

CHAPTER III. ASSESSMENT

The Assessment component of **SOAP** includes 3 components:

- i. Diagnosis
- ii. Etiology
- iii. Prognosis

i. Diagnosis

a) **Classification System.**

Diagnosis of Periodontal Diseases and Conditions is based upon the classification system developed at the 1999 International Workshop for a Classification of Periodontal Diseases and Conditions. (See Armitage, G.C. Development of a Classification System for Periodontal Diseases and Conditions. Annals of Periodontology 4 (1): 1-6, 1999)

Refer to List III. A . below: Classification of Periodontal Diseases and Conditions

Note that each of the findings documented in the Observation component of SOAP (Chapter II) should be included by a diagnosis.

One approach to reconciling patient symptoms/history (Subjective) and findings (Observations) with diagnoses is to use the Classification System (List III.A) as a checklist: include all relevant diagnoses that are supported by Subjective and Objective data.

Presumptive and Final Diagnoses

Diagnoses may be described as either presumptive or final:

PRESUMPTIVE : Diagnosis is made after completion of the initial examination and it is used to guide therapy.

FINAL : Diagnosis is made at the re-evaluation examination which is performed 4-8 weeks following the initial phase of therapy (Phase I etiologic phase – refer to Chapter IV Planning)
The final diagnosis is used to guide further therapy.

- Note that one or more diagnosis per patient is likely. The diagnoses in List III. A are NOT mutually exclusive; several diagnoses are likely. For example, data may support the following diagnoses in the same patient at the same time:

- I. Gingival Disease
 - A. Plaque-induced gingival disease
 - 2. Gingival Disease Modified by systemic factors
 - a. associated with endocrine system
 - 1) pregnancy-associated gingivitis – localized, moderate
 - B. Non-plaque-induced gingival lesions
 - 5. Gingival manifestations of systemic conditions
 - a. mucocutaneous disorders
 - 1) lichen planus
- II. Chronic Periodontitis
 - A. Generalized - initial
 - B. Localized – moderate
- VI. Abscesses of the Periodontium
 - B. Periodontal abscess
- VII. Periodontitis Associated with Endodontic Lesions
 - A. Combined periodontic-endodontic lesion
- VIII. Developmental or Acquired Deformities and Conditions
 - A. Localized tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis
 - 1. Tooth anatomic factors
 - 2. Dental restorations/appliances
 - B. Mucogingival deformities and conditions around teeth
 - 1. Gingival/soft tissue recession
 - a. facial or lingual surfaces
 - b. interproximal (papillary)
 - 2. Lack of keratinized gingiva
 - D. Occlusal Trauma
 - 2. Secondary occlusal trauma

List A. Classification System for Periodontal Diseases and Conditions

- I. Gingival Diseases
 - A. Dental plaque-induced gingival diseases*
 1. Gingivitis associated with dental plaque only
 - a. without other local contributing factors
 - b. with local contributing factors (See VIII A)
 2. Gingival diseases modified by systemic factors
 - a. associated with the endocrine system
 - 1) puberty-associated gingivitis
 - 2) menstrual cycle-associated gingivitis
 - 3) pregnancy-associated
 - a) gingivitis
 - b) pyogenic granuloma
 - 4) diabetes mellitus-associated gingivitis
 - b. associated with blood dyscrasias
 - 1) leukemia-associated gingivitis
 - 2) other
 3. Gingival diseases modified by medications
 - a. drug-influenced gingival diseases
 - 1) drug-influenced gingival enlargements
 - 2) drug-influenced gingivitis
 - a) oral contraceptive-associated gingivitis
 - b) other
 4. Gingival diseases modified by malnutrition
 - a. ascorbic acid-deficiency gingivitis
 - b. other
 - B. Non-plaque-induced gingival lesions
 1. Gingival diseases of specific bacterial origin
 - a. *Neisseria gonorrhoea*-associated lesions
 - b. *Treponema pallidum*-associated lesions
 - c. streptococcal species-associated lesions
 - d. other
 2. Gingival diseases of viral origin
 - a. herpesvirus infections
 - 1) primary herpetic gingivostomatitis
 - 2) recurrent oral herpes
 - 3) varicella-zoster infections
 - b. other
3. Gingival diseases of fungal origin
 - a. *Candida*-species infections
 - 1) generalized gingival candidosis
 - b. linear gingival erythema
 - c. histoplasmosis
 - d. other
4. Gingival lesions of genetic origin
 - a. hereditary gingival fibromatosis
 - b. other
5. Gingival manifestations of systemic conditions
 - a. mucocutaneous disorders
 - 1) lichen planus
 - 2) pemphigoid
 - 3) pemphigus vulgaris
 - 4) erythema multiforme
 - 5) lupus erythematosus
 - 6) drug-induced
 - 7) other
 - b. allergic reactions
 - 1) dental restorative materials
 - a) mercury
 - b) nickel
 - c) acrylic
 - d) other
 - 2) reactions attributable to
 - a) toothpastes/dentifrices
 - b) mouthrinses/mouthwashes
 - c) chewing gum additives
 - d) foods and additives
 - 3) other
6. Traumatic lesions (factitious, iatrogenic, accidental)
 - a. chemical injury
 - b. physical injury
 - c. thermal injury
7. Foreign body reactions
8. Not otherwise specified (NOS)

- II. Chronic Periodontitis[†]
 - A. Localized
 - B. Generalized
- III. Aggressive Periodontitis[†]
 - A. Localized
 - B. Generalized
- IV. Periodontitis as a Manifestation of Systemic Diseases
 - A. Associated with hematological disorders
 - 1. Acquired neutropenia
 - 2. Leukemias
 - 3. Other
 - B. Associated with genetic disorders
 - 1. Familial and cyclic neutropenia
 - 2. Down syndrome
 - 3. Leukocyte adhesion deficiency syndromes
 - 4. Papillon-Lefèvre syndrome
 - 5. Chediak-Higashi syndrome
 - 6. Histiocytosis syndromes
 - 7. Glycogen storage disease
 - 8. Infantile genetic agranulocytosis
 - 9. Cohen syndrome
 - 10. Ehlers-Danlos syndrome (Types IV and VIII)
 - 11. Hypophosphatasia
 - 12. Other
 - C. Not otherwise specified (NOS)
- V. Necrotizing Periodontal Diseases
 - A. Necrotizing ulcerative gingivitis (NUG)
 - B. Necrotizing ulcerative periodontitis (NUP)
- VI. Abscesses of the Periodontium
 - A. Gingival abscess
 - B. Periodontal abscess
 - C. Pericoronal abscess
- VII. Periodontitis Associated With Endodontic Lesions
 - A. Combined periodontic-endodontic lesions
- VIII. Developmental or Acquired Deformities and Conditions
 - A. Localized tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis
 - 1. Tooth anatomic factors
 - 2. Dental restorations/appliances
 - 3. Root fractures
 - 4. Cervical root resorption and cemental tears
 - B. Mucogingival deformities and conditions around teeth
 - 1. Gingival/soft tissue recession
 - a. facial or lingual surfaces
 - b. interproximal (papillary)
 - 2. Lack of keratinized gingiva
 - 3. Decreased vestibular depth
 - 4. Aberrant frenum/muscle position
 - 5. Gingival excess
 - a. pseudopocket
 - b. inconsistent gingival margin
 - c. excessive gingival display
 - d. gingival enlargement (See I.A.3. and I.B.4.)
 - 6. Abnormal color
 - C. Mucogingival deformities and conditions on edentulous ridges
 - 1. Vertical and/or horizontal ridge deficiency
 - 2. Lack of gingiva/keratinized tissue
 - 3. Gingival/soft tissue enlargement
 - 4. Aberrant frenum/muscle position
 - 5. Decreased vestibular depth
 - 6. Abnormal color
 - D. Occlusal trauma
 - 1. Primary occlusal trauma
 - 2. Secondary occlusal trauma

I. Gingival Diseases. Discussion of gingival diseases in this manual is limited to Plaque-induced gingival diseases.

A. Dental plaque-induced gingival diseases *

1. Gingivitis associated with dental plaque only

NOTE: * may occur on a periodontium without attachment loss

OR

*on a periodontium with attachment loss that is not progressing

Table III. 1 Characteristics Common to All Plaque-Induced Gingival Diseases

- Includes gingival diseases associated with plaque, endogenous hormonal fluctuations, drugs, systemic diseases, malnutrition
- Signs and symptoms are confined to the gingiva
- The presence of dental plaque is required to initiate and/or exacerbate the severity of the lesion
- Clinical signs of inflammation are present:
 - Enlarged gingival contours due to edema or fibrosis
 - Color change to red (erythema) and/or bluish/red (cyanosis)
 - Bleeding upon stimulation (eg. probing)
 - Increased gingival exudate
 - Elevated sulcular temperature
- Clinical signs and symptoms are associated with stable attachment levels either on
 - * a periodontium with no attachment loss OR
 - * a stable but reduced periodontium
- The clinical signs and symptoms are REVERSIBLE by removing the etiology/etiologies
- Gingival Diseases play a *possible* role as precursor to attachment loss around teeth

Table III. 2. Characteristics of Plaque-Induced Gingivitis on Periodontium WITHOUT Attachment Loss

- Absence of attachment loss
- Absence of bone loss
- Plaque present at the gingival margin
- Disease begins at the gingival margin
- Change in gingival color (erythema or cyanosis)
- Change in gingival contour (enlargement due to edema or fibrosis)
- Elevated sulcular temperature
- Increased gingival exudate
- Bleeding upon provocation
- Histological changes
- REVERSIBLE with plaque removal

Table III. 3. Characteristics of Plaque-Induced Gingivitis on a Reduced Periodontium

- Pre-existing attachment loss or bone loss may be present but periodontal treatment has resulted in a REDUCED but STABLE connective tissue attachment and stable alveolar bone height
- There is RETURN of plaque-induced inflammation at the gingival margin but NO evidence of progressive attachment loss
- Clinical signs and symptoms of plaque-induced gingivitis on reduced periodontium are IDENTICAL to characteristics listed in Table III.2.

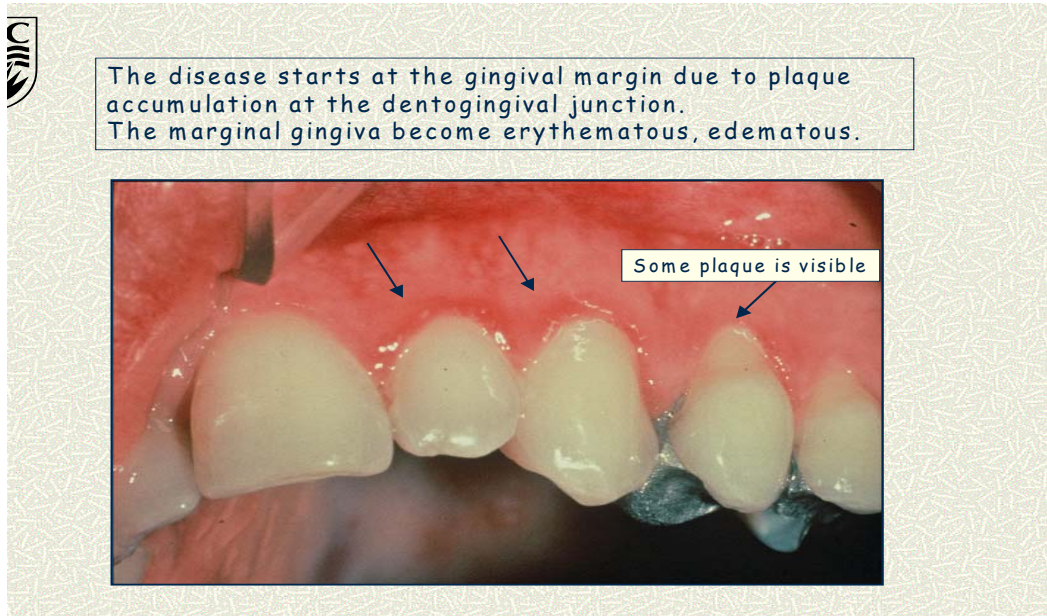


Figure III.1. Early stage of plaque-induced gingival inflammation.
Changes in color and contour begin at the gingival margin

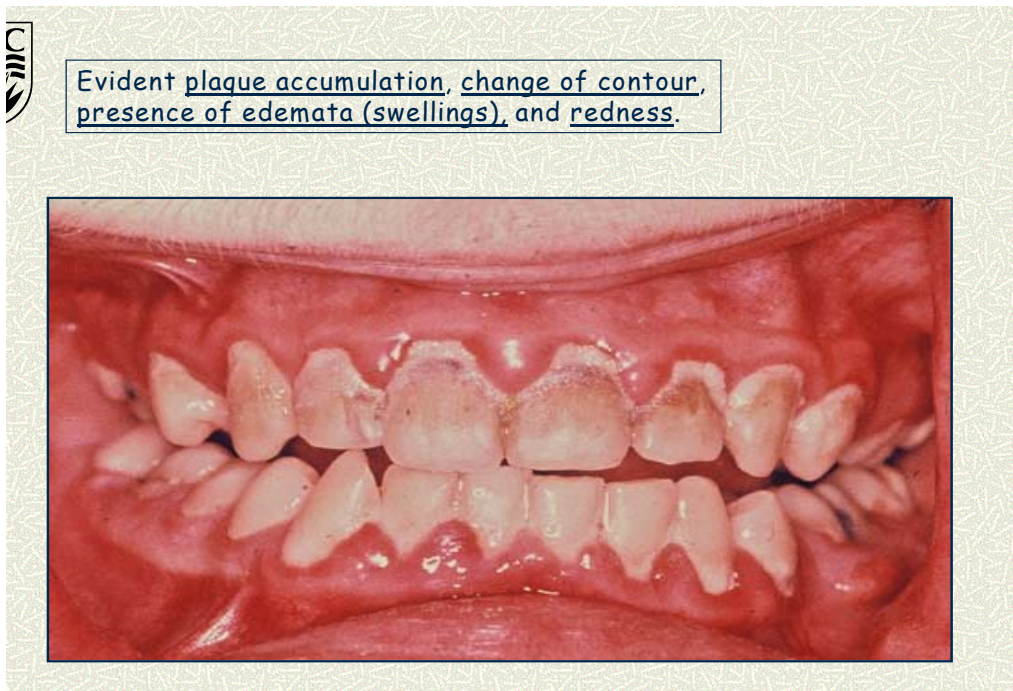


Figure III.2. Advanced stage of plaque-induced gingival inflammation.
Changes in color, contour/texture (edema) involve the marginal and papillary tissues.

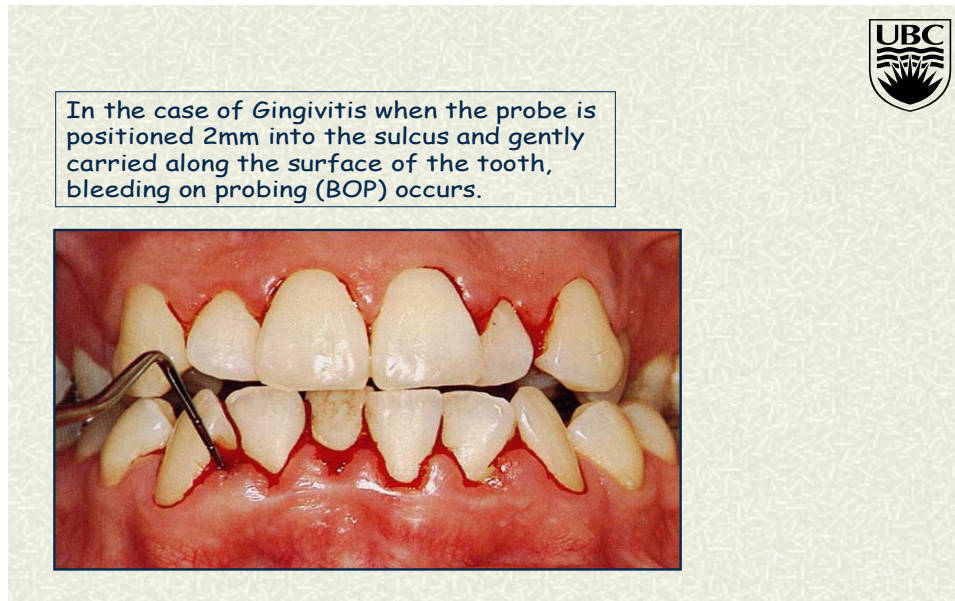


Figure III. 3. Bleeding upon provocation (bleeding).

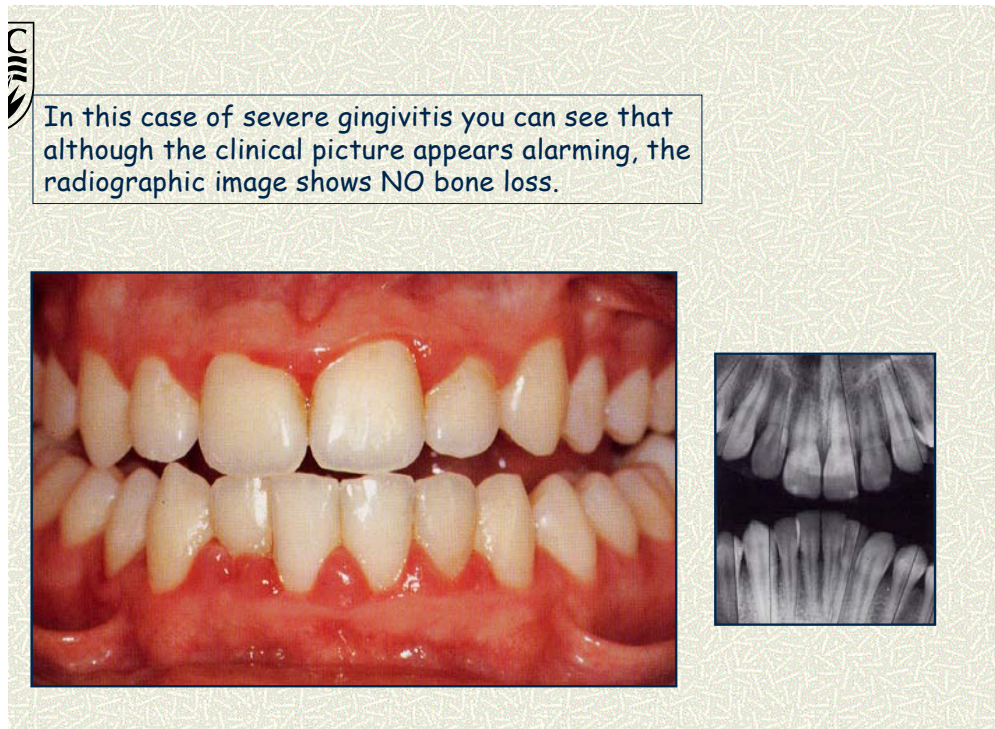


Figure III. 4. Plaque-induced gingival inflammation in the absence of attachment loss or bone loss.

Stages of Gingivitis			
Stage	I. <u>Initial Lesion</u>	II. <u>Early Lesion</u>	III. <u>Established Lesion</u>
Time (days)	2-4 day	4-7 days	14-21 days
Blood Vessels	Vascular dilation/ Vasculitis	Vascular proliferation	Same as Stage II, plus blood stasis
Junctional & Sulcular Epithelium	Infiltrated by PMNs	Same as Stage I, Rete peg formation Atrophic areas	Same as Stage II, but more advanced
Predominant Immune Cells	PMNs	Lymphocytes	Plasma cells
Collagen	Perivascular loss	Increased loss around infiltrate	Continued loss
Clinical Findings	Gingival fluid flow	Erythema Bleeding on Probing Changes in color, size, texture, etc.	

Table III. 1. Stages of Plaque-induced Gingival Inflammation.

Histopathologic changes include:

- proliferation of basal junctional epithelium leading to apical and lateral cell migration
- vasculitis of blood vessels adjacent to the junctional epithelium
- progressive destruction of the collagen fibers
- progressive inflammatory/immune cellular infiltrate

As the inflammation progresses, there is an increase in sulcular temperature and in sulcular exudate.



Figure III. 5. Qualification of plaque-induced gingival inflammation.

Inflammation may be qualified as to

- Severity:
 - Mild
 - Moderate
 - Severe
- Distribution:
 - Localized
 - Generalized

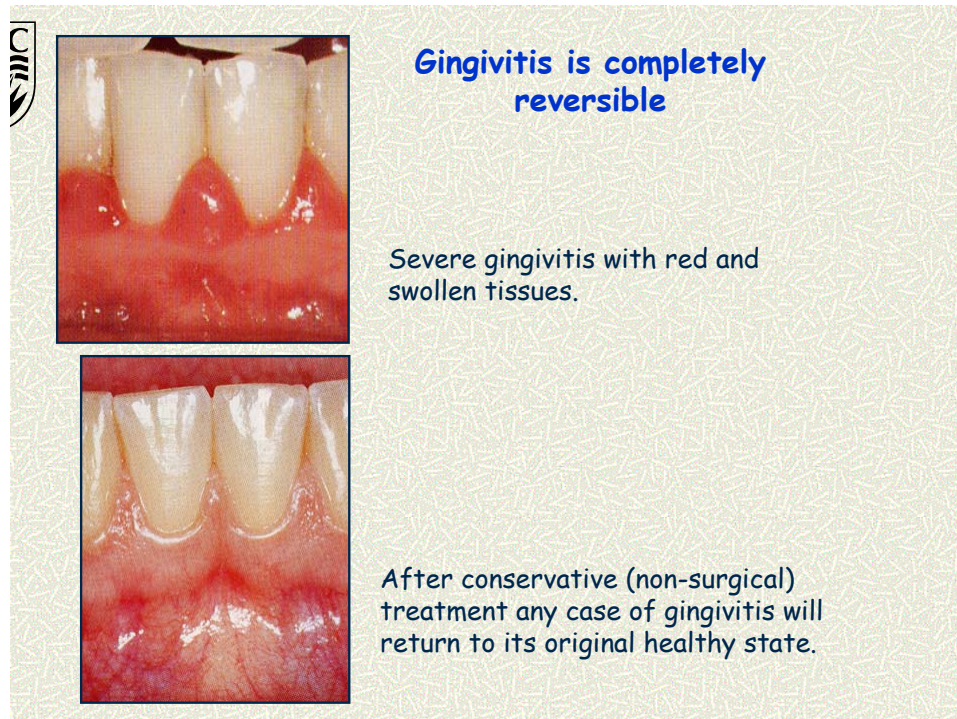


Figure III. 6. Plaque-induced gingival inflammation is reversible if the etiologic factors are eliminated.

Local Contributing Factors that modify or predispose to plaque-induced gingival disease include:

- Calculus
- Tooth anatomic factors
- Dental restorations, appliances
- Root fractures
- Cervical root resorption, cemental tears

A. Dental plaque-induced gingival diseases *

2. *Gingival disease associated with systemic factors*

a. *Endocrine System*

Table III. 4. Characteristics of Puberty-Associated Gingivitis

- Plaque present at gingival margin
- Pronounced inflammatory response of gingiva
- MUST be circumpubertal in occurrence
- Change in gingival color (erythema, cyanosis)
- Change in gingival contour with possible change of gingival size (enlargement)
- Increased gingival exudate
- Bleeding upon provocation (probing)
- Absence of attachment loss
- Absence of bone loss
- Reversible following puberty and removal of plaque

Table III. 5. Characteristics of Menstrual Cycle-Associated Gingivitis

- Plaque present at gingival margin
- Modest inflammatory response of gingiva prior to ovulation
- Must be at ovulatory surge
- Increase in gingival exudate by at least 20% during ovulation
- Absence of attachment loss, bone loss
- Reversible following ovulation and removal of plaque

Table III. 6. A. Characteristics of Pregnancy-Associated Gingivitis

- Plaque present at gingival margin
- Pronounced inflammatory response of gingiva
- Onset is in pregnancy (2nd or 3rd trimester)
- Change in gingival color, gingival contour
- Increase in gingival exudate
- Bleeding upon provocation
- Absence of attachment loss, bone loss
- Reversible at parturition and elimination of plaque

Table III. 6.B. Characteristics of Pregnancy-Associated Pyogenic Granuloma

- Plaque present at gingival margin
- Pronounced inflammatory response of gingiva
- Can occur anytime during pregnancy
- More common in maxilla
- More common interproximally
- Sessile or pedunculated protuberant mass
- Not a neoplasm; has histologic appearance of pyogenic granuloma
- Regresses following parturition

**Table III. 7. Characteristics of
Diabetes Mellitus-Associated Gingivitis**

- Plaque present at gingival margin
- Pronounced inflammatory response of gingiva
- Change in gingival color and contour
- Increased gingival exudate
- Bleeding upon provocation
- Most commonly associated in children with poorly controlled Type I DM
- Absence of bone loss/attachment loss
- Reversible with control of diabetic state
- Reduction of dental plaque can limit severity of lesion

2 . Gingival Diseases modified by systemic factors

b . associated with blood dyscrasias

1) leukemia-associated gingivitis

Table III. 8. Characteristics of Leukemia-Associated Gingivitis

- Pronounced inflammatory response of gingiva in relation to the plaque present; however, plaque is NOT prerequisite for oral lesions
- Gingival lesions are primarily found in acute leukemias
- Change in gingival color
- Change in gingival contour with possible modification of gingival size
- Enlargement first observed at the interdental gingiva
- Bleeding upon provocation (may be one of the initial oral signs)
- Reductions in dental plaque can limit the severity of the lesion

3. Gingival Diseases modified by medications
a. drug-influenced gingival enlargements

**Table III. 9. Characteristics of Drug-Influenced
Gingival Enlargement**

- Variation in interpatient and inpatient pattern
- Predilection for anterior gingiva
- Higher prevalence in children
- Onset within 3 months of medication start
- Change in gingival contour leading to modification of gingival size
- Enlargement first observed in at the interdental gingiva
- Change in gingival color
- Increased gingival exudate
- Bleeding upon provocation
- Found in gingiva with or without bone loss but is NOT associated with attachment loss
- Pronounced inflammatory response of gingiva in relation to the plaque present
- Reductions in dental plaque can limit the severity of the lesion
- Must be using phenytoin, cyclosporine A, or certain calcium channel blockers; the plasma concentrations to induce the lesion have not been clearly defined in humans

Table III. 10. Characteristics of Oral Contraceptive-Associated Gingivitis

- Plaque present at gingival margin
- Pronounced inflammatory response of gingiva
- Change in gingival color
- Change in gingival contour with possible modification of gingival size
- Increased gingival exudate
- Bleeding upon provocation
- Reversible following discontinuation of oral contraceptives; removal of plaque

II. Chronic Periodontitis

Gingivitis is inflammation limited to the gingival

In contrast, periodontitis

- ✓ is inflammation of the supporting tissues of the teeth.
- ✓ is usually a progressively destructive change leading to loss of bone and periodontal ligament (PDL)
- ✓ includes an extension of inflammation from gingiva into the bone and PDL

Table III. 11. Histopathologic Features of Periodontitis

- **Periodontal pocketing**
 - ✓ Apical migration of the JE as compared to the CEJ
 - ✓ Loss of collagen fibers subjacent to the pocket epithelium
- **Bone loss**
 - ✓ PMN infiltration of junctional and pocket epithelium
 - ✓ Dense inflammatory cell infiltrate with plasma cells, lymphocytes and macrophages



✓ Microbiological features

Current concepts of the etiology of periodontitis implicate a bacterial infection as the primary cause of the disease.

Putative periodontal pathogens include:

- *Porphyromonas gingivalis* (P.g.)
- *Actinobacillus actinomycetemcomitans* (A.a.)
- *Bacteroides Forsythus* (B.f.)
- Non-classified spirochetes
- *Prevotella intermedia* (P.I.)
- *Cambylobacter rectus* (C.r.)
- *Eubacterium species*
- *Treponema denticola* (T.d.)
- *Fusobacterium nucleatum* (F.n.)
- *Eikenella corrodens* (E.c.)

It appears that various complexes of putative periodontal pathogens can initiate and perpetuate the disease in a susceptible host.

Table III. 12. Microbiological Features of Periodontitis

There are different types of periodontitis (refer to List III. A). Discussion of periodontitis in this manual will focus upon Chronic Periodontitis as it is the most prevalent type of periodontitis.

Table III. 13. CHRONIC PERIODONTITIS

Clinical and characteristics

- Most prevalent in adults but can occur in younger age groups
- Usual onset is after age 35
- Amount of destruction is consistent with the presence of local factors
- Subgingival calculus is a frequent finding
- Associated with a variable microbial pattern
- Slow to moderate progression but may have periods of rapid progression
- Can be further classified by
 - ✓ Extent/distribution: number of sites involved
 - Localized \leq 30% sites affected
 - Generalized $>$ 30 sites affected
 - ✓ Severity: based upon the amount of clinical attachment loss (CAL)
 - Mild 1-2 mm CAL
 - Moderate 3-4 mm CAL
 - Severe \geq 5 mm CAL
- May be modified by and/or associated with systemic disease
- Can be modified by factors other than systemic disease such as
 - ✓ Cigarette smoking
 - ✓ Emotional stress

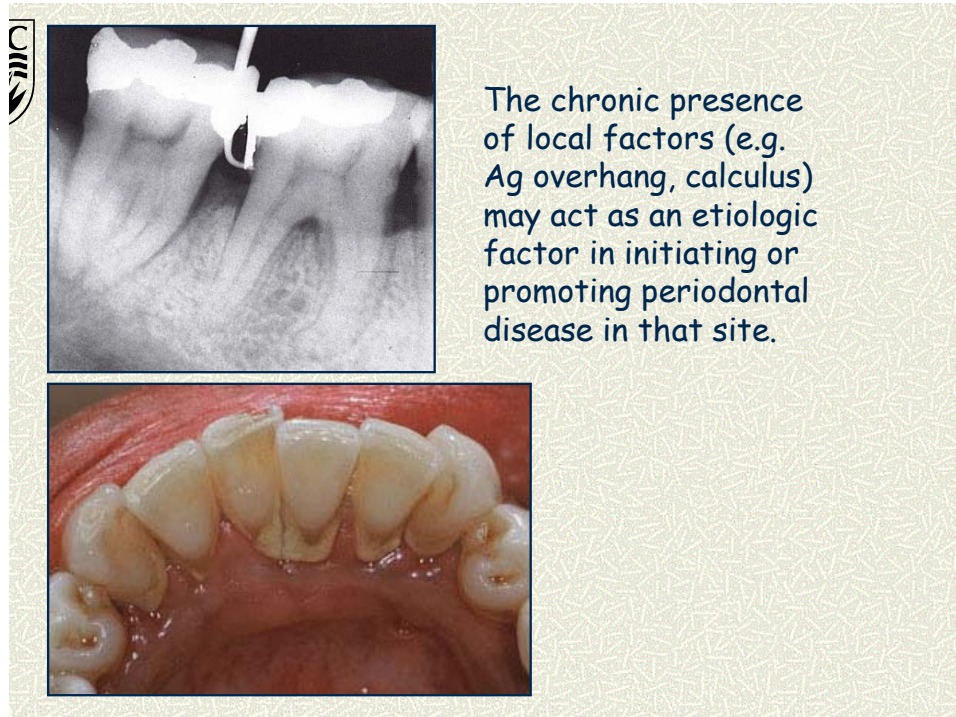


Figure III. 7. Examples of Local Factors: overhang, calculus

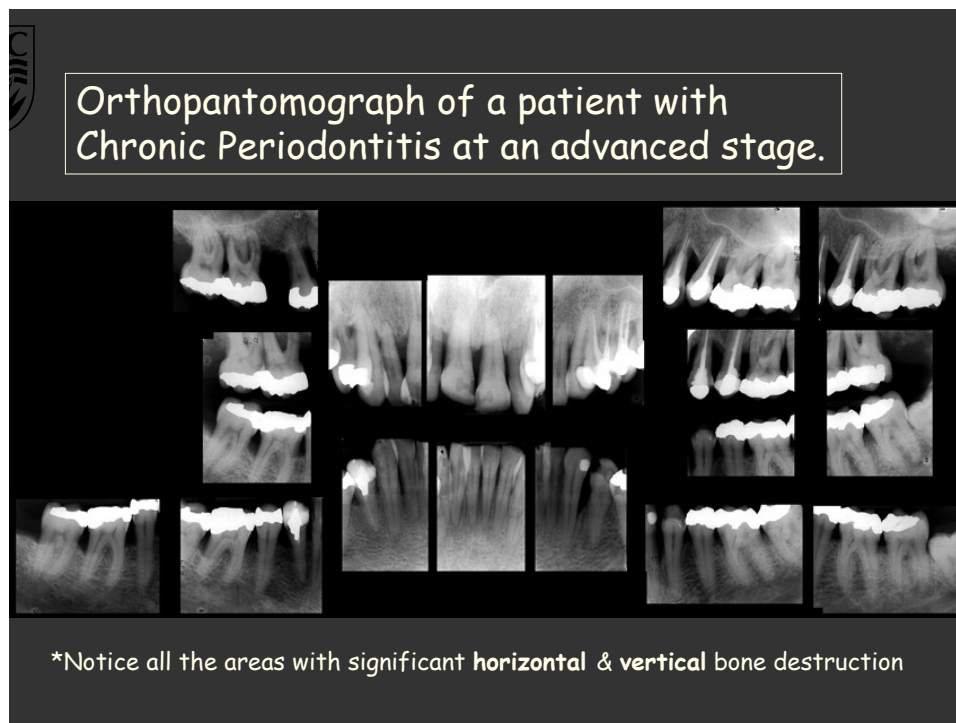


Figure III. 8. Chronic Periodontitis at Advanced Stage

ii. Etiology

Etiologies may be classified as primary or secondary/contributory.

Gingivitis: the Primary etiology of plaque-induced gingival inflammation is PLAQUE.

Periodontitis: The Primary etiology of periodontitis is PLAQUE.

As noted in List III. A. Classification of Periodontal Diseases and Conditions, both plaque-induced gingival inflammation and periodontitis may be associated with and/or modified by a variety of factors. It is important to identify both primary and secondary etiologic factors involved in each diagnosis.

Recall from List III. A. that plaque-induced gingival inflammation may be modified by systemic factors, medications, malnutrition. Periodontitis may be associated with systemic diseases: hematological and/or genetic disorders.

It is also important to include from List III.A. **“VII. Developmental or acquired deformities and conditions”** those factors that may modify or predispose to plaque-induced gingival diseases/periodontitis.

For example, for diagnoses of plaque-induced gingivitis and chronic periodontitis, the primary etiology is plaque. However, in addition to the factors included in List III.A. consider the contributory/compromising roles of

- ✓ Calculus - retains plaque; impedes oral hygiene
- ✓ Improper methods of flossing; aggressive brushing methods with hard toothbrush, etc – contribute to recession, gingival clefts, mucogingival involvements
- ✓ Xerostomia – compromises protective functions of saliva; increases risk of plaque-induced diseases (caries, gingivitis, periodontitis)
- ✓ Mucocutaneous disorders – associated oral discomfort may prevent performance of adequate oral hygiene
- ✓ occlusal habits (clenching, bruxing), gum-chewing habit – factors in primary or secondary trauma; may be etiologic factor for TMD

A discussion of etiologic factors should also include those factors that may complicate/compromise the patient's ability to perform adequate oral hygiene and/or to proceed with recommended periodontal therapy. These associated compromising factors may affect the periodontal prognosis.

- ✓ TMD – arthralgia, myalgia, reduced range of mandibular movements may compromise performance of oral hygiene and professional delivery of periodontal therapy
- ✓ Arthritis involving hands – compromises patient's ability to perform oral hygiene
- ✓ Arthritis of cervical spine – may compromise delivery of periodontal therapy
- ✓ Medical history of neurologic disorders, behavioral and psychiatric disorders may impede patient's participation in maintaining oral health, performing oral hygiene
- ✓ etc

iii. Prognosis

a) Definitions:

- Prognosis is a prediction of the
 - *Duration*
 - *Course* and
 - *Termination* of a disease and its response to treatment.
- Prognosis must be determined *after* the diagnosis is made and *before* treatment is planned
- *Diagnostic Prognosis* is the general prognosis without treatment. In general, the diagnostic prognoses for plaque-induced gingival disease and periodontitis are POOR.
- *Therapeutic Prognosis* is the prognosis with treatment. For periodontitis, therapeutic prognosis is divided into
 - General or overall prognosis for the entire dentition
 - Specific prognosis for individual teeth
- Therapeutic prognosis is influenced by the extent to which the etiology associated with that particular diagnosis can be eliminated or controlled.
 - For example, if an etiology cannot be controlled then the treatment may be compromised. In this manner, smoking habit and/or poorly-controlled diabetes can compromise the success of periodontal treatment.

b) Prognosis for Gingival Disease

- Prognosis for gingival disease depends upon the role of plaque-induced inflammation in the overall disease process.
- If plaque-induced inflammation is the **ONLY** pathologic change, *then* the prognosis is favorable (good) provided:
 - ✓ all local irritants/factors are eliminated (refer to List III. A. VIII)
 - ✓ gingival contours conducive to the preservation of health are attained
 - ✓ the patient cooperates by maintaining good oral hygiene (patient compliance)
- If any of the above factors cannot be achieved, then the prognosis is downgraded.

c) Prognosis for Periodontitis

- General Prognosis. The following factors must be considered:
 - Patient's medical status
 - Age of the patient relative to the amount of disease present
 - Rate of disease progression
 - Individual tooth prognosis
 - Etiologic factors
 - Patient cooperation with recommended home care, therapy
 - Oral habits: smoking, oral habits (clenching/bruxing; TMD)
 - Patient economic factors
 - Knowledge and ability of the dentist

- Specific Prognosis/Individual Tooth prognosis. The following factors must be considered for each tooth:
 - % of bone loss
 - distribution of bone loss around the root (circumferential; one surface)
 - pattern of bone loss (vertical, horizontal)
 - crown:root ratio
 - root form; root proximity
 - presence and severity of furcations; length of root trunk, width of entrance; convergence/divergence of roots
 - probing depth
 - mobility
 - caries; pulpal involvement
 - tooth position and occlusal relationship; anatomic limitations

- strategic value of the tooth to the dentition
- knowledge and skill of the dentist

d) Assigning Prognosis, References

Many factors must be considered when assigning the prognosis of the dentition in general and of specific individual teeth. A number of references have approached the problem of assigning prognoses and the following references are useful:

McGuire, M. K. Prognosis versus actual outcome: A long-term survey of 100 treated periodontal patients under maintenance care. *J. Periodontology* 62:51, 1991

The McGuire reference is discussed in Clinical Periodontology by Carranza, Newman, 1996, 8th edition, page 398.

e) Criteria for Prognoses

GOOD Prognosis: one or more of the following conditions must be met:

- ✓ adequate remaining bone support (periodontal support)
- ✓ adequate control of etiologic factors to ensure that the tooth/teeth would be relatively easy to maintain
- ✓ adequate patient cooperation (compliance)

FAIR Prognosis: one or more of the following conditions must be met:

- ✓ attachment loss to the point that the tooth could not be considered to have a good prognosis and/or
- ✓ Class I furcation involvement but location and depth of furcation would allow maintenance with good patient compliance
- ✓ some tooth mobility
- ✓ adequate maintenance possible
- ✓ acceptable patient compliance

POOR Prognosis: one or more of the following conditions must be met:

- ✓ moderate or severe attachment loss
- ✓ Class I and/or Class II furcations not easily accessible to maintenance
- ✓ Class III furcation
- ✓ Tooth mobility of grade II or greater
- ✓ Poor root form
- ✓ Significant root proximity
- ✓ Difficult to maintain areas and/or
- ✓ Doubtful patient compliance

HOPELESS Prognosis: one or more of the following conditions must be met:

- ✓ Advanced bone loss that is inadequate to maintain the tooth in health, comfort and function
- ✓ Not maintainable
- ✓ Extraction recommended