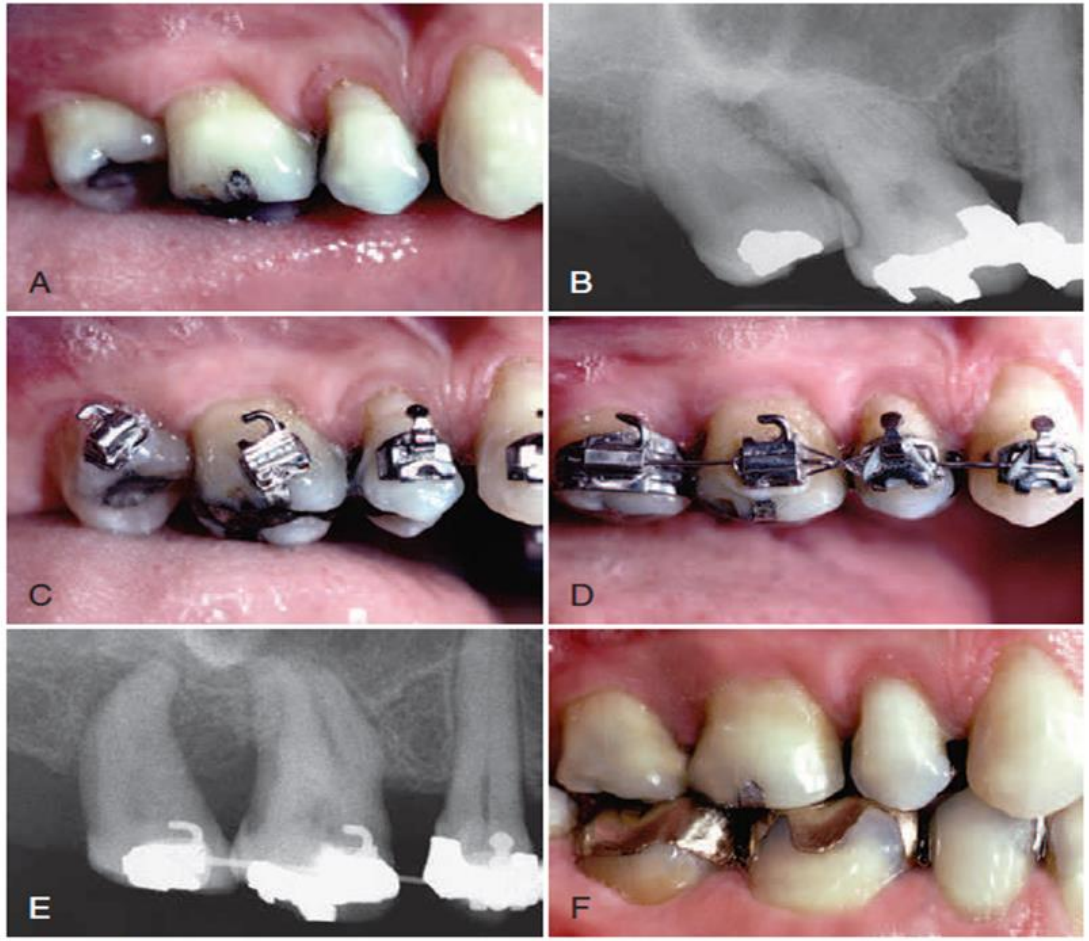
Orthodontic tooth movement in adults with periodontal tissue breakdown

- In the periodontally healthy patient, orthodontic brackets are positioned on the posterior teeth relative to the marginal ridges and cusps. However, some adult patients may have marginal ridge discrepancies caused by uneven tooth eruption. When marginal ridge discrepancies are encountered, the decision as to where to place the bracket or band is not determined by the anatomy of the tooth. In these patients, it is important to assess these teeth radiographically to determine the interproximal bone level.
- If the bone level is oriented in the same direction as the marginal ridge discrepancy, leveling of the marginal ridges will level the bone.
- If the bone level is flat between adjacent teeth and the marginal ridges are at significantly different levels, orthodontic correction of the marginal ridge discrepancy produces a hemiseptal defect in the bone. This could cause a periodontal pocket between the two teeth.
- If the bone is flat and a marginal ridge discrepancy is present, the orthodontist should not level the marginal ridges orthodontically.
- In these situations, it may be necessary to equilibrate the crown of the tooth.



eFig. 56A.6 This patient showed overeruption of the maxillary right first molar and a marginal ridge defect between the second premolar and first molar (A). The pretreatment periapical radiograph (B) showed that the interproximal bone was flat. To avoid creating a hemiseptal defect, the occlusal surface of the first molar was equilibrated (C and D), and the malocclusion was corrected orthodontically (E and F).

- For some patients, the equilebrated technique may require endodontic therapy and restoration of the tooth because of the required amount of reduction of the length of the crown. This approach is acceptable if the treatment results in a more favorable bone contour between the teeth.
- Some patients have discrepancies between both the marginal ridges and the bony levels between two teeth. However, these discrepancies may not be of equal magnitude; orthodontic leveling of the bone may still leave a discrepancy in the marginal ridges.
- In these patients, the crowns of the teeth should not be used as a guide for completing orthodontic therapy. The bone should be leveled orthodontically, and any remaining discrepancy between the marginal ridges should be equilibrated. This method produces the best occlusal result and improves the patient's periodontal health.



eFig. 56A.2 Before orthodontic treatment, this patient had significant mesial tipping of the maxillary right first and second molars, causing marginal ridge discrepancies and deep periodontal pockets (A). The tipping produced root proximity between the molars as well as a disruption of the normal gingival anatomy (B). To eliminate the root proximity, the brackets were placed perpendicular to the long axes of the teeth (C). This method of bracket placement facilitated root alignment and elimination of the root proximity, as well as leveling of the marginal ridge discrepancies (D through F).

Orthodontic treatment considerations for periodontal patients:

- Orthodontic therapy can provide several benefits to adult periodontal patients. The following seven factors should be considered:

1. Aligning crowded or malpositioned maxillary or mandibular anterior teeth permits adult patients better access to clean all surfaces of their teeth.
2. Vertical orthodontic tooth repositioning can improve certain types of osseous defects in periodontal patients. Often, moving the tooth eliminates the need for resective osseous surgery.
3. Orthodontic treatment can improve the aesthetic relationship of the maxillary gingival margins before restorative dentistry. Avoids gingival recontouring, entail bone removal and exposure of the roots of the teeth
4. Orthodontic treatment allows open gingival embrasures to be corrected to regain lost papillae.
5. Orthodontic treatment could improve adjacent tooth positioning before implant placement or tooth replacement. Drifting and tipping of the adjacent dentition.
6. A common tooth malalignment problem that results in periodontal pockets is the mesially tipped molar. Can be corrected orthodontically.

Periodontal surgery associated with ortho

- The extent of periodontal osseous surgery depends on the type of defect

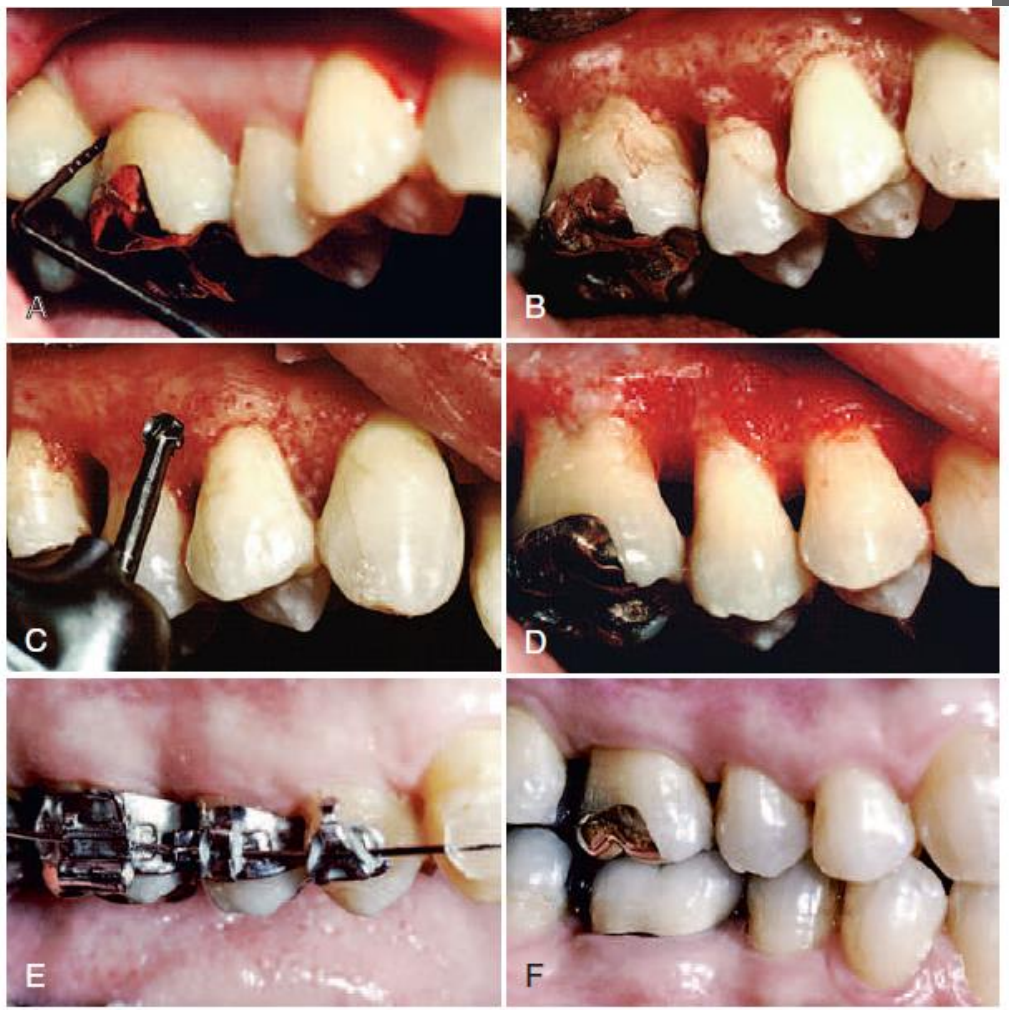
Osseous Craters

- An osseous crater is an interproximal, two-wall defect that does not improve with orthodontic treatment. Some shallow craters (4 to 5 mm pocket) may be maintained nonsurgically durin orthodontic treatment. However, if surgical correction is necessary, this type of osseous lesi can easily be eliminated by reshaping the defec and reducing the pocket depth. This in turn enhances the ability to maintain these interproximal areas during orthodontic treatment. The need for surgery is based on

I. The patient's response to initial root planing

II. The location of the defect,

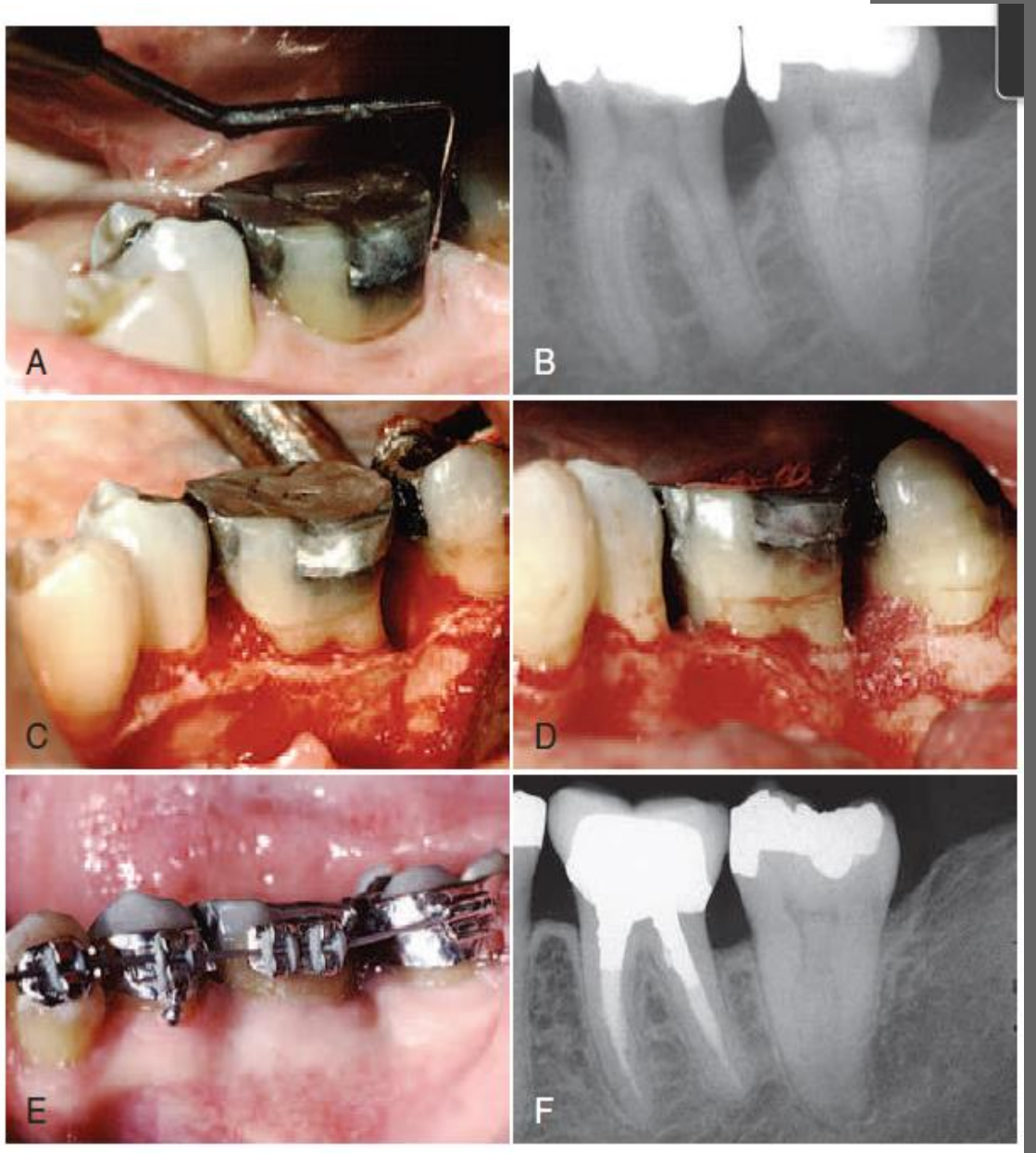
III. The predictability of maintaining defects nonsurgically, while the patient is wearing orthodontic appliances.



eFig. 56A.3 This patient had a 6-mm probing defect distal to the maxillary right first molar (A). When this area was flapped (B), a cratering defect was apparent. Osseous surgery was performed to alter the bony architecture of the buccal and lingual surfaces to eliminate the defect (C and D). After 6 weeks, the probing pocket defect had been reduced to 3 mm and orthodontic appliances were placed on the teeth (E). Because the crater was eliminated before orthodontic therapy, the patient could maintain the area during and after orthodontic treatment (F).

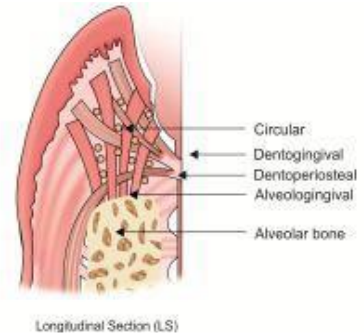
Three-Wall Intrabony Defects

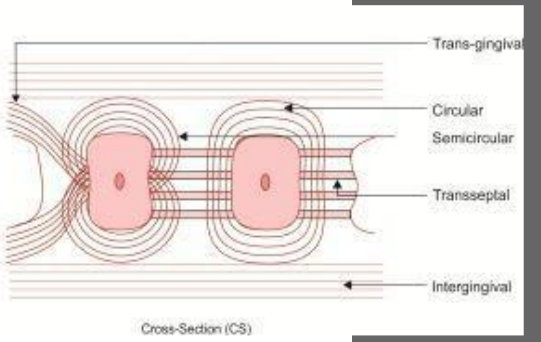
- Three-wall osseous defects are amenable to pocket reduction with regenerative periodontal therapy. Bone grafts using either autogenous bone from the surgical site or allografts, along with resorbable membranes, have been successful in regenerating three-wall defects.
- If the result is stable 3 to 6 months after periodontal surgery, orthodontic treatment may be initiated.



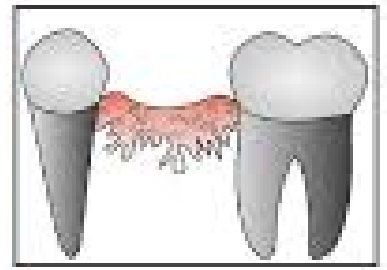
eFig. 56A.4 This patient had a significant periodontal pocket (A) distal to the mandibular right first molar. Periapical radiograph (B) confirmed the osseous defect. A flap was elevated (C), revealing a deep, three-wall osseous defect. Freeze-dried bone (D) was placed in the defect. Six months after the bone graft, orthodontic treatment was initiated (E). The final periapical radiograph shows that the preorthodontic bone graft helped regenerate bone and eliminate the defect distal to the molar (F).

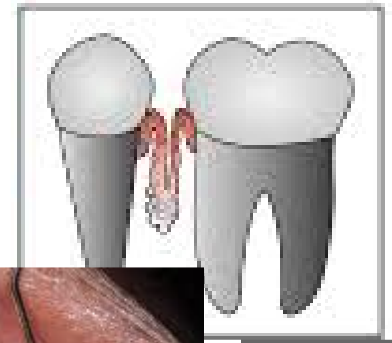
Minor surgery associated with Orthodontic treatment

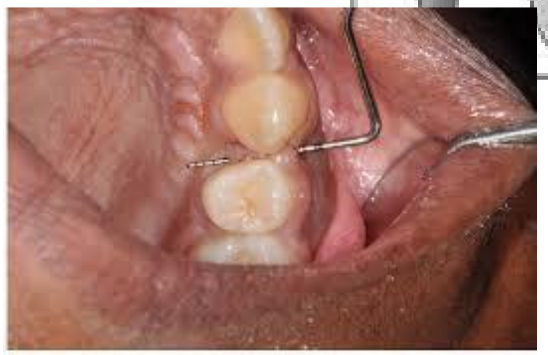




- 1.Fiberotomy: trans-septal fiber. It includes non-elastics collagenous fibers, which seems to contribute to rotational relapse after orthodontic treatment.
- 2.Frenotomy: It is indicatedin case of very hyperplastic type of frenum, with a fan like attachment. It may obstruct diastema closure. Frenectomy is not favorable anymore as is considered as a destructive procedure. It might also lead to loss of interdental papilla.
- 3.Gingivectomy: it may be used to increase the clinical crown during or after ortho treatment and in case of gingival discrepancy is apparent
- 4. Removal of gingival invagination. This invagination is happen after closure of extracted spaceby ortho treatment. Sometimes thisinvagination is still in place for more than 5 years. It is advised sometimes to remove this invagination surgically.
- 5. Use of implants: in orthodontic treatment, osseointegrated implant may be used. Close cooperation between orthodontist, periodontist, and oral surgeon is important for optimal treatment planning and implant positioning.







Periodontal-Prosthodontic interrelationship

Preprosthetic Surgery

Management of Mucogingival Problems

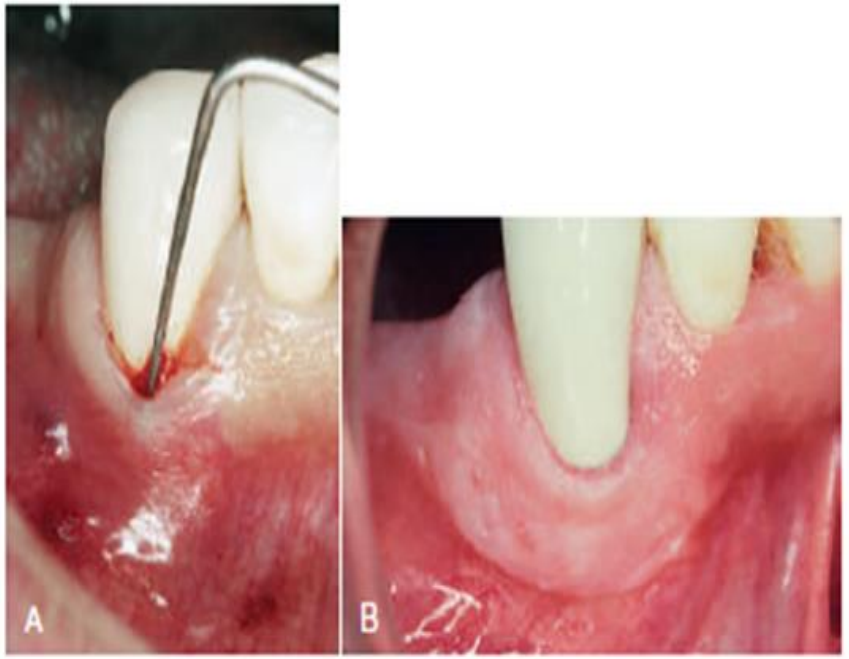
Periodontal plastic surgical procedures may be undertaken for a variety of reasons.[7] The most common techniques include those that increase the gingival dimensions and achieve root coverage. These procedures are often indicated before restoration for prosthetic reasons (eFig. 69.1) and in conjunction with orthodontic tooth movement.[34] Root coverage procedures may also be undertaken for purposes of comfort and aesthetics (eFig. 69.2).[7] At least 2 months of healing is recommended after soft tissue grafting procedures before initiating restorative dentistry[35] (see Chapter 65).

Preservation of Ridge Morphology After Tooth Extraction

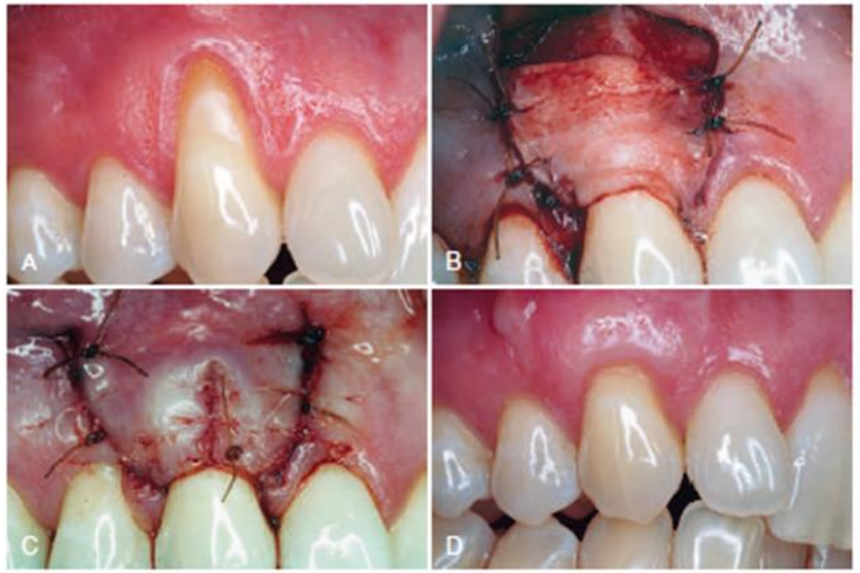
Alveolar ridge resorption is a common consequence of tooth loss.[1,2] Ridge preservation procedures have been shown to be useful in anticipation of the future placement of a dental implant or for pontics used for fixed bridges, as well as in cases where unaided healing would result in an unaesthetic deformity[16,26,27,33,36,42] (eFig. 69.3).

Crown-Lengthening Procedures

Surgical crown-lengthening procedures are performed to provide retention form to allow for proper tooth preparation, impression



eFig. 69.1 In preparation for a removable partial denture, this canine has received a gingival graft to increase attached gingiva and deepen the vestibule. (A) Before therapy. Note minimal attached gingiva. (B) After therapy, there is abundant attached gingiva and vestibular depth.



eFig. 69.2 Connective tissue graft placed under a double-papilla flap has been used to provide root coverage for a maxillary right canine. (A) Maxillary canine before therapy. (B) Connective tissue graft placed over denuded root surface. (C) Papilla placed over connective tissue. (D) Final result.

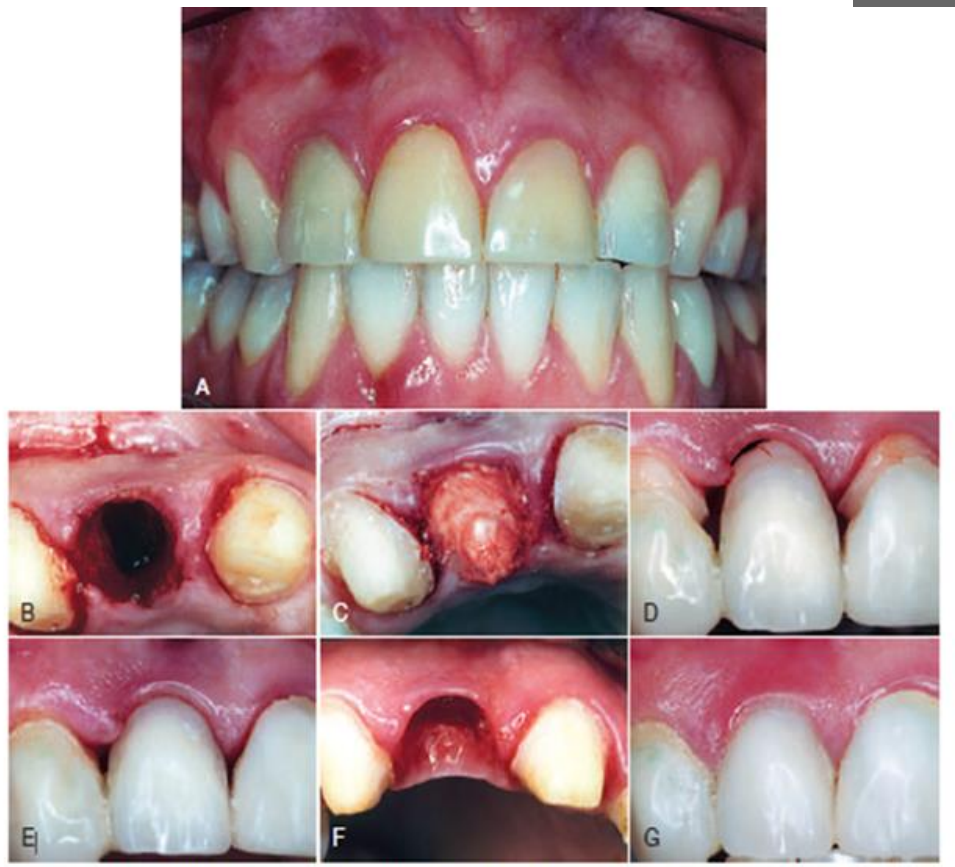
procedures,[23] and placement of restorative margins (eFig. 69.4),[23] and to adjust gingival levels for aesthetics.[35,50] It is important that crown-lengthening surgery is done in such a manner that the biologic width is preserved. The *biologic width* is defined as the physiologic dimension of the junctional epithelium and connective tissue attachment (see Chapter 70). This measurement has been found to be relatively constant at approximately 2 mm (±30%).[11] The healthy gingival sulcus has an average depth of 0.69 mm (eFig. 69.5).[21] It has been theorized that infringement on the biologic width by the placement of a margin of a restoration within its zone may result in gingival inflammation,[21] pocket formation, and alveolar bone loss[38] (eFig. 69.6). Consequently, it is recommended that there be at least 3 mm between the gingival margin and bone crest.[13,41,44,47] This allows for adequate biologic width when the restoration is placed 0.5 mm within the gingival sulcus[44,47] (eFig. 69.7).

Surgical crown lengthening may include the removal of soft tissue or both soft tissue and alveolar bone. Reduction of soft tissue alone is indicated if there is adequate attached gingiva and more than 3 mm of tissue coronal to the bone crest (eFig. 69.8). This may be accomplished by either gingivectomy or flap technique (see Chapter 60). Inadequate attached gingiva and less than 3 mm of soft tissue require a flap procedure and bone recontouring (eFig. 69.9). In the case of caries or tooth fracture, to ensure margin placement on sound tooth structure and retention form, the surgery should provide at least 4 mm from the apical extent of the caries or fracture to the bone crest (eFig. 69.10).

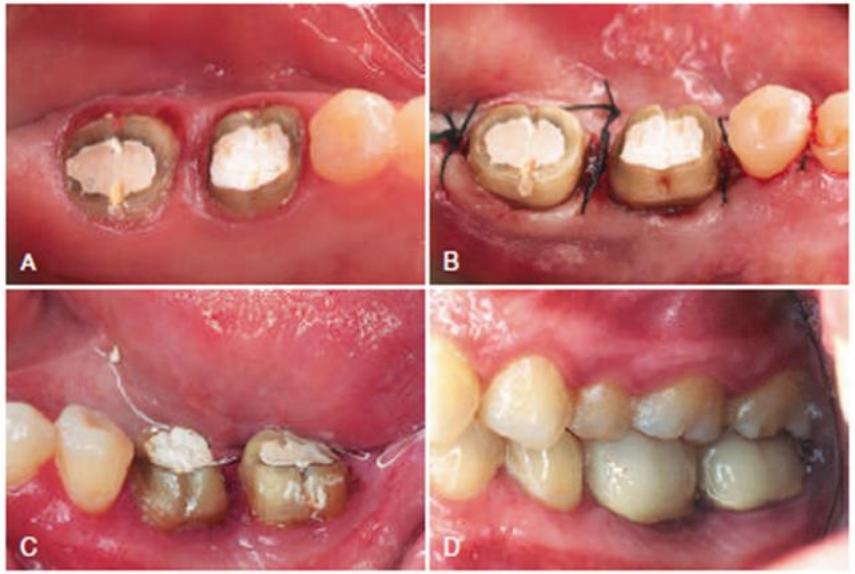
With the advent of predictable implant dentistry, it is important to carefully evaluate the value of crown lengthening for restorative therapy as opposed to tooth removal and replacement with a dental implant (eBox 69.1).

Alveolar Ridge Reconstruction

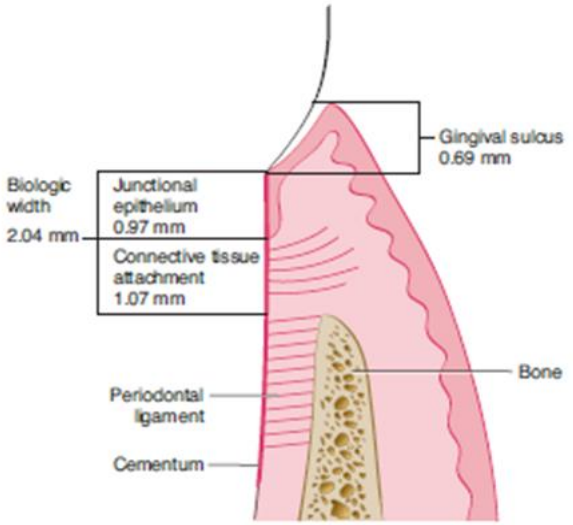
Patients are frequently seen with alveolar ridge resorption after tooth loss (see Chapter 75). To provide for adequate anatomic dimensions for the construction of an aesthetic pontic (see Chapter 70, or for a discussion on the placement of dental implants see Chapter 75), alveolar ridge reconstruction is undertaken.[42,43,45] In the case of aesthetic pontic construction, small defects may be treated with soft tissue ridge augmentation (eFig. 69.11). For larger defects and in those sites receiving dental implants, hard tissue modalities are used[43,45] (eFig. 69.12).



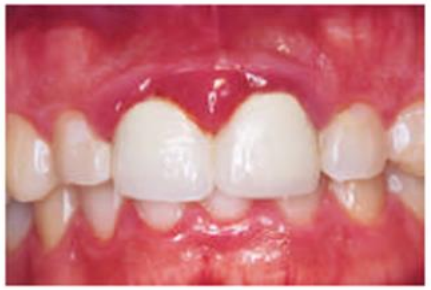
eFig. 69.3 (A) The maxillary right lateral incisor has failed endodontically, with a fistulous tract noted exiting from the attached gingiva. (B) The tooth is atraumatically removed and the socket debrided while maintaining the surrounding anatomic integrity. (C) In an effort to reduce ridge collapse, the socket is grafted with a combination of deproteinized bovine bone and calcium sulfate. (D) Provisional fixed partial denture is placed, with an ovate pontic extending 2 mm into the socket and supporting the surrounding tissues. (E–F) After 8 weeks, the socket has healed, preserving the gingival and papillary architecture, in preparation for an aesthetic final prosthesis. (G) Final restoration.



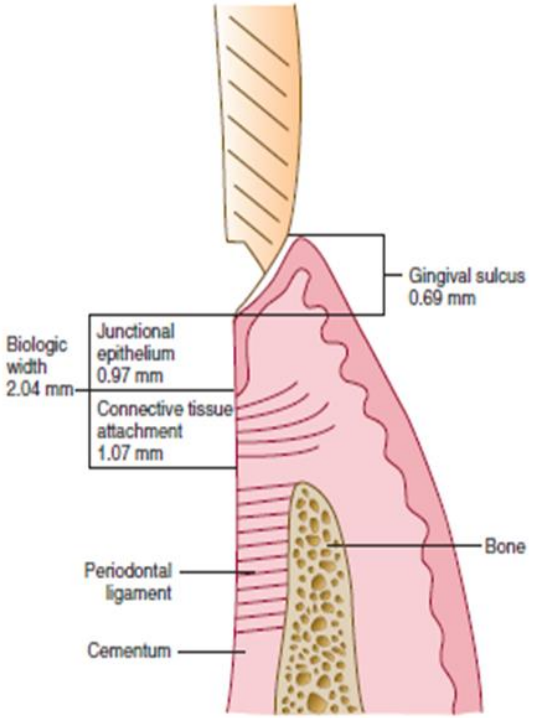
eFig. 69.4 Surgical crown lengthening has provided these otherwise unrestorable mandibular molars with improved retention and restorative access for successful restorations. (A) Before crown lengthening. (B) Crown-lengthening surgery completed. Note increased clinical crown. (C) Buccal view after surgery. (D) Final restorations.



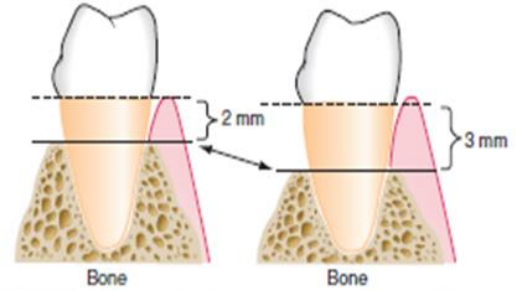
eFig. 69.5 The biologic width has been estimated to be about 2 mm. Efforts should be made to preserve its integrity.



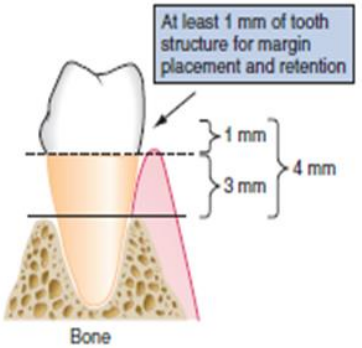
eFig. 69.6 Although gingival inflammation around crowns may have a variety of causes, infringement of biologic width must be considered.



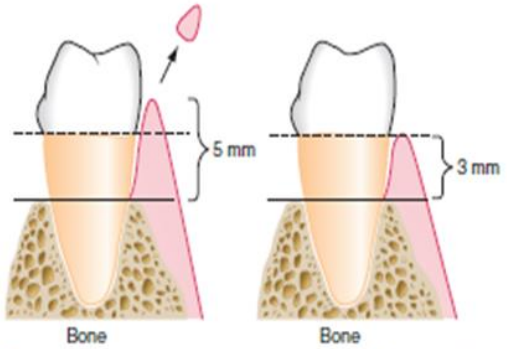
eFig. 69.7 Placement of the restorative margin 0.5 mm into the sulcus allows for the maintenance of the biologic width.



eFig. 69.9 With less than 3 mm of soft tissue between the bone and gingival margin, or less-than-adequate attached gingiva, a flap procedure and osseous recontouring are required for crown lengthening.



eFig. 69.10 In the case of caries or fracture, at least 1 mm of sound tooth structure should be provided above the gingival margin for proper restoration.



eFig. 69.8 Greater than 3 mm of soft tissue between the bone and gingival margin, with adequate attached gingiva, allows crown lengthening by gingivectomy.

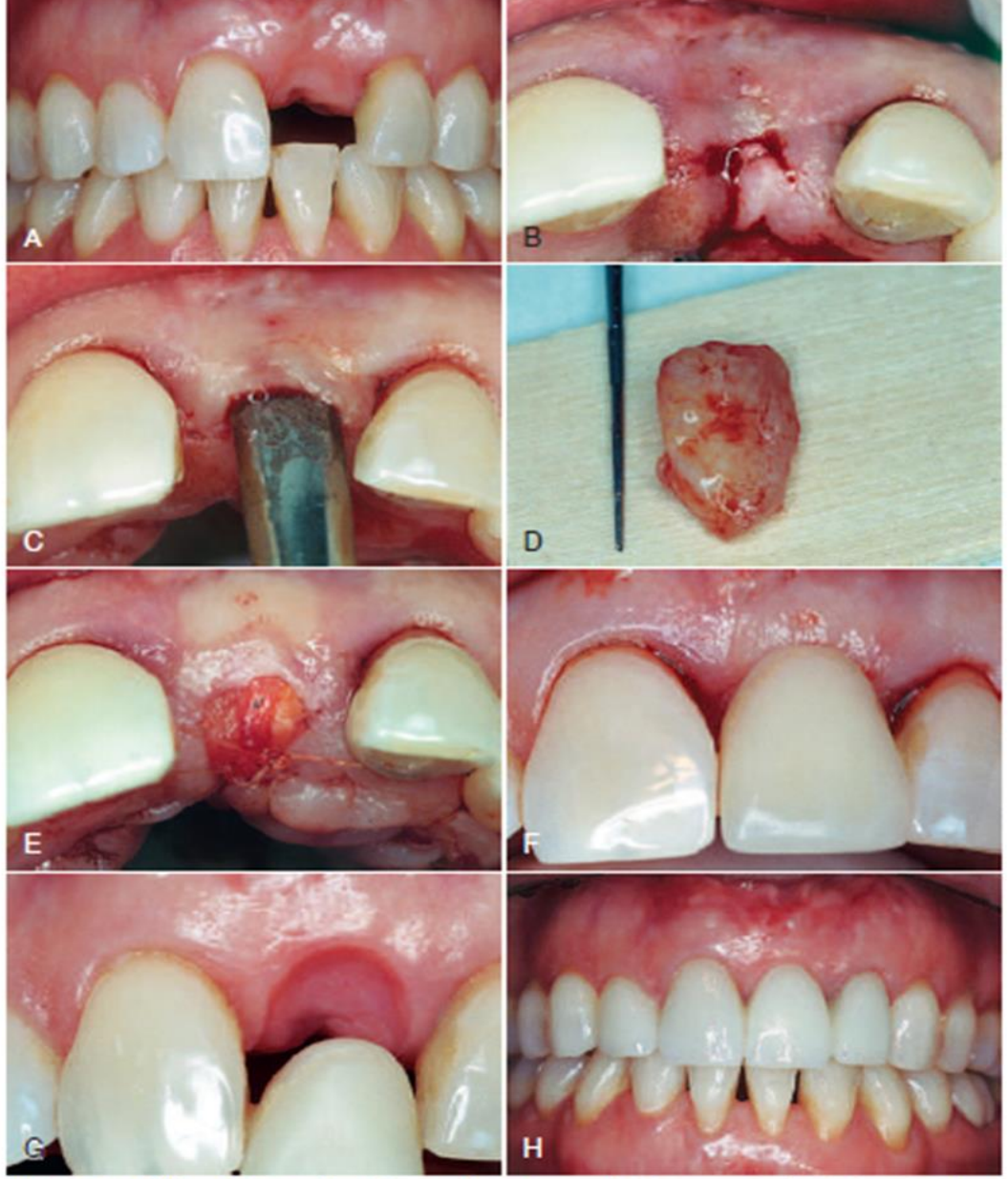
eBOX 69.1 Surgical Crown Lengthening

Indications

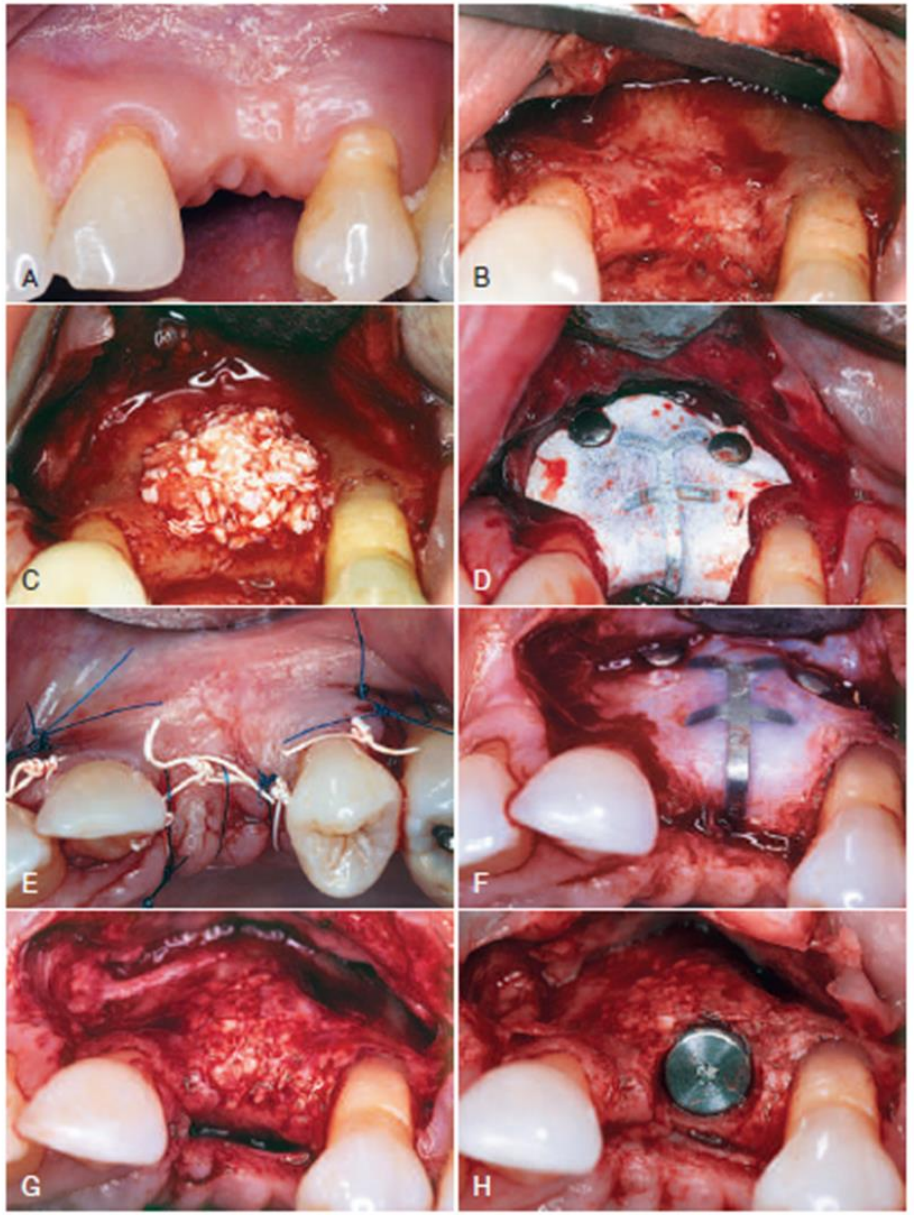
Subgingival caries or fracture.
Inadequate clinical crown length for retention.
Unequal or unaesthetic gingival heights.

Contraindications

Surgery would create an unaesthetic outcome.
Deep caries or fracture would require excessive bone removal on contiguous teeth.
The tooth is a poor restorative risk.



eFig. 69.11 (A) Loss of the maxillary left central incisor has resulted in an unaesthetic alveolar ridge defect. (B–E) An incision is made at the ridge crest, a pouch is created, and a soft tissue graft harvested from the palate is placed into the pouch. (F–H) A removable appliance with an ovate pontic is placed in light contact with the grafted site. Swelling around the pontic apex results in a tissue concavity from which the more natural-appearing final restoration emerges.



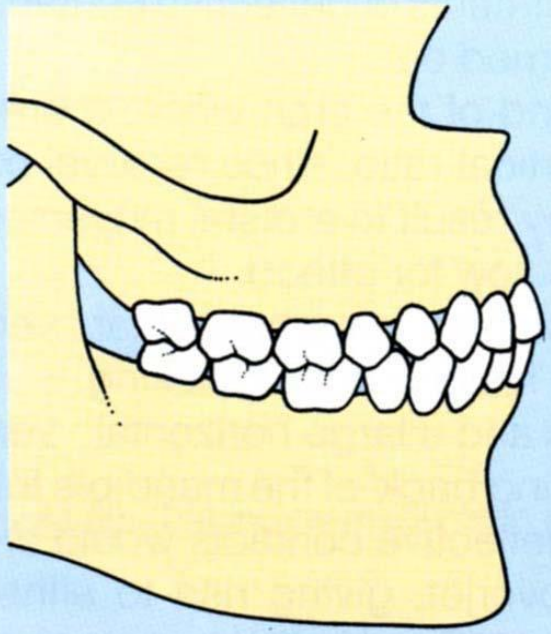
eFig. 69.12 Postextraction ridge defect is grafted with a combination of autogenous and deproteinized bovine bone and contained by nonresorbable barrier membrane.[53] After 8 months, the site is reopened and the membrane removed. A comparison of parts B and G shows significant reconstitution of hard tissue, in this case used for the installation of a dental implant. (A) Edentulous ridge before surgery. (B) Flap reflection to visualize defect. (C) Graft material placed over resorbed ridge. (D) Nonresorbable titanium-reinforced membrane placed over graft material. (E) Graft site sutured. (F) Surgical site reopened 8 months after surgery. (G) New bone over ridge. (H) Implant placed into augmented ridge.

Thank you

Periodontal response to external forces

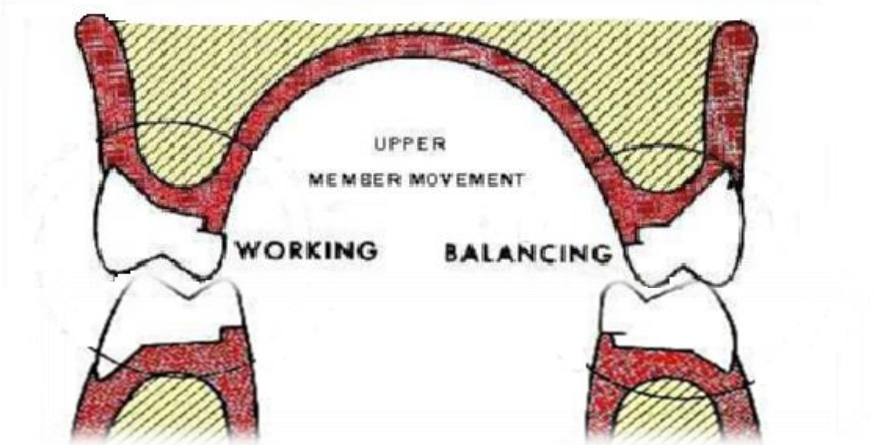
Dr Hadeel Mohammed Abbood

Occlusion:



- Is the functional relationship between all the masticatory system, including:
  - the teeth,
  - supporting structure of the teeth,
  - temporomandibular joint,
  - neuromuscular component
  - and the craniofacial skeleton.
- It is a dynamic relationship and should be evaluated physiologically as well as morphologically. The process of evaluation of occlusion should include an evaluation of each component of this system.

Common terms used in occlusion:



- 1- **Intercuspal position (ICP):** it is the synonym of central occlusion. It represents the position of the mandible when there is a maximal intercuspation between the maxillary and the mandibular teeth.
- 2- **Muscular Contact position (MCP):** the position of the mandible when lifted into contact from resting position.
- 3- **Excursive movement:** any mandibular movement away from ICP.
- 4- **Laterotrusion:** or the working movement. It is the movement of the mandible laterally to the right or left of the ICP.
- **5- Laterotrusive** side: Working side. It is the of either dental arch corresponding to the side of the mandible moving away from the midline.
- 6- **Medotrusive side:** the side of either dental arch corresponding to the side of mandibular movement toward the midline. Synonym: balancing side, or nonworking side.

- 7- Protrusion: movement of the mandible anterior to the ICP.
- 8- Retrusion: movement of the mandible posteriorly from the ICP.
- 9- Retruded position: centric relation. It is the most cranial position of the mandible along the retruded path of closure.
- 10- Guidance: Pattern of opposing tooth contact during excursive movement of the mandible. The teeth making such contact cause separation of the other teeth. Also called disclusion.
- 11- Interference: any contact, in ICP or excursions, that prevents the remaining occlusal surfaces from achieving stable contact. Synonym: supracontact.

Classification of occlusion:

1. Physiologic occlusion: it is present when there is no sign of dysfunction or disease are present and no treatment required.
2. Non-physiologic (or traumatic) occlusion: it is associated with dysfunction or a disease due to injury, and treatment may be indicated.
3. Therapeutic occlusion: is the result of a specific intervention designed to treat dysfunction or disease.

Signs and symptoms of non-physiologic occlusion

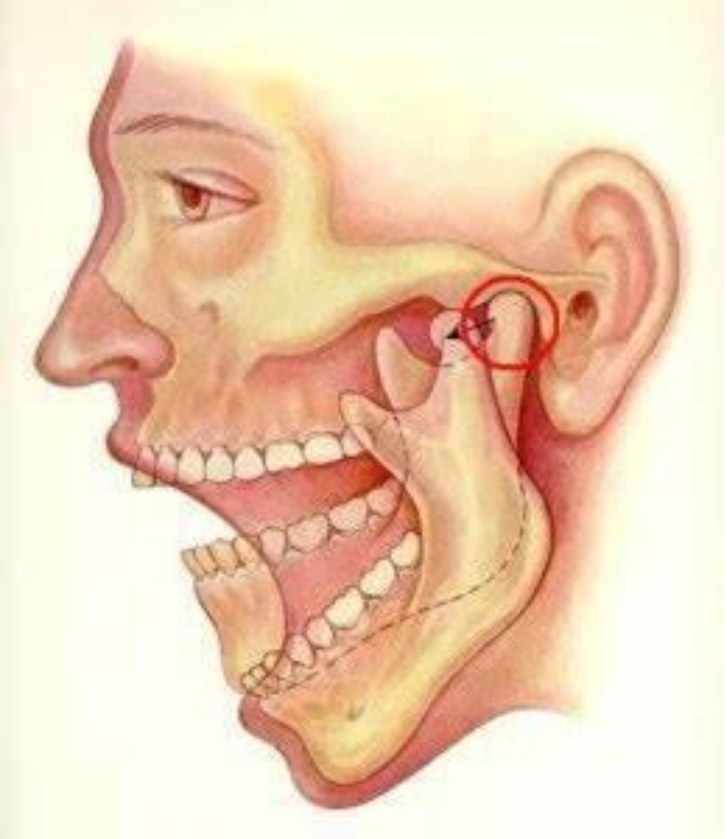
- These include damaged teeth, restorations, abnormal mobility, fremitus, widened periodontal space, pain, and a subjective sense of biting discomfort.
- In traumatic occlusion, occlusion of teeth causes periodontal injury. It is therefore, different from malocclusion because not all malocclusion cause injury and pain.

Assessment of occlusion

- This can be divided into

1. **Tempromandubular disorder screening**
2. **Intraoral evaluation of occlusion.**

Tempromandubular disorder screening:



It does take around 5 minutes and should be included in all routine dental examination. TMD screening can be done by 5 steps:

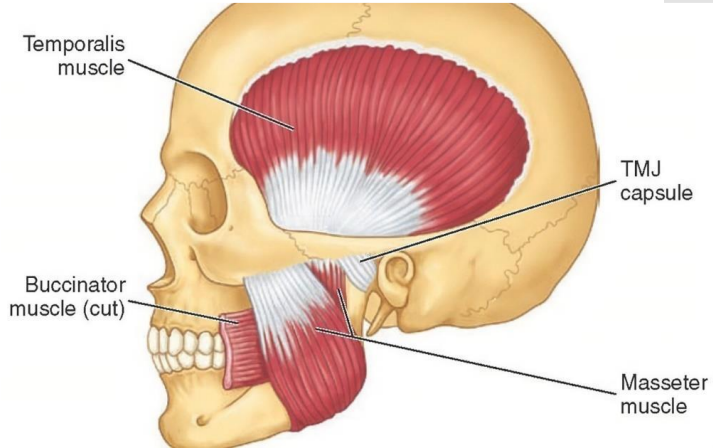
a. Measuring the maximal interincisal opening and recording the measures in mm.

b. Observing the opening-closing pathway and record any deviation from the midline.

c. Auscultation of the TMJ sounds. Two types of sounds can be described discrete clicks or diffuse grating sounds.

d. Palpation of any TMJ tenderness bilaterally

e. Palpation of any tenderness in the masseter and temporalis muscles (in both sides and from the origin to the insertion of the muscles). The severity of pain should be recorded.



Intraoral evaluation of occlusion.

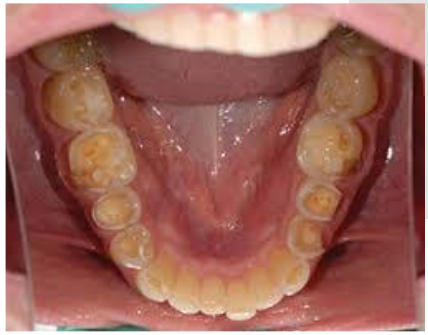
- This includes four aspects:

A. Evaluation of the ICP: The patient should be able to close into ICP consistently from MCP without searching for a stable or comfortable bite. Mylar strips, occlusal indicator wax and marking ribbon can be used in the patient's mouth and asking him to close and hold. Any presence or absence of contact on all cusped teeth should be documented.

B. Excursive movement: The quality of tooth contact patterns during mandibular movement out of the ICP are recorded. Using Mylar strip inside patient's mouth and ask the patient to move the mandible into protrusion and laterotrusion.

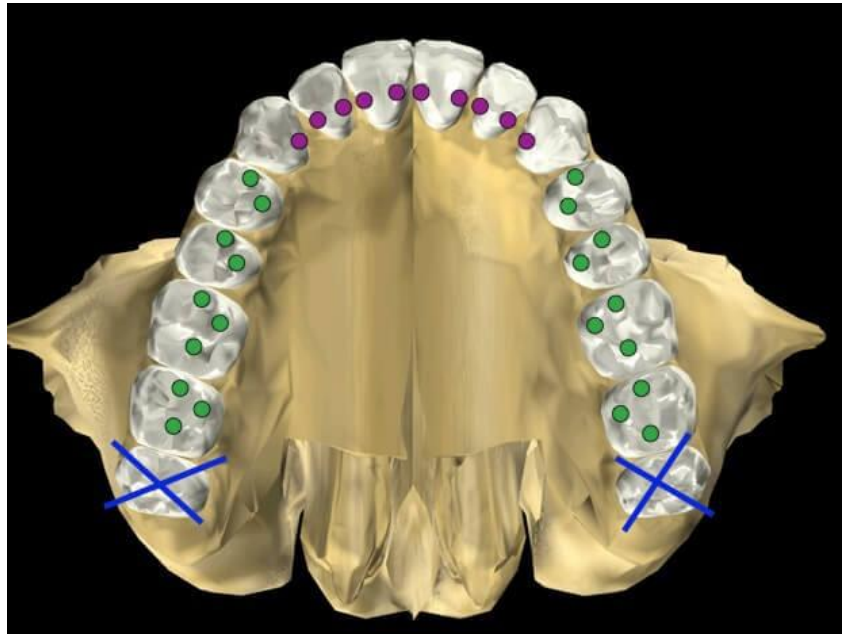
C. Tooth mobility: Tooth mobility are evaluated and recorded.

D. Attrition: It is defined as tooth wear due to tooth-to-tooth contact. A certain amount of attrition might be acceptable. However, accelerated attrition with presence of significant wear facets should be noted.



Occlusal evaluation:

1. All the findings from occlusal examination should be reevaluated in the context of physiologic and nonphysiologic occlusion. Occlusion should meet the requirement of occlusal stability, which are:
2. Intercuspal position:
   A. Light or absent anterior contacts
   B. Well-distributed posterior contacts.
   C. Coupled contact between posterior teeth
   D. Cross tooth stabilization
   E. Forces directed along long axis of each tooth.
3. Smooth excursive movement without interference
4. No trauma from occlusion
5. Favorable subjective response to occlusal form and function.



Adaptive capacity of the periodontium to occlusal forces

- The periodontium tries to accommodate to the occlusal forces. This accommodation is influenced by four factors:

1. Magnitude: when occlusal forces increase, few changes in the periodontium will occur . these are:
   A. Widening of the periodontal ligament space
   B. Increase in the number and width of the periodontal ligament fibers.
   C. Increase in density of the alveolar bone.
2. Direction: Periodontium is designed to withstand occlusal forces directed along the long axis of the tooth. When the direction of the occlusal forces change, this will cause reorientation of the stress and strains within the periodontium. Lateral forces and torque forces are more likely to injure the periodontium.
3. Duration: constant forces are more injurious to periodontium than intermittent.
4. Frequency: the more frequent the application of intermittent force the more injurious the force to the periodontium.

Trauma from occlusion:

- It is defined as the resultant injury to the tissue due to occlusal forces exceeds the adaptive capacity of the periodontal tissue. An occlusion that produces such injury is called traumatic occlusion. This excessive traumatic force may cause other signs and symptoms other than periodontal injury, such as temporomandibular joints injury, tooth wear and painful spasm.

Classification of trauma from occlusion

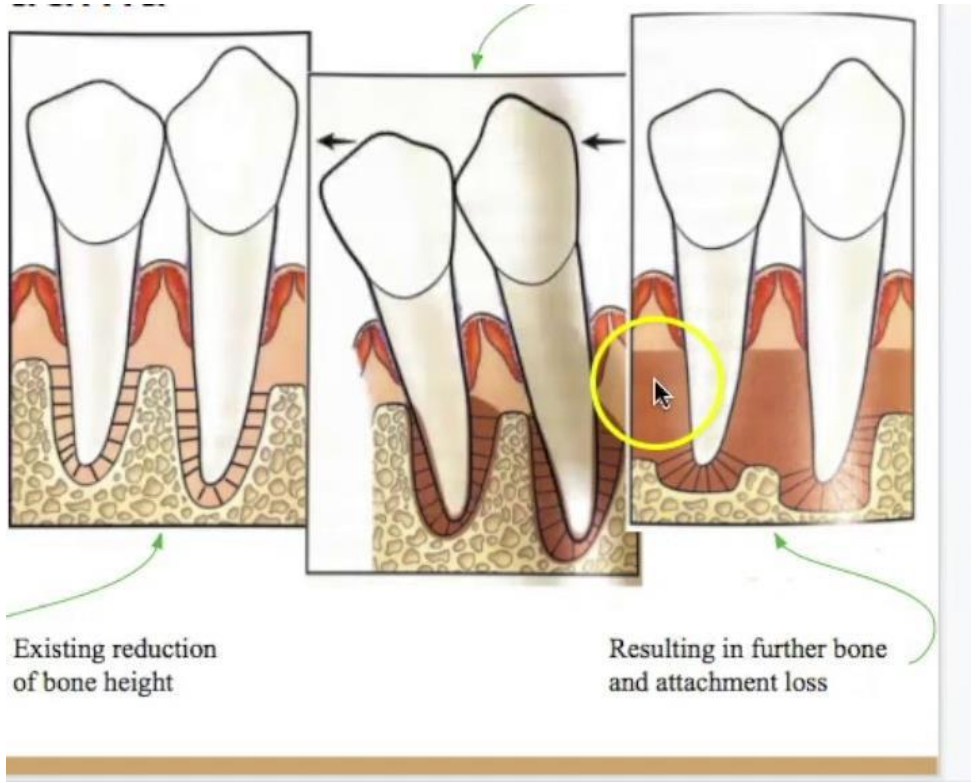
- Trauma from occlusion can be classified according to duration of injury into:

1. Acute trauma from occlusion: results from an abrupt occlusal impact, such as that produced by biting on a hard object, restorations or prosthetic appliance that alter or interfere with the direction of occlusal forces. The results are tooth pain, sensitivity to percussion, and increase tooth mobility. If the cause treated, the symptoms will subside the injury will heal. In case of not treating the cause, periodontal necrosis and abscess formation might occur, cementum tears, or might develop into symptom-free chronic condition.
2. Chronic trauma from occlusion: More common than acute condition. Usually develops from gradual changes in occlusion produced by tooth wear, drifting movement, and extrusion of teeth, combined with parafunctional habits such as bruxism and clenching rather than as a sequela of acute condition.

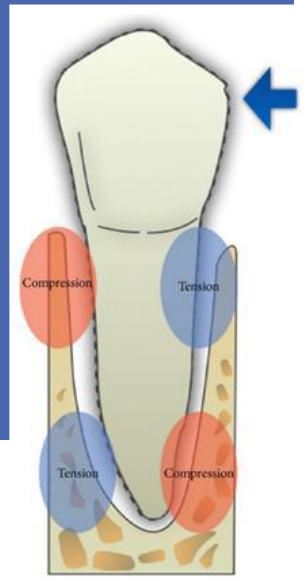
- Malocclusion without periodontal injury is not a traumatic occlusion.

Primary and secondary TFO

- TFO can be classified according to the cause into primary and secondary TFO. TFO may be caused by alteration of occlusal forces or reduced capacity of the periodontium to withstand occlusal forces or both of them.
- Primary trauma from occlusion results when there is alteration in the occlusal forces that cause injury to the periodontium. In this type of injury, there is no pocket formation and no apical migration of the junctional epithelium and no change in the level of connective tissue attachment. In addition, the supracrestal fibers are intact.
- Secondary TFO: It occurs when the adaptive capacity of the tissue to withstand occlusal forces is impaired by bone loss due to marginal inflammation. This reduced periodontal attachment area will change the leverage on the remaining tissue and the periodontium becomes more vulnerable to injury from an occlusal force that was previously well-tolerated.

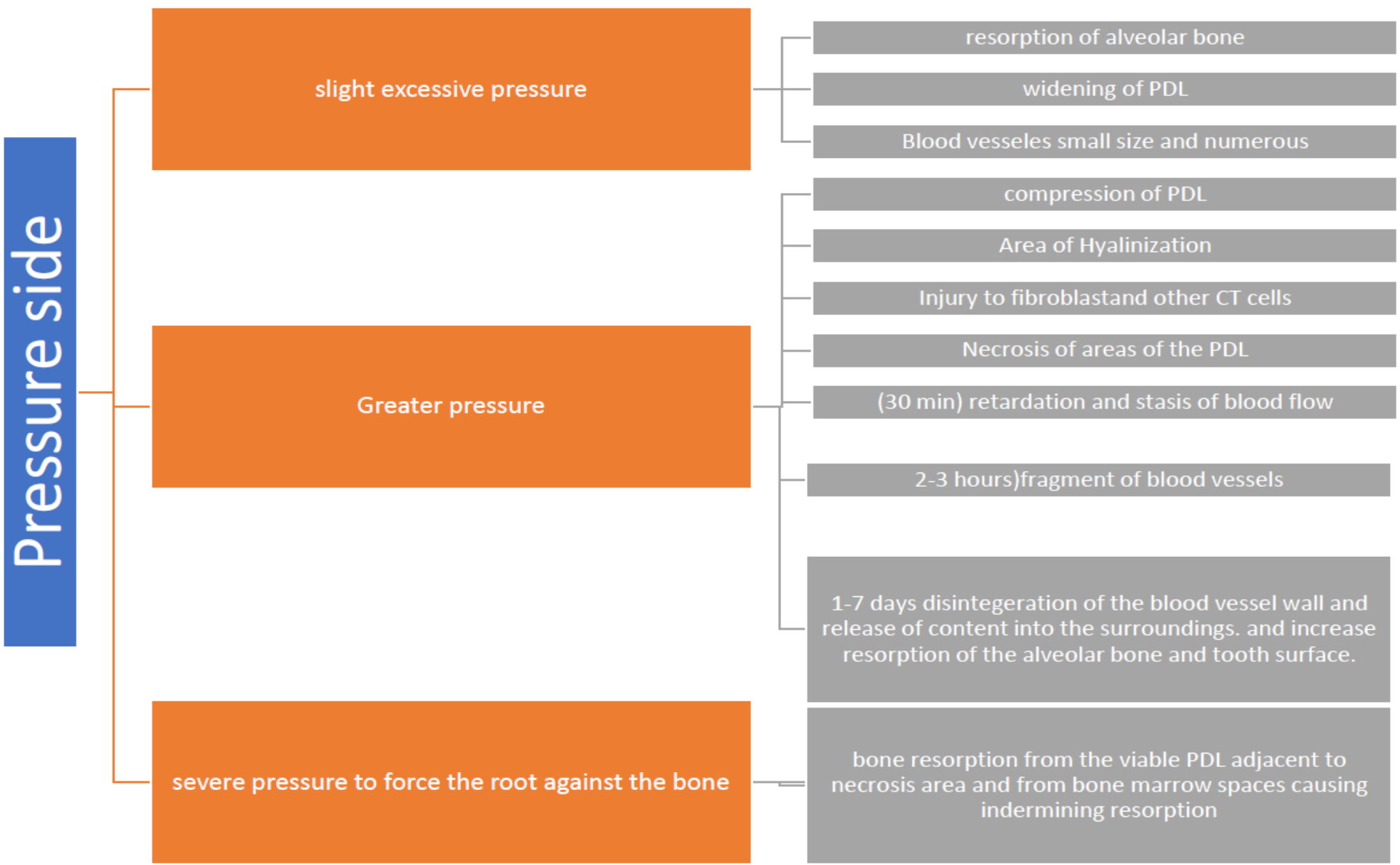

Existing reduction
of bone height
Resulting in further bone
and attachment loss

Stages of tissue response to trauma from occlusion:

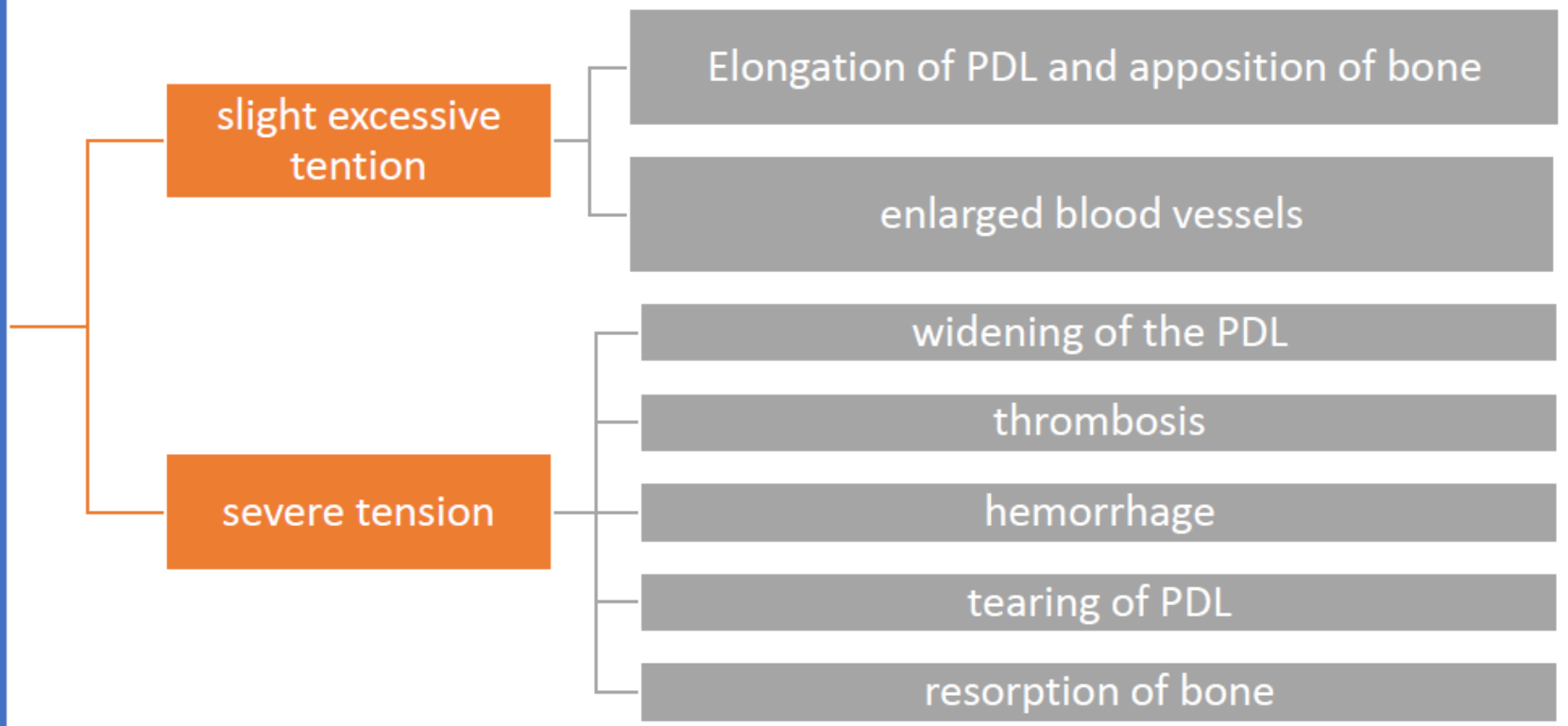


➢ Stage I: Injury

- The periodontium is remodeled to cushion the impact of the forces. the ligament is widened at the expense of the bone, resulting in angular bone defects without periodontal pocket and the tooth becomes loose.
- In single rooted tooth, under the force of occlusion, the tooth rotates around a fulcrum line which is normally located between the middle third and the apical third of the clinical root. This creates areas of pressure and tension on opposites sides of the root. if jiggling forces are exerted, these different lesions may coexist in the same area.
- Furcations are the most susceptible area to injuries from excessive occlusal forces in multirooted teeth. Histological changes in injury stage includes, temporary depression in mitotic activity, proliferation and differentiation of fibroblast, depression in collagen formation and bone formation too.


Pressure side
slight excessive pressure
resorption of alveolar bone
widening of PDL
Blood vesseles small size and numerous
Greater pressure
compression of PDL
Area of Hyalinization
Injury to fibroblastand other CT cells
Necrosis of areas of the PDL
(30 min) retardation and stasis of blood flow
2-3 hours)fragment of blood vessels
1-7 days disintegeration of the blood vessel wall and release of content into the surroundings. and increase resorption of the alveolar bone and tooth surface.
severe pressure to force the root against the bone
bone resorption from the viable PDL adjacent to necrosis area and from bone marrow spaces causing indermining resorption

Tension side



- o Stage II: Repair
- Repair is a continuous process in the periodontium, and TFO stimulates increase in reparative activity.
- Histologically, the damaged tissue removed, and formation of new cell and fibers, bones and cementum are formed in an attempt to restore injured tissue. In case that damage exceeds reparative capacity of the tissue forces remain traumatic.

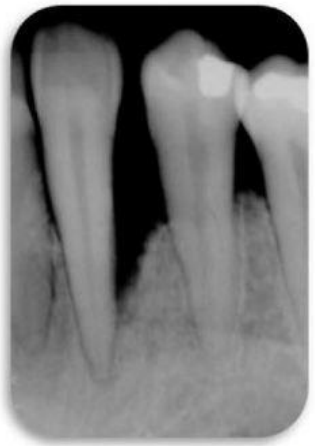


FIGURE 1. Initial radiograph: vertical bone resorption on the distal aspect of tooth #33.

- o Stage III: Adaptive remodeling of the periodontium
- A remodeling occurs in case that the injury is exceeding the repair capacity. The remodelling process is an attempt of the body to modify the tissue in a way that reduce injurious forces.
- Histologically, a thickening in the PDL with funnel shape at the crest will appear. In addition to angular bony defect with no pocket formation, increased vascularization and loosening of the involved teeth.

Relationship between plaque-induced periodontal diseases and trauma from occlusion

- TFO does not induce periodontal inflammation. However, it might constitute an additional risk factor for the progression and severity of periodontitis. There are few theories that have been proposed to explain the interaction between TFO and periodontitis:
- 1- TFO might alter the pathway of extension of gingival inflammation to the periodontium by affecting the collagen density and increased number of leukocytes, osteoclasts, and blood vessels in the coronal region of the increasingly mobile tooth. Angular bony defect due to TFO might redirect the gingival inflammation to the PDL and existing pockets might become intrabony.
- 2- Root resorption might occur due to TFO. When these lesions uncovered by apical migration of gingival inflammation, they might favor the accumulation of plaque and calculus and subsequently deeper lesion.
- 3- Supragingival plaque can become subgingival if the tooth is tilted orthodontically or migrates into an edentulous area resulting in the transformation of suprabony pocket into intrabony pocket.
- 4- Increase mobility of an affected tooth might exacerbate plaque metabolites and increase their diffusion.

Clinical and radiographic signs of trauma from occlusion

- This includes:

1. Increased width of the periodontal space with often thickening of the lamina dura along the lateral aspects of the roots in the apical region and the bifurcation area.
2. A vertical rather than horizontal defect in the interdental septum.
3. Radiolucency and condensation of the alveolar bone.
4. Root resorption.

Pathologic tooth migration:

- It represents a displacement that result when the balance among the factors that maintain physiologic tooth position is disturbed by periodontal disease. It is relatively common and might represent an early sign of periodontitis.

Pathogenesis:

- There are two major factors that play an important role in maintaining the normal position of the teeth:
- 1- Health and height of the periodontium.
- 2- Forces excerted on the teeth, includes occlusal forces and pressue of the tongue lips and cheeks.
- Any defect in any of these two factors or both of them together results in pathologic tooth mobility

1- Weakened periodontal support:

- In the presence of normal occlusal and muscular forces together with normal periodontal support, the tooth hold its position with no change. However, in periodontitis and reduced periodontal support, occlusal or other muscular forces might which were normal forces might become injurious. Sometimes abnormal forces that can be withstood by intact periodontal support, causes the tooth to migrate when periodontal support weakened by disease. Example on this, diastemata between the teeth.

2- Changes in the forces exerted on the teeth

- Changes in the magnitude, direction or frequency of the forces exerted on the teeth can induce pathologic migration of a tooth or a group of teeth. These forces do not have to be abnormal to produce tooth migration in the presence of weakened periodontal support. It might occur as a result of unreplaced missing teeth or failure to replace first molar or other causes.

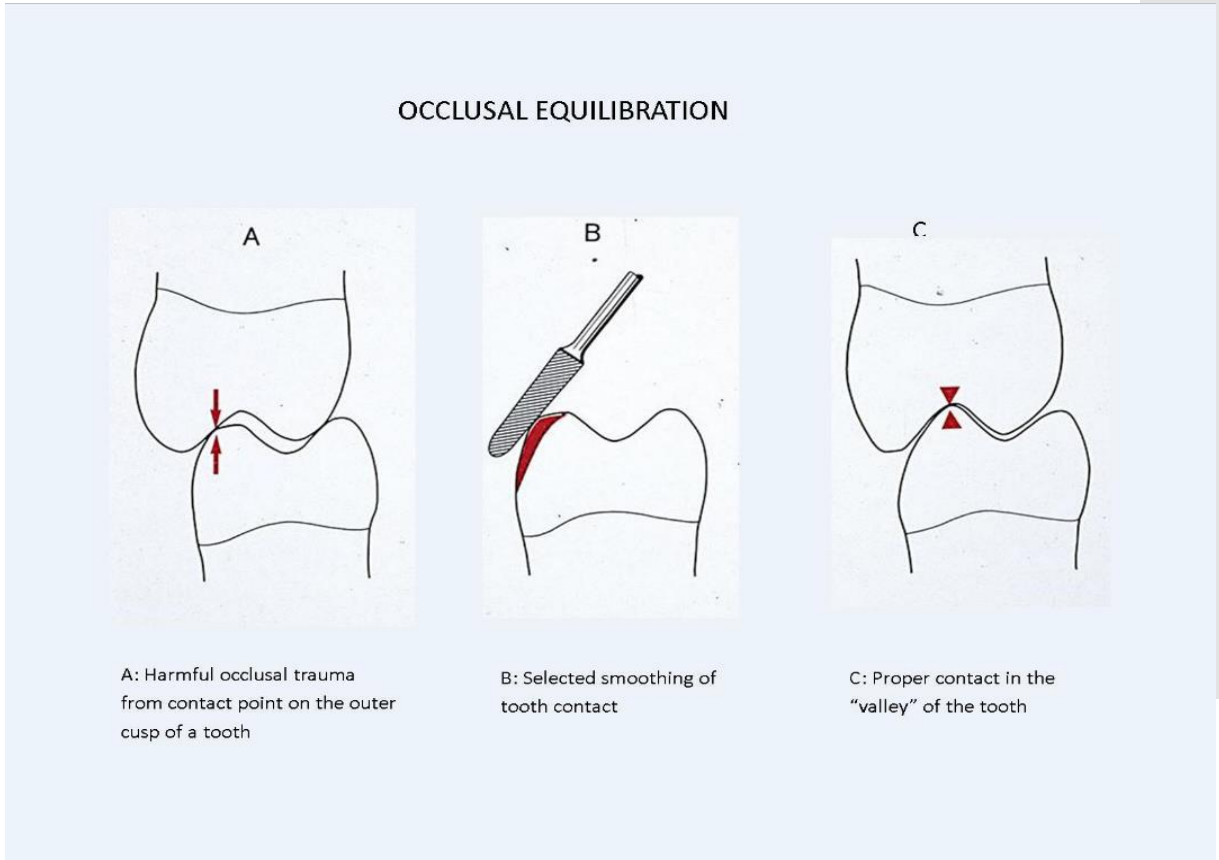
Treatment

- TFO is reversible. The periodontium has the capacity to modulate itself and adapt to the injurious forces. However, in some cases, modulation is not enough, the source of injury needs to be removed for repair to occur. If the conditions do not permit the teeth to escape from the injury and adapt to it, periodontal
- damage persists and worsens. In addition, the accumulation of plaque and presence of periodontal inflammation may impair the reversibility of the traumatic lesion.

- The purpose of occlusal therapy is to establish stable functional relationships favorable to the oral health of the patient, including periodontium. A variety of procedures could contribute to this objective: interocclusal appliance therapy, occlusal adjustment, restorative procedures, orthodontic tooth movement, and orthognathic surgery. there are few guidelines to be applied to occlusal therapy:
- 1- Sound biologic rationale for the intervention.
- 2- Occlusal intervention should be considered adjunct to periodontal therapy
- 3- Significant irreversible occlusal changes should be considered in the context of restorative care planned for the patient.
- 4- Thorough informed consent to be provided to the patient. It is important the patient understand the main goal of occlusal intervention is to increase stability and comfort not to treat periodontal disease.

Occlusal adjustment:

- Or coronoplasty is the selective reshapening of the occlusal surfaces with the goal of establishing a stable nontraumatic occlusion. It is an invasive irreversible intervention. Occlusal adjustment should be differed until inflammation is controlled, time is allowed to healing and reevaluation determined that any remaining tooth mobility is due to excessive load on teeth rather than decrease periodontal support.



Intercuspal appliance:

- generally fabricated from hard acrylic resin has the advantages of providing a reversible means of redistribution of occlusal forces and minimizing excessive forces on individual teeth. A full coverage maxillary appliance is particularly useful in managing bruxism as part of an overall comprehensive treatment plan for the patient. In periodontitis patients, this type of appliance provides sense of bite comfort and reduce destructive consequence of bruxism.

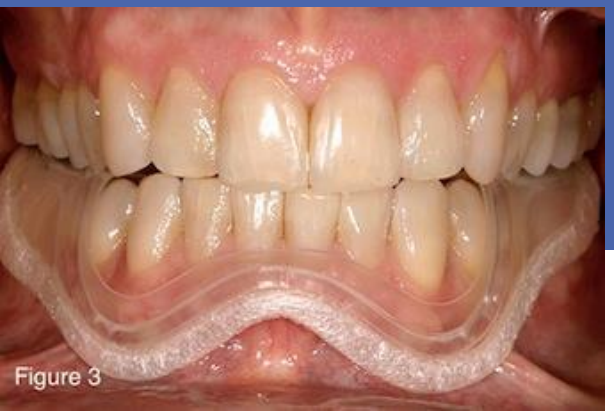


Figure 3

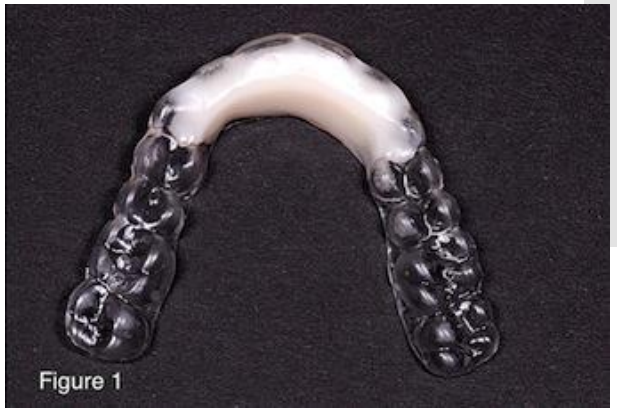


Figure 1

Thank you

Non-surgical treatment Sonic and ultrasonic instrumentation

Dr Hadeel Mohammed Abbood

Non surgical treatment

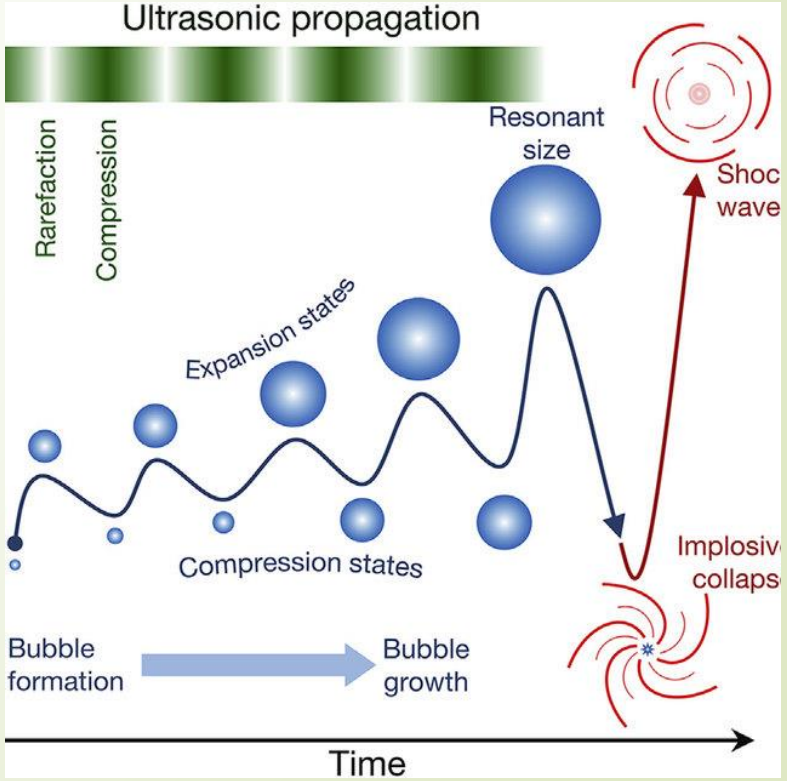
- Initial treatment
- Extended for benefits and less destructive
- Scalers and irrigators

Power driven instruments

- They may be used alone or combination with hand instruments.
- Evidence indicates that power-driven instruments provide clinical outcomes similar to those derived from hand instruments.
- Power instrumentation has the potential to make scaling less demanding and more time efficient.
- Potential hazards from using power-driven devices include:
  - Rough root surfaces,
  - Production of bioaerosols,
  - Interference with cardiac pacemakers.

Mechanism of Action of Power Scalers

- Various physical factors play a role in the mechanism of action of power scalers:
  - frequency, stroke, and water flow.
- Water contributes to three physiologic effects that play a role in the efficacy:
  - acoustic streaming, acoustic turbulence, and cavitation.
- Acoustic steaming is unidirectional fluid flow caused by ultrasound waves.
- Acoustic turbulence is created when the movement of the tip causes the coolant to accelerate, producing an intensified swirling effect. This turbulence continues until cavitation occurs.
- Cavitation is the formation of bubbles in water caused by the high turbulence. The bubbles implode and produce shock waves in the liquid, thus creating further shock waves throughout the water.
- In vitro, the combination of the 3 effects has been shown to disrupt biofilm.



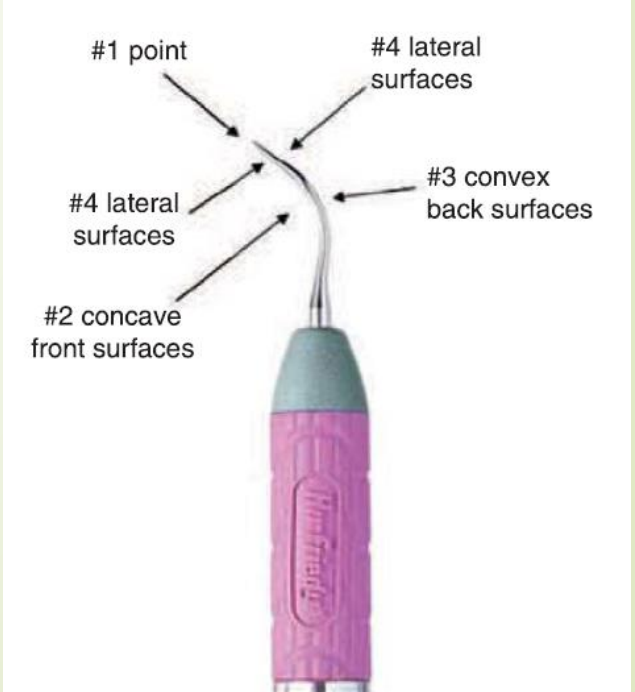
Type and Benefit of Power Instruments

Sonic units

- work at a frequency of 2000 to 6500 cycles per second
- use a high- or low-speed air source from the dental unit.
- Water is delivered via the same tubing used to deliver water to a dental handpiece.
- Sonic scaler tips are large in diameter and universal in design.
- A sonic scaler tip moves in an elliptical or orbital stroke pattern. This stroke pattern allows the instrument to be adapted to all tooth surfaces.

Magnetostrictive ultrasonic scaler

- Work in a frequency range of 18,000 to 50,000 cycles per second.
- Electric power --Metal stacks change dimension magnetostrictive waves.
- Vibrations travel from the metal stack to a connecting body causes the vibration of the working tip.
- Tips move in an elliptical or orbital stroke pattern.
- This gives the tip four active working surfaces.



Piezoelectric ultrasonic scale

- work in a frequency range of 18,000 to 50,000 cycles per second.
- Ceramic disks located in the handpiece power the piezoelectric technology and change in dimension as electric energy is applied.
- Piezoelectric tips move primarily in a linear pattern, giving the tip two active surfaces.
- Various insert tip designs and shapes are available for use.


UDS-K
6
7
4
8
3
2
MAX
MIN

BOX 51.1 Advantages and Disadvantages of Mechanized Instruments Compared With Manual Instruments

Advantages

Increased efficiency
- Multiple surfaces of tip are capable of removing deposits
- No need to sharpen
- Less chance for repetitive stress injuries
- Large handpiece size
- Reduced lateral pressure
- Less tissue distention
- Water
- Lavage
- Irrigation
- Acoustic microstreaming

Disadvantages

More precautions and limitations
- Client comfort (water spraying)
- Aerosol production
- Temporary hearing shifts
- Noise
- Less tactile sensation
- Reduced visibility

From Darby ML, Walsh MM: *Dental hygiene,* ed 3, St. Louis, 2010, Saunders.

Efficiency

- Modified tip designs allow for improved access in many areas, including furcations.
- Newer, slimmer designs operate effectively at lower power settings, thus improving patients' comfort.

Tip Designs

- Some tips are designed to remove heavy supragingival calculus or debride periodontal pockets definitively.
- Large-diameter tips are created with a universal design and are indicated for the removal of large, tenacious deposits.
- A medium to medium-high power setting is generally recommended.
- Thinner-diameter tips may be site specific in design.
- The straight-tip design is ideal for use in treating patients with gingivitis and deplaquing maintenance patients.
- The right and left contra-angled instruments allow for greater access and adaptation to root morphology. These inserts are designed to work on a low-power setting (not available in our clinics).

Clinical outcomes

- The primary expected clinical outcomes from scaling and root planing are reductions in bleeding and probing depth and a gain in clinical attachment.
- Comparing power scalers with hand instruments, both types demonstrate similar outcomes
- Because the opening of a furcation is narrower than with conventional hand instruments, power scalers may be recommended as a means to improve access when scaling this type of defect.

Special Considerations

- Power-driven instruments must be used with some caution.
- Roots may be rougher post scaling than with hand instruments. Power driven instruments may increase the roughness of resin or glass ionomer restorative materials; therefore, repolishing post scaling is recommended
- Due to aerosol production, proper infection control procedures need to be implemented. Power-driven devices produce bioaerosols and splatter, which can contaminate the operator and remain in the air for up to 30 minutes. If patient has been diagnosed with contagious disease (droplet transmission), it is recommended to use hand instrument . Or using good infection control practices to minimize the hazard:
  - preprocedural rinsing with 0.12% chlorhexidine and
  - high-speed evacuation
  - A minimum of 30 min time period between patients is recommended

  These are the most efficient ways to reduce bioaerosols

Cardiac Pacemakers

- The use of ultrasonics on patients with cardiac pacemakers is somewhat controversial.
- Newer models of pacemakers often have bipolar titanium insulation that is believed to make ultrasonic and sonic instruments generally safe for use.
- An in vivo study supports this; 12 patients underwent continuous electrocardiogram monitoring during piezoelectric ultrasonic scaling and had no abnormal pacemaker functions. Conversely, an in vitro study found that ultrasonic scalers interfered with the activity of dual-system pacemakers.
- If in doubt, consult with the physician regarding any precautions or warnings from the manufacturer of the product.

BOX 51.2 Indications, Precautions, and Contraindications for Use of Mechanized Instruments

Indications

- Supragingival debridement of dental calculus and extrinsic stains
- Subgingival debridement of calculus, oral biofilm, root surface constituents, and periodontal pathogens
- Removal of orthodontic cement
- Gingival and periodontal conditions and diseases
- Surgical interventions
- Margination (reduces amalgam overhangs)

Precautions

- Unshielded pacemakers
- Infectious diseases: human immunodeficiency virus, hepatitis, tuberculosis (active stages)
- Demineralized tooth surface
- Exposed dentin (especially associated with sensitivity)
- Restorative materials (porcelain, amalgam, gold, composite)
- Titanium implant abutments unless using special insert (e.g., Quixonic SofTip Prophy Tips)
- Children (primary teeth)
- Immunosuppression from disease or chemotherapy
- Uncontrolled diabetes mellitus

Contraindications

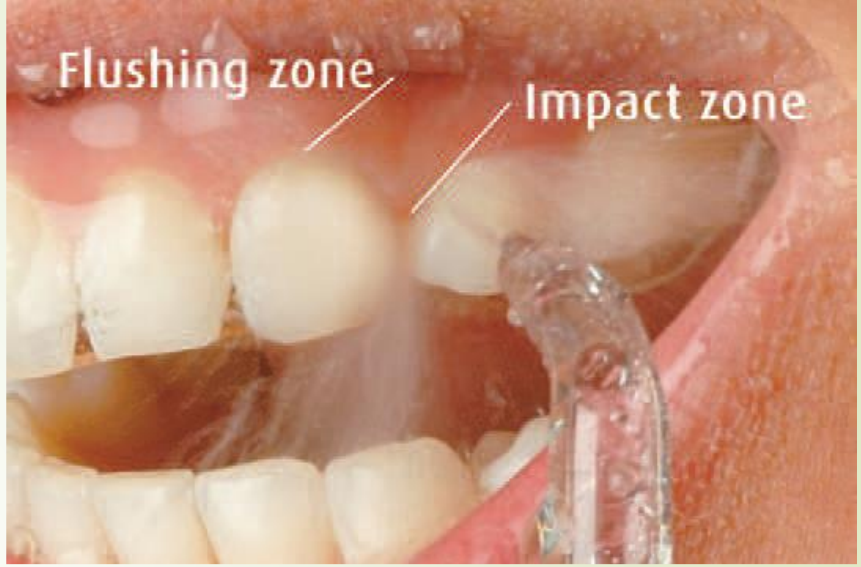
- Chronic pulmonary disease: asthma, emphysema, cystic fibrosis, pneumonia
- Cardiovascular disease with secondary pulmonary disease
- Swallowing difficulty (dysphagia)

- Instrumentation with the ultrasonic device is different from hand instrumentation.
- A pen grasp with light pressure is preferred, as is using an extraoral fulcrum.
- Deposits are removed coronally to apically.
- For deposits in the embrasure area, a horizontal or transverse stroke is recommended.
- A deplaquing stroke should be used when the focus is removal of biofilm and soft debris for the resolution of gingival inflammation. This stroke entails accessing every square millimeter

Home and Self-Applied Irrigation

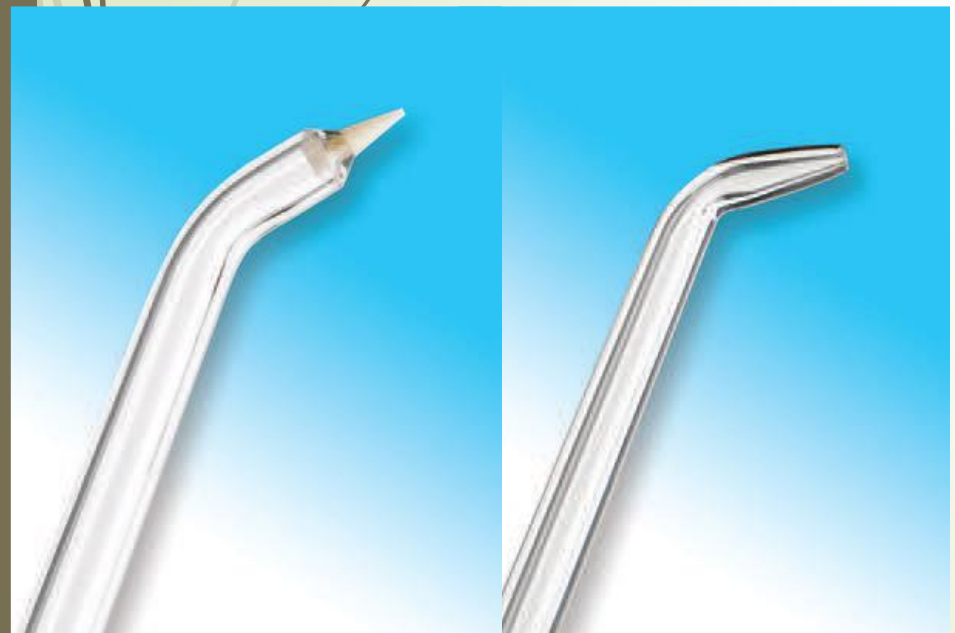
- The oral irrigator (also called a dental water jet or water flosser) was introduced in 1962.
- Contrary to myth and misunderstanding,
- Emerging evidence indicates that the oral irrigator effectively removes biofilm
- As effective as dental floss when added to toothbrushing
- No adverse events have been reported

Mechanism of Action of Irrigation



- pulsation and pressure phase.
- Pulsation creates a decompression phase that allows the water or solution to penetrate subgingivally.
- Followed by a compression phase that expels bacteria and debris from the pocket.
- Physiologically, pulsation, along with pressure and water velocity, creates shear hydraulic forces that are capable of removing bacterial biofilm from treated areas.
- Clinical efficacy 1200 to 1400 pulses per minute set at a minimum of 60 psi.
- The oral irrigator is safe to use at higher pressure settings.
- Many types of oral irrigators are commercially available, but as with other self-care products, research available from one product brand should not be extrapolated to other brands, because they may have used different pressure settings and pulsation rates

- A variety of tips can be used with an oral irrigator.
- Tips placed above the gingival margin at 90 degree angle result in a pocket penetration of 50% on average
- Tips that are placed supragingivally are recommended for full-mouth irrigation or cleansing.
- These tips include a traditional jet tip, with bristles or filaments



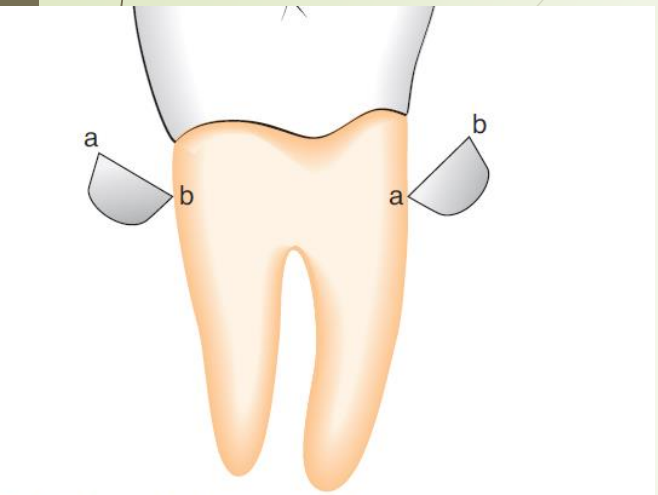
- The soft, site-specific subgingival tip penetrates to about 90% of the 6mm PD And 64% of the 7 mm PD
- The subgingival tip is generally used after full-mouth cleaning for localized irrigation of a specific site that is difficult to access, such as a deep pocket, a furcation, an implant, or a crown and bridge

- Evaluated outcomes include removal of plaque biofilm and reductions in calculus, gingivitis, bleeding on probing, probing depth, periodontal pathogens, and inflammatory mediators
- The use of an antimicrobial agent, such as diluted chlorhexidine, or an essential oil generally enhances reductions in gingivitis and bleeding.
- Irrigation for a period of 3 to 6 months results in reduced periodontal bacteria.
- Small improvements in probing depth.
- Incidence of bacteremia can range from 7% in people with gingivitis to 50% in those with periodontitis.
- In comparison, the incidence of bacteremia from string flossing has been shown to be 40% in people with periodontitis and 41% in periodontally healthy individuals.

Individuals With Special Considerations

- Both children and adults undergoing orthodontic therapy have shown significant benefits from using a dental water jet.
- For individuals with implants, a modified jet tip with filaments has been found to be both safe and effective. Patients who used the oral irrigator at 60 psi with warm water, had twice the reduction in bleeding around implants compared with patients who used floss. No adverse events were reported.
- The site-specific subgingival tip has also been shown to be safe and effective for use on implants.
- Oral irrigator has also been found to improve periodontal health in people with type 1 or 2 diabetes.

Root Planing *(Universal Curettes)*



50.23 Adaptation of the universal curette on a posterior tooth.

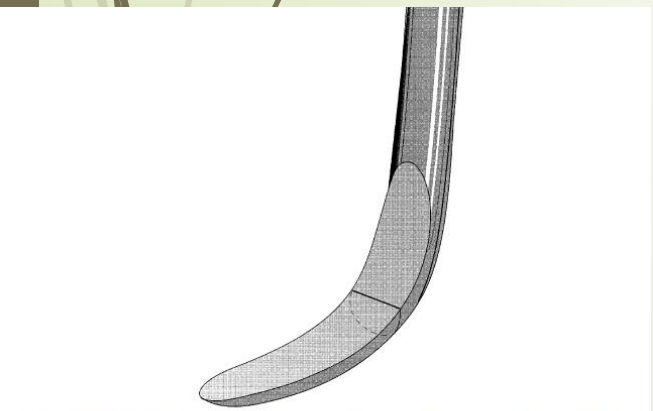
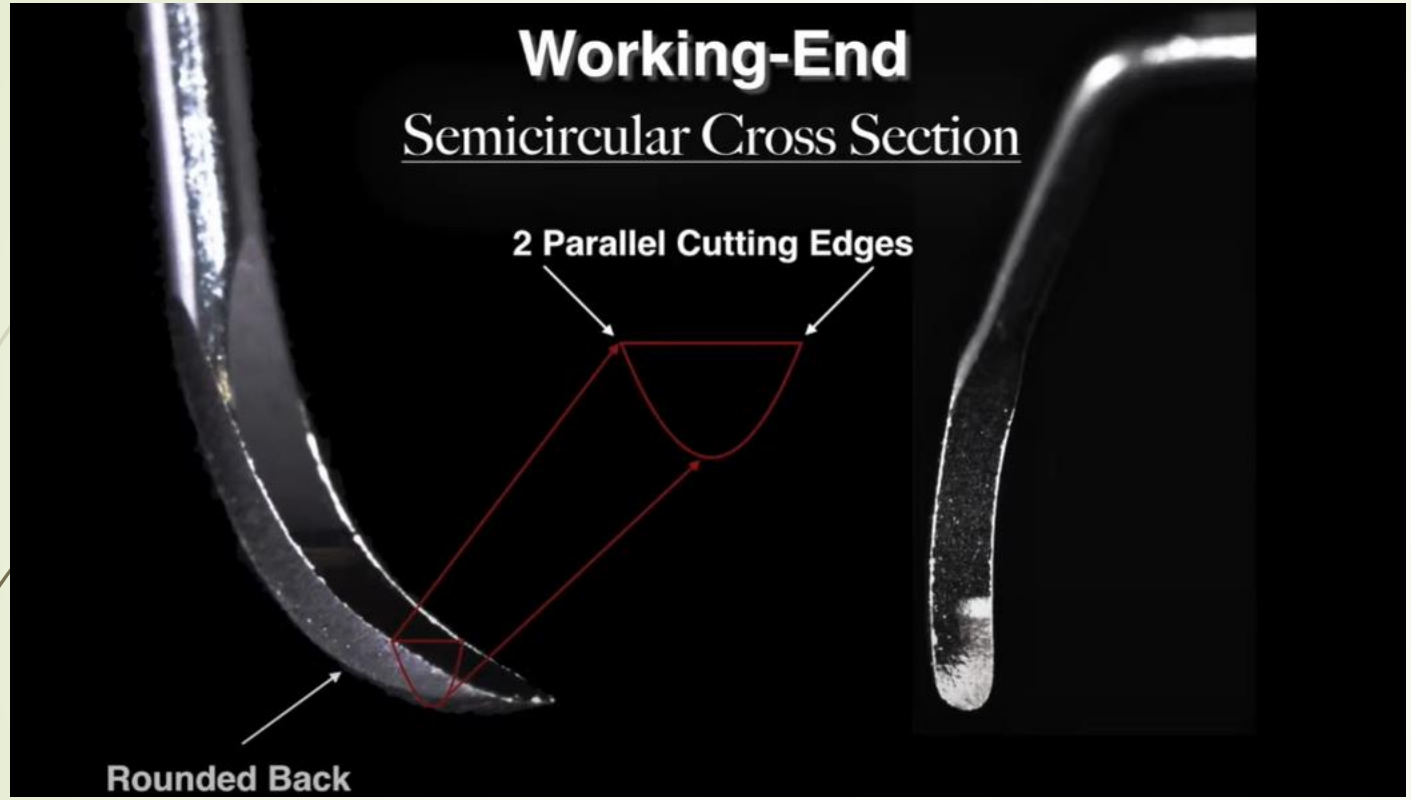
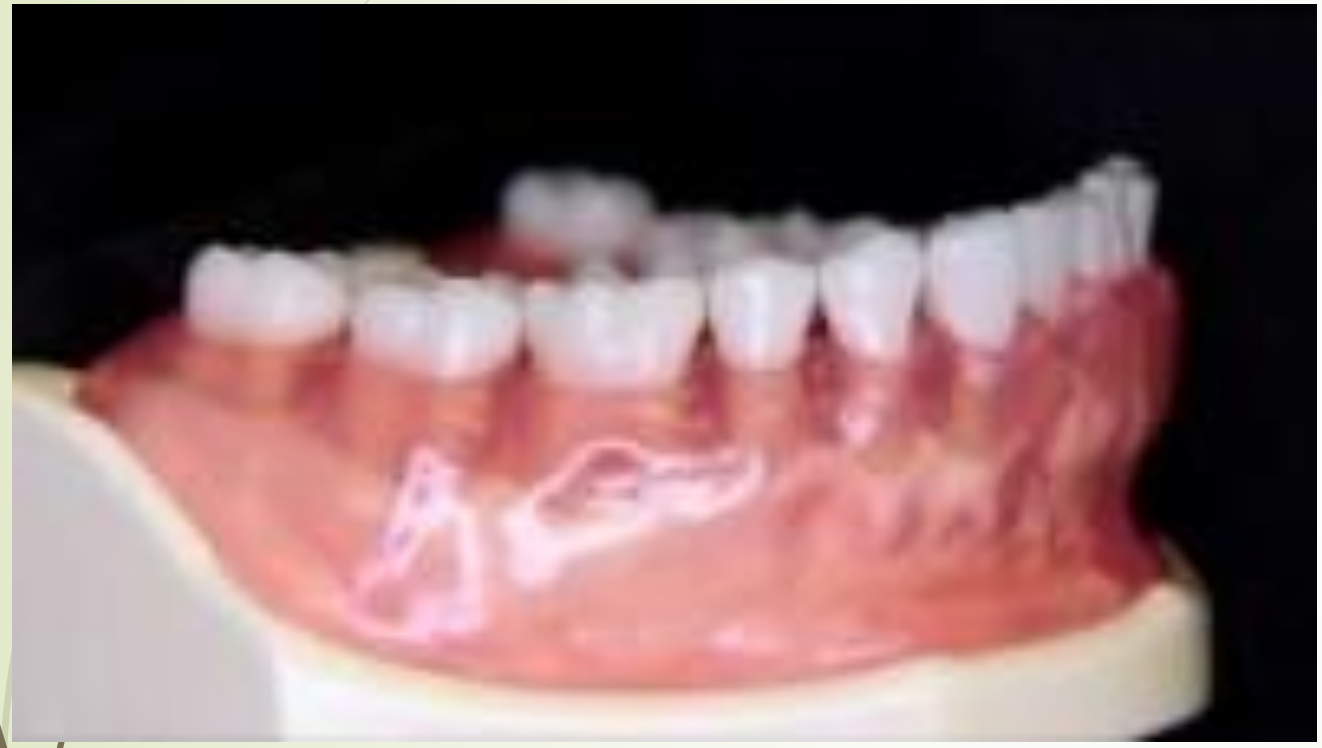


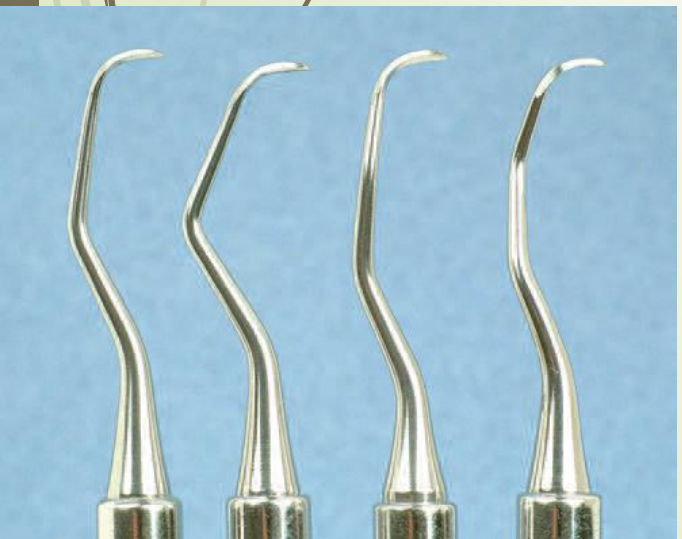
Fig. 50.14 Basic characteristics of a curette: spoon-shaped blade and rounded tip.

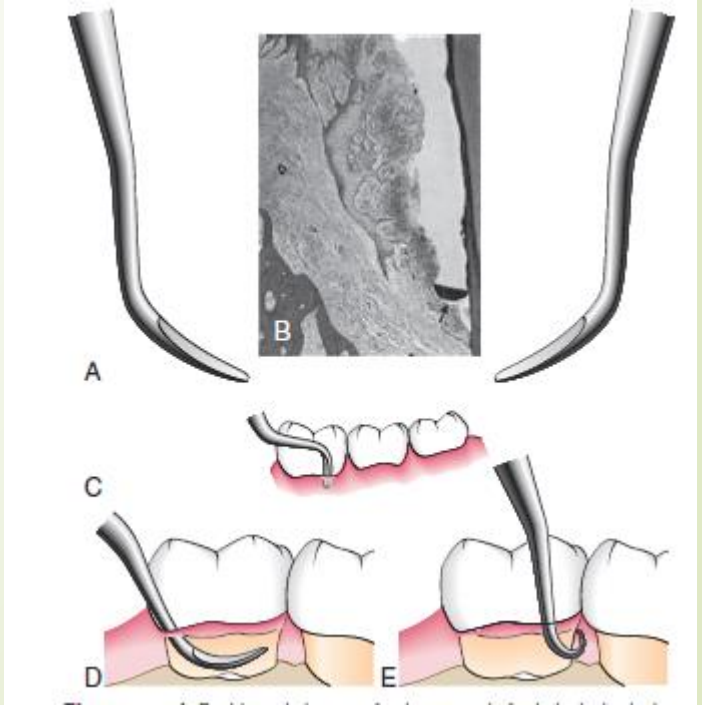
- The working ends of the universal curette are designed in pairs so that all surfaces of the teeth can be treated with one double-ended instrument or a matched pair of single-ended instruments.
- Both cutting edges of the universal curette blade are used.
- In any given quadrant, when approaching the tooth from the facial aspect, one end of the universal curette adapts to the mesial surfaces and the other end adapts to the distal surfaces. When approaching from the lingual aspect in the same quadrant, the double ended universal curette must be turned end for end because the blades are mirror images. This means that the end that adapts to the mesial surfaces on the facial aspect also adapts to the distal surfaces on the lingual aspect, and vice versa.
- Both ends of the universal curette are used for instrumentation of the anterior teeth.
- On posterior teeth, however, because of the limited access to distal surfaces, a single working end can be used to treat both mesial and distal surfaces by using both of its cutting edges. To do this, the instrument is first adapted to the mesial surface with the handle nearly parallel to the mesial surface. Because the face of the universal curette blade is honed at 90 degrees to the lower shank. the lower shank must be tilted slightly toward the tooth.
- The distal surface of the same posterior tooth can be instrumented with the opposite cutting edge of the same blade. This cutting edge can be adapted at proper working angulation by positioning the handle so that it is *perpendicular* to the distal surface.


Working-End
Semicircular Cross Section
2 Parallel Cutting Edges
Rounded Back



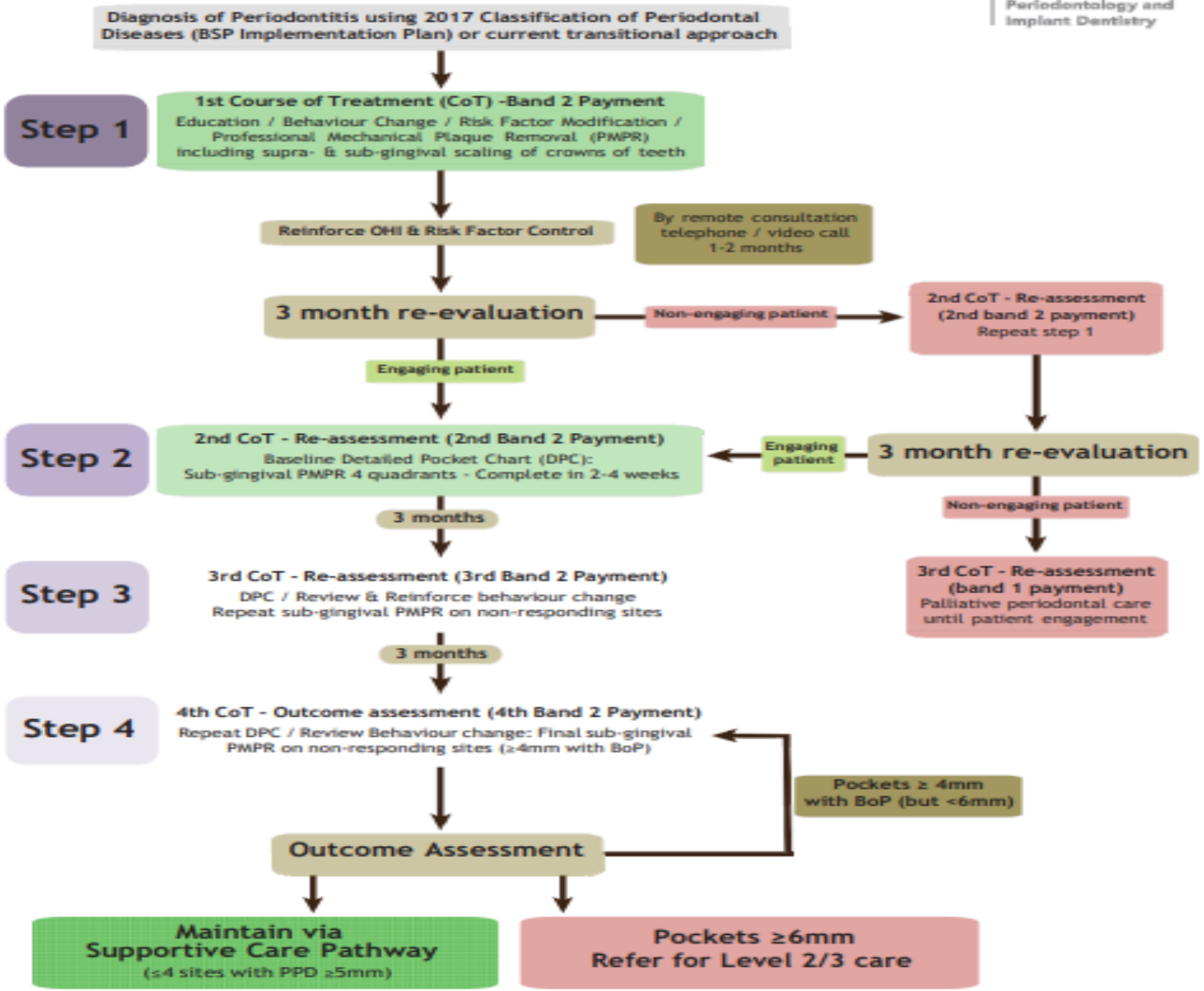
- When adapting the universal curette blade, as much of the cutting edge as possible should be in contact with the tooth surface, except on narrow convex surfaces such as line angles.
- When the cutting edge is adapted to the tooth, pressure should be concentrated on the lower third of the blade during scaling and root planing strokes.
- The primary advantage of these curettes is that they are designed to be used universally on all tooth surfaces, in all regions of the mouth.
- However, universal curettes have limited adaptability for the treatment of deep pockets in which apical migration of the attachment has exposed furcations, root convexities, and developmental depressions.
- For this reason, many clinicians prefer Gracey curettes and the newer modifications of Gracey curettes, which are area specific and specially designed for subgingival scaling and root planing in periodontal patients.





Phased Management of Periodontitis in NHS General Dental Practice – Full Care Pathway adapted to UDA Banding

BSP British Society of Periodontology and Implant Dentistry



Notes:
Remote consultation by dentist / hygienist /therapist or Oral Health Educator
Non-engaging pts offered a 2nd band 2 STEP-1 attempt to engage, then 3/12ly Band 1 Step 1 until engage

Thank you