**Periodontology**

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**Clinical diagnosis and examination**

**Part 2**

**-Second Visit**

**-Examination of Extra-oral Structures**

Clinical examination should begin with an evaluation of the extraoral structures for abnormalities.

**1-Examination of temporomandibular joint**

The temporomandibular joints should be assessed for pain, crepitus, clicking, and range of motion. The muscles of mastication should be palpated for pain and tenderness**.**

**2-Examination of Lymph Nodes**

Because periodontal, periapical, and other oral diseases may result in lymph node changes, the diagnostician should routinely examine and evaluate head and neck lymph nodes. Lymph nodes can become enlarged and/or indurated as a result of an infectious episode, malignant metastases, or residual fibrotic changes. Inflammatory nodes become enlarged, palpable, tender, and fairly immobile. The overlying skin may be red and warm. Patients are often aware of the presence of “swollen glands.” Primary herpetic gingivostomatitis, necrotizing ulcerative gingivitis (NUG), and acute periodontal abscesses may produce lymph node enlargement. After successful therapy, lymph nodes return to normal in a matter of days or a few weeks.

**-Oral Examination:**

**Examination of the Oral Cavity**

The entire oral cavity should be carefully examined. The examination should include the lips, floor of the mouth, tongue, palate, and oropharyngeal region, as well as the quality and quantity of saliva. Although findings may not be related to the periodontal problem, the dentist should detect all pathologic changes present in the mouth.

**Oral Hygiene**

The cleanliness of the oral cavity is appraised in terms of the extent of accumulated food debris, plaque, and tooth surface stains.

Disclosing solution may be used to detect plaque that would otherwise be unnoticed. The amount of plaque detected, however, is not necessarily related to the severity of the disease present. For example, aggressive periodontitis is a destructive type of periodontitis in which plaque is minimal.

**Oral malodor**

**Halitosis**, also termed *fetor ex ore, fetor oris,* and *oral malodor, is* foul or offensive odor emanating from the oral cavity. Mouth odors may be of diagnostic significance, and their origin may be either oral or Extraoral (remote).

Halitosis is caused primarily by volatile sulfur compounds, specifically, hydrogen sulfide and methyl mercaptan, which result from the bacterial putrefaction of proteins containing sulfur amino acids .These products could be involved in the transition from health to gingivitis and then to periodontitis

**1-Local sources** of mouth odors are mainly the tongue and the gingival Sulcus and include retention of food particles on and between the teeth, coated tongue, necrotizing ulcerative gingivitis (NUG), dehydration states, caries, artificial dentures, smoker's breath, and healing surgical or extraction wounds. Chronic periodontitis with pocket formation may also cause unpleasant mouth odor from any accumulated debris and the increased rate of putrefaction of the saliva.

**2-Extraoral sources** of mouth odors include various infections or lesions of the respiratory tract (bronchitis, pneumonia, bronchiectasis).

Alcoholic breath, the acetone odor of diabetes, and the uremic breath that accompanies kidney dysfunction are examples of the last group.

**Examination of the Teeth**

The teeth are examined for caries, poor restorations, developmental defects, anomalies of tooth form, wasting, hypersensitivity, and proximal contact relationships. The stability, position, and number of implants and their relationship to the adjacent natural dentition is also examined.

**Wasting Disease of the Teeth**

Wasting is defined as any gradual loss of tooth substance characterized by the formation of smooth, polished surfaces, without regard to the possible mechanism of this loss. The forms of wasting are erosion, abrasion, and attrition.

***Erosion***,

Also called corrosion, is a sharply defined wedge-shaped depression in the cervical area of the facial tooth surface. The long axis of the eroded area is perpendicular to the vertical axis of the tooth. The surfaces are smooth, hard, and polished. Erosion generally affects a group of teeth. In the early stages, it may be confined to the enamel, but it generally extends to involve the underlying dentin, as well as the cementum.

The etiology of erosion is not known. Decalcification by acidic beverages or citrus fruits, combined with the effect of acid salivary secretion are suggested causes.

**Abrasion**

Refers to the loss of tooth substance induced by mechanical wear other than that of mastication. Abrasion results in saucer-shaped or wedge-shaped indentations with a smooth, shiny surface. Abrasion starts on exposed cementum surfaces rather than on the enamel and extends to involve the dentin of the root. A sharp “ditching” around the cemento-enamel junction appears to be the result of the softer cemental surface, as compared with the much harder enamel surface.

Continued exposure to the abrasive agent, combined with decalcification of the enamel by locally formed acids, may result in loss of enamel, followed by loss of the dentin of the crown.

***Toothbrushing and abrasion***

Toothbrushing with an abrasive dentifrice and the action of clasps are frequently mentioned, but aggressive toothbrushing is the most common cause. Tooth position (facial) is also a major factor in the abrasive loss of the root surface. The degree of tooth wear from toothbrushing depends on the abrasive effect of the dentifrice and the angle of brushing. Horizontal brushing at right angles to the vertical axis of the teeth results in the severest loss of tooth substance. Occasionally, abrasion of the incisal edges occurs as a result of habits such as holding objects (e.g., a bobby pin or tacks) between the teeth.

**Attrition**

is occlusal wear resulting from functional contacts with opposing teeth. Such physical wear patterns may occur on incisal, occlusal, and approximal tooth surfaces. A certain amount of tooth wear is physiologic, but accelerated wear may occur when abnormal anatomic or unusual functional factors are present. Occlusal or incisal surfaces worn by attrition are called facets. When active tooth grinding occurs, the enamel rods are fractured and become highly reflective to light. Thus shiny, smooth, and curvilinear facets are usually the best indicator of ongoing frictional activity.

**Dental Stains**

These are pigmented deposits on the teeth. They should be carefully examined to determine their origin.

* **Intrinsic Stains**
  + Preeruptive causes
    - Disease
      * i. Alkaptonuria
      * ii. Hematological disorders
      * iii. Disease of enamel and dentin
      * iv. Liver diseases.
  + Medications
    - i. Tetracycline stains and other antibiotic use
    - ii. Fluorosis stain.
  + Posteruptive causes of discoloration
    - Pulpal changes
    - Trauma
    - Dentin hypercalcification
    - Dental caries
    - Restorative materials and operative procedures
    - Aging
    - Functional and parafunctional changes
* **Extrinsic Stains**
  + Daily acquired stains
    - Plaque
    - Food and beverages
    - Tobacco use
    - Poor oral hygiene
    - Gingival hemorrhage.
  + Chemicals
    -  Chlorhexidine
    -  Metallic stains

**Hypersensitivity**

Root surfaces exposed by gingival recession may be hypersensitive to thermal changes or tactile stimulation. Patients often direct the clinician to the sensitive areas. These may be located by gentle exploration with a probe or cold air.

**Enamel Loss**

* Occlusal Wear
* Tooth Brush Abrasion
* Dietary Erosion
* Abfraction
* Parafunctiona Habits

**Cemental Loss**

* Gingival Recession
* Periodontal Disease
* Root Planning
* Perodontal Surgery

**Proximal Contact Relations**

Open contacts allow food impaction. The tightness of contacts should be checked by means of clinical observation and with dental floss. Abnormal contact relationships may also initiate occlusal changes such as a shift in the median line between the central incisors, with labial flaring of the maxillary canine, buccal, or lingual displacement of the posterior teeth, and an uneven relationship of the marginal ridges.

**Tooth Mobility**

All teeth have a slight degree of physiologic mobility, which varies for different teeth and at different times of the day. It is greatest on arising in the morning and progressively decreases. The increased mobility in the morning is attributed to slight extrusion of the tooth because of limited occlusal contact during sleep. During the waking hours, mobility is reduced by chewing and swallowing forces, which intrude the teeth in the sockets. These 24-hour variations are less marked in persons with a healthy periodontium than in those with occlusal habits such as bruxism and clenching. Single-rooted teeth have more mobility than multirooted teeth, with incisors having the most.

Mobility is primarily in a horizontal direction, although some axial mobility occurs, to a lesser degree.Abnormal mobility most often occurs faciolingually.

Mobility is graded according to the ease and extent of tooth movement as follows:

• Normal mobility

• Grade I: Slightly more than normal.

• Grade II: Moderately more than normal.

• Grade III: Severe mobility faciolingually and/or mesiodistally, combined with vertical displacement Mobility beyond the physiologic range is termed abnormal or pathologic.

**Increased mobility is caused by one or more of the following factors:**

1. Loss of tooth support (bone loss) can result in mobility. The amount of mobility depends on the severity and distribution of bone loss at individual root surfaces

2. The length and shape of the roots, and the root size compared with that of the crown. A tooth with short, tapered roots is more likely to loosen than one with normal-size or bulbous roots with the same amount of bone loss.

2. Trauma from occlusion (i.e., injury produced by excessive occlusal forces or incurred because of abnormal occlusal habits such as bruxism and clenching) is a common cause of tooth mobility. Mobility is also increased by hypofunction. Mobility produced by trauma from occlusion occurs initially as a result of resorption of the cortical layer of bone, leading to reduced fiber support, and later as an adaptation phenomenon resulting in a widened periodontal space.

3. Extension of inflammation from the gingiva or from the periapex into the periodontal ligament results in changes that increase mobility. The spread of inflammation from an acute periapical abscess may increase tooth mobility in the absence of periodontal disease.

4. Periodontal surgery temporarily increases tooth mobility immediately after the intervention and for a short period.

5. Tooth mobility is increased in pregnancy and is sometimes associated with the menstrual cycle or the use of hormonal contraceptives. This is unrelated to periodontal disease and occurs presumably because of physicochemical changes in the periodontal tissues.

6. Pathologic processes of the jaws that destroy the alveolar bone and/or the roots of the teeth can also result in mobility. Osteomyelitis and tumors of the jaws belong in this category.

**Pathologic Migration of the Teeth**

Alterations in tooth position should be carefully noted, particularly with a view toward identifying abnormal forces, a tongue-thrusting habit, or other habits that may be contributing factors . Premature tooth contacts in the posterior region that deflect the mandible anteriorly contribute to destruction of the periodontium of the maxillary anterior teeth and to pathologic migration .

The loss of posterior teeth can lead to the facial “flaring” of the maxillary anterior dentition. This is due to the increased trauma that the mandibular anterior dentition places against the palatal surface of the maxillary anterior dentition.

Pathologic migration of anterior teeth in young persons may be a sign of localized aggressive (juvenile) periodontitis.