

# CLASSIFICATIONS IN PERIODONTICS

- An Update -



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**Suchetha A**



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# CLASSIFICATIONS IN PERIODONTICS

## An Update

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Bangalore, India



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# CLASSIFICATIONS IN PERIODONTICS: An Update

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## Foreword

It is my privilege to write the foreword for this book "Classifications in Periodontics – An update".

'Classification of various diseases and conditions' is a prime topic in the field of health sciences, which helps the students and professionals in their diagnosis and treatment planning. In Periodontology, there are numerous classifications available, which becomes taxing for the students as well as the clinicians. So, the editorial team with their skills and expertise have consolidated a spectrum of classifications from various authentic sources relevant to the subject in the form of a handbook. The hallmark of this book is the 'extensive categorisation' of each and every aspect of periodontology.

I am confident that this well-illustrated book with its easy language and concise information will make an interesting and informative reading for the students as well as the clinicians.

Hearty congratulations to the editorial team for all the efforts and hard work that they have undertaken. I wish them good luck for the book and in all their future endeavours.

Prof. Dr. Prashanth (MDS)  
Department of Orthodontics  
DAPMRV Dental College  
Bangalore

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## ABSCESS

- I. Depending on the location (Carranza 1980, Carranza 1990)
  - a) Gingival abscess
  - b) Periodontal abscess
- II. Depending on the duration (Carranza 1990)
  - a) Acute
  - b) Chronic
- III. Depending on the number
  - a) Single
  - b) Multiple
- IV. According to the location
  - a) Gingival abscess
  - b) Periodontal abscess
  - c) Pericoronal abscess
- V. According to the depth (Carranza 1990)
  - a) Abscess on the crown
  - b) Abscess on the root
- VI. Depending on the type (Carranza 1990, pg 496)
  - a) Periodontitis
  - b) Non periodontitis

## **ABSCESS**

- I. Depending on the location of the abscess (Girlette and Ban Hous 1980, Carranza 1990)
  - a) Gingival abscess
  - b) Periodontal abscess
- II. Depending on the course of the lesion (Galego-Fsul et al 1995 & Carranza 1990)
  - a) Acute
  - b) Chronic
- III. Depending on the number (Topoll et al.1990)
  - a) Single
  - b) Multiple
- IV. According to the periodontal tissue affected (Meng et al 1999)
  - a) Gingival abscess
  - b) Periodontal abscess
  - c) Pericoronal abscess
- V. According to (Carranza 11<sup>th</sup> edition)
  - a) Abscess on supporting periodontal tissue on the lateral aspect of the root
  - b) Abscess on soft tissue wall of a deep periodontal pocket
- VI. Depending on the cause of the acute infection (Lindhe 5<sup>th</sup> edition, pg 496)
  - a) Periodontitis related abscess
  - b) Non periodontitis related abscess

**ANTIPLAQUE AGENTS**

**I. Based on Mechanism of Action: (Kornman 1986)**

**a. First generation**

The agents that do not exhibit any significant substantivity (only minutes) were categorized as first-generation.

Antimicrobial agents - certain antibiotics, quaternary ammonium compounds (cetylpyridinium chloride), essential oil, phenolic compounds, fluorides including monofluorophosphate and sodium fluoride, oxidizing agents, plant alkaloids and iodines including povidone iodine. (Kornman-1986)

**b. Second generation**

Second-generation antimicrobial agents are characterized by high substantivity, that is, retention of 25-30% after each 1-minute of mouth rinse.

Such compounds remain active in situ for hours.

E.g. Bisbiguanides (such as chlorhexidine), amine fluoride and stannous fluoride mouth rinse and triclosan, when associated with a copolymer of polyvinyl methyl ether and maleic acid copolymer.

**c. Third generation**

Substances with mild antibacterial effect but that interfere with bacterial adhesion are referred to as third-generation antimicrobial agents.

E.g. The aminoalcohols (octapinol, delmopinol). These substances are currently being tested, but from a clinical point of view, second-generation antimicrobial agents are still the agents of choice for the time being.

**d. Fourth generation**

The mouth flora will be individually characterized.

In the laboratory a cocktail of non adherent bacteria would be prepared and this mixture given to a particular individual at more or less frequent intervals, until such time as he or she acquires a personal flora, mainly comprising handicapped bacteria.

Class:

But some resilient th

However remains as being

**II. According**

**a. Group**

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acid

of

ca

**b. C**

?

c

**III**

But some normal bacteria remain; they will always be more resilient than the rest and thus grow back quickly.

However, whether this approach is feasible or even desirable remains to be seen and the strategy must be described at present as being rather futuristic.

## II. According to Elley B M in 1999

**a. Group A agents:** Described as **antiplaque** (by definition, chemicals that inhibit plaque formation to such an extent that they prevent the development of gingivitis). It includes chlorhexidine, acidified sodium chlorate, salifluor and delmopinol. The efficacy of these chemicals is reflected in that in mouth wash form they can be used instead of conventional mechanical plaque removal.

**b. Group B agents:** Include cetylpyridinium chloride, essential oil and triclosan rinses.

These rinses should be used as adjuncts to mechanical cleaning, such as tooth brushing, and are termed **plaque inhibitory agents**.

**c. Group C agents** (low to moderate activity): are chemicals rinse with little or no effect on plaque accumulation and would be expected to have a largely cosmetic role, such as breath freshening. Rinses in this group include products containing sanguinarine, oxygenating agents and rinses containing the saturated pyrimidine, hexetidine.

## III. Individual Agents

1) Cationic organic molecules

Bisbiguanides: Chlorhexidine

Quaternary ammonium chloride: Cetylpyridinium chloride

Plant alkaloids: Sanguinarine

2) Non charged phenolic agents

Essential oils

Triclosan

3) Natural products

Acacia arabica

Neem

---

- 4) Anionic surfactants
  - Sodium lauryl sulphate
  - Sodium dodecylsulphate
- 5) Metal salts
  - Zinc chloride
  - Tin
  - Copper
- 6) Fluorides
- 7) Oxidative enzymes
  - Hydrogen peroxide
  - Urea peroxide
- 8) Surface modifying agents
  - Delmopinol hydrochloride
- 9) Antibiotics
- 10) Agents softening mineralization of calculus
  - Acids
  - Alkalis
  - Chelating agents
  - Enzymes
  - Urea
  - Miscellaneous
- 11) Agents inhibiting mineralization
  - Metals
  - Bisphosphonates
  - Vitamin C
  - Pyrophosphate

## ANTI-CALCULUS AGENTS

### I. First Generation:

#### a. Act by dissolution

1. Chelating agents - Sodium hexametaphosphate
2. Acids - Aromatic sulphuric acid  
20% trichloroacetic acid
3. Alkalies
4. Spring salts
5. Sodium ricinolate

#### b. Act by altering calculus attachment

1. Silicones
2. Ion exchange resins

#### c. Act by plaque inhibition

1. Antibiotics - Nidamycin
2. Antiseptics - Chloramines

#### d. Act by matrix disruption

1. Enzymes - Mucinase
2. Trypsin, chymotrypsin
3. Carboxypeptidase, lipase, amylase
4. Urea (30%)

### II. Second Generation:

#### a. Inhibits crystal growth

1. Vitamin C
  2. Pyrophosphates
  3. Pyrophosphates and NaF
  4. Zinc salts
-

5. Bisphosphonates
6. Polymers and copolymers
- b. *Calculus softening agents*
  1. EDTA – Calcium chelating agent

## ANTIBIOTICS

### I. Based on Chemical Structure: (Tripathi)

1. **Sulfonamides and related drugs:**  
Sulfa diazine and others, Sulfones dapsone (DDS)  
Para amino salicylic acid (PAS)
2. **Diaminopyrimidines:**  
Trimethoprim, Pyrimethamine.
3.  **$\beta$  lactam antibiotics:**  
Penicillins, Cephalosporins, monobactams, carbapenems.
4. **Tetracyclines:**  
Oxytetracycline, doxycycline etc.
5. **Nitro benzene derivative:**  
Chloramphenicol
6. **Aminoglycosides:**  
Streptomycin, Gentamycin, neomycin
7. **Macrolide antibiotics:**  
Erythromycin, Azithromycin, oxythromycin
8. **Polypeptide antibiotics:**  
Polymyxin B, colistin, bacitracin, tyrothricin.
9. **Nitrofurantoin derivatives:**  
Nitrofurantoin, furazolidone
10. **Nitroimidazoles:**  
Metronidazole, tinidazole

11. **Quinolones:**

Nalidixic acid, norfloxacin, ciprofloxacin.

12. **Nicotinic acid derivative:**

Isoniazid, pyrazinamide, ethionamide

13. **Polyene antibiotics:**

Nystatin, amphotericin B

14. **Imidazole derivative:**

Miconazole, ketoconazole, clotrimazole

15. **Others:**

Rifampin, clindamycin, spectinomycin, vancomycin, lincomycin, sodium fusidate, cycloserine, viomycin, ethambutol, thiacetazone, clofazimine, griseofulvin

**II. Mechanism of action:** Ref.: (Textbook of Pharmacology for Dental and Allied Health Sciences – Padmaja Uday Kumar 2nd Edition)

- a. Inhibit cell wall synthesis : Penicillins, cephalosporin, cycloserine, vancomycin, bacitracin.
- b. Causes leakage from cell membrane :
  1. Polypeptides – Polymyxins, colistin, citracin
  2. Polyenes – Amphotenicin B, nystatin, hamycin
- c. Inhibit protein synthesis: Tetracyclines, cloramphenicol, erythromycin, clindamycin, linezolid
- d. Cause misreading of m-RNA code (bind to 30S ribosomes): aminoglycosides, streptomycin
- e. Interfere with DNA function: Rifampin, norfloxacin, metronidazole.
- f. Interfere with intermediary metabolism: sulfonamides, sulfones, para amino salicylic acid, trimethoprim, pyrimethamine, ethambutol

**III. Types of Organisms against Which Primarily active:** (Padmaja Uday Kumar 2nd Edition)

1. Antibacterial: Penicillin, amino glycosides, erythromycin.
  2. Antifungal: Griseofulvin, amphotericin B, ketoconazole
  3. Antiviral: Idoxuridine, acyclovir, amantadine, zidovudine.
-



4. Antiprotozoal: Chloroquine, pyrimethamine and metronidazole, diloxanide etc.
5. Anthelmintic: Mebendazole, piperazine, pyrantel – niclosamide etc.

#### IV. Type of Action: (Padmaja Uday Kumar 2nd Edition)

1. Primarily bacteriostatic: Sulfonamides, tetracycline, chloramphenicol, erythromycin, ethambutol
2. Primarily bactericidal: Penicillin, amino-glycosides, polypeptides, rifampicin, cotrimoxazole
3. Both: cephalosporin, vancomycin, nalidixic acid.

### ANTIOXIDANTS

#### I. Classification of Antioxidants:

##### a. Endogenous antioxidants (cellular antioxidant - made in the body)

- Glutathione
- Coq10
- Alpha lipoic acid

##### b. Antioxidants phytochemicals - photosynthetic nutrients/ plant pigments.

###### 1. Carotenoids - best known as precursor of vit. A

- Lycopene, lutein, zeaxanthin
- Fat-soluble
- Found in colorful foods

###### 2. Bioflavonoids or flavonoids - another type of plant pigment, found in fruits and vegetables also in red wine, beer, chocolate, coffee, algae and soya.

Major classes are:

- Flavonones
- Flavones
- Flavonols

#### Classif

- Isoflav
- clover
- Catecl
- Chalc
- Anthc

#### c. Herbal anti

and garlic

#### d. Antioxidan

Vitamin A

Vitamin C

Vitamin E

#### Antioxida

Selenium

Zinc

#### Antioxida

Melatonin

### II. Antioxidan

According to  
of Oral Med

#### A) Enzym

seleniu  
transfer

#### B) Non-en

##### 1) Nu

glu

tea

##### 2) No

ca

### III. J Indian S

Reactive ox

#### A) Based

- Isoflavones - soya, dong quai, black cohosh, icorice and red clover
  - Catechins - green tea
  - Chalcones
  - Anthocyanidins- grape seed and pycnogenol
- c. **Herbal antioxidants** - such as milk thistle, ginkgo biloba, curcumin and garlic
- d. **Antioxidant vitamins**
- Vitamin A
  - Vitamin C
  - Vitamin E
- Antioxidant minerals**
- Selenium
  - Zinc
- Antioxidant hormone**
- Melatonin

## II. Antioxidants

According to Shetti A, Keluskar V, Aggarwal A - J Indian Academy of Oral Medicine and Radiology. 2009 Nov; 21 (1): 1 - 6

- A) **Enzymatic:** Superoxide dismutase, glutathione peroxidase, selenium, catalase, glutathione reductase, glutathione transferase
- B) **Non-enzymatic:** They are further subdivided into two.
- 1) **Nutrient:** Alpha tocopherol,  $\beta$ -carotene ascorbate, glutathione, selenium, proanthocyanidin, lycopene, green tea
  - 2) **Non nutrient:** Ceruloplasmin, transferrin, uric acid, peptides camosine anserine

III. J Indian Soc Periodontol. 2013 July-August; 17(4): 411-416  
Reactive oxygen species in periodontitis

- A) **Based on mode of action:**
-

1) **Preventative antioxidants:**

**Antioxidant enzymes:** Superoxide dismutase enzymes (1, 2, and 3), catalase, glutathione peroxidase, DNA repair enzymes, others.

**Metal ion sequestrators:** Albumin, lactoferrin, transferrin, haptoglobin, ceruloplasmin, hemopexin, carotenoids, superoxide dismutase, catalase, glutathione peroxidase, glutathione reductase, uric acid, polyphenolic flavonoids.

2) **Scavenging (chain breaking) antioxidants:** Ascorbate, carotenoids, uric acid, alpha tocopherols, polyphenols, bilirubin, albumin, ubiquinone, reduced glutathione and other thiols.

**B) Based on location:**

1) **Intracellular:** Superoxide dismutase (1, 2 and 3), catalase, glutathione peroxidase, DNA repair enzymes, others, reduced glutathione, ubiquinone(reduced form)

2) **Extracellular:** Superoxide dismutase enzyme 2, selenium glutathione peroxidase, reduced glutathione, lactoferrin, transferrin, haptoglobin, ceruloplasmin, albumin, ascorbate, carotenoids, and uric acid.

3) **Membrane associated:** Alpha tocopherol.

## ANTIMICROBIAL PEPTIDES

### I. According to Boman, H.G. 1995

1. Anionic peptides: Dermicidin
2. Cationic peptides: Cecropin, LL37
3. Cationic peptides with specific amino acids: PR 39, prophenin, indolicidin
4. Peptides that form disulphide bridges: Brevinins, tachyplecin, defensins, NK-lysin, drosomycin
5. Fragmented peptides: Lactoferricin

### II. According to Wang, 2010: Based on the types of secondary structures

1. Alpha ( $\alpha$ ):  $\alpha$  strands e.g., magainins and LL-37
2. Beta ( $\beta$ ):  $\beta$  strands

3. Alpha beta ( $\alpha\beta$ ): Comprises both  $\alpha$ -helical and  $\beta$ -strands in the 3D structure e.g.  $\beta$ -defensins
4. Non-alpha beta (non- $\alpha\beta$ ): contains neither  $\alpha$ -helical nor  $\beta$ -strands e.g. indolicidin.

### AIDS CDC SURVEILLANCE CLASSIFICATION 1993

- I. **Category A** - Includes patients with acute symptoms or asymptomatic diseases, along with individuals with persistent generalized lymphadenopathy, with or without malaise, fatigue, or low-grade fever.
- II. **Category B**-Patients with symptomatic conditions such as oropharyngeal or vulvovaginal candidiasis, herpes zoster, oral hairy leukoplakia, idiopathic thrombocytopenia, or constitutional symptoms like fever, diarrhea, and weight loss.
- III. **Category C**- Patients with outright AIDS, as manifested by life-threatening conditions or identified through CD4+ T lymphocyte levels of less than 200 cells/mm.<sup>3</sup>

### CLASSIFICATION OF BONE GRAFTS AND THEIR SUBSTITUTES

- I. **According to Degree of Inductive Potential.** (Carranza 11<sup>th</sup>edi pg-872)

#### 1. Osteoinductive Graft

##### i. Autogenous bone graft

- a. Extra oral
  - Fresh
  - Frozen or preserved.
- b. Intra oral
  - Osseous coagulum
  - Tuberosity
  - Bone blend
  - Bone swaging

**ii. Allograft:**

- a. Demineralized freeze-dried bone allograft (DFDBA)
- b. Freeze – dried bone allografts (FDBA)

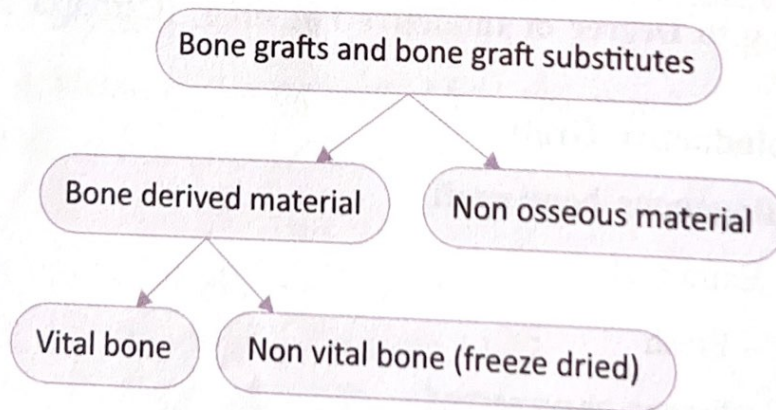
**2. Osteoconductive Graft:****i. Allograft**

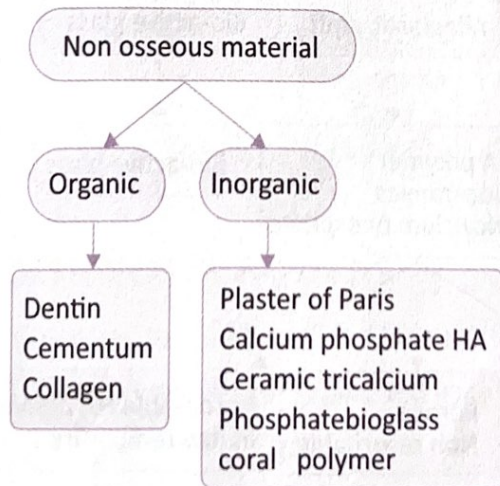
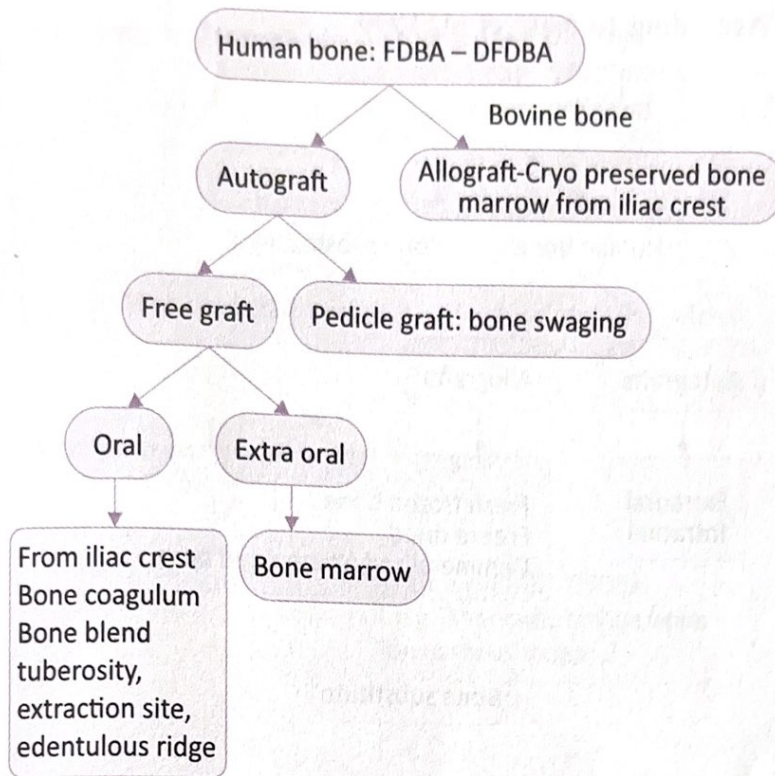
- FDBA
- DFDBA

**ii. Alloplast – Porous hydroxyapatite.****3. Osteoneutral Graft:** Grafts that are totally inert and serve only as space fillers.**i. Alloplastic materials**

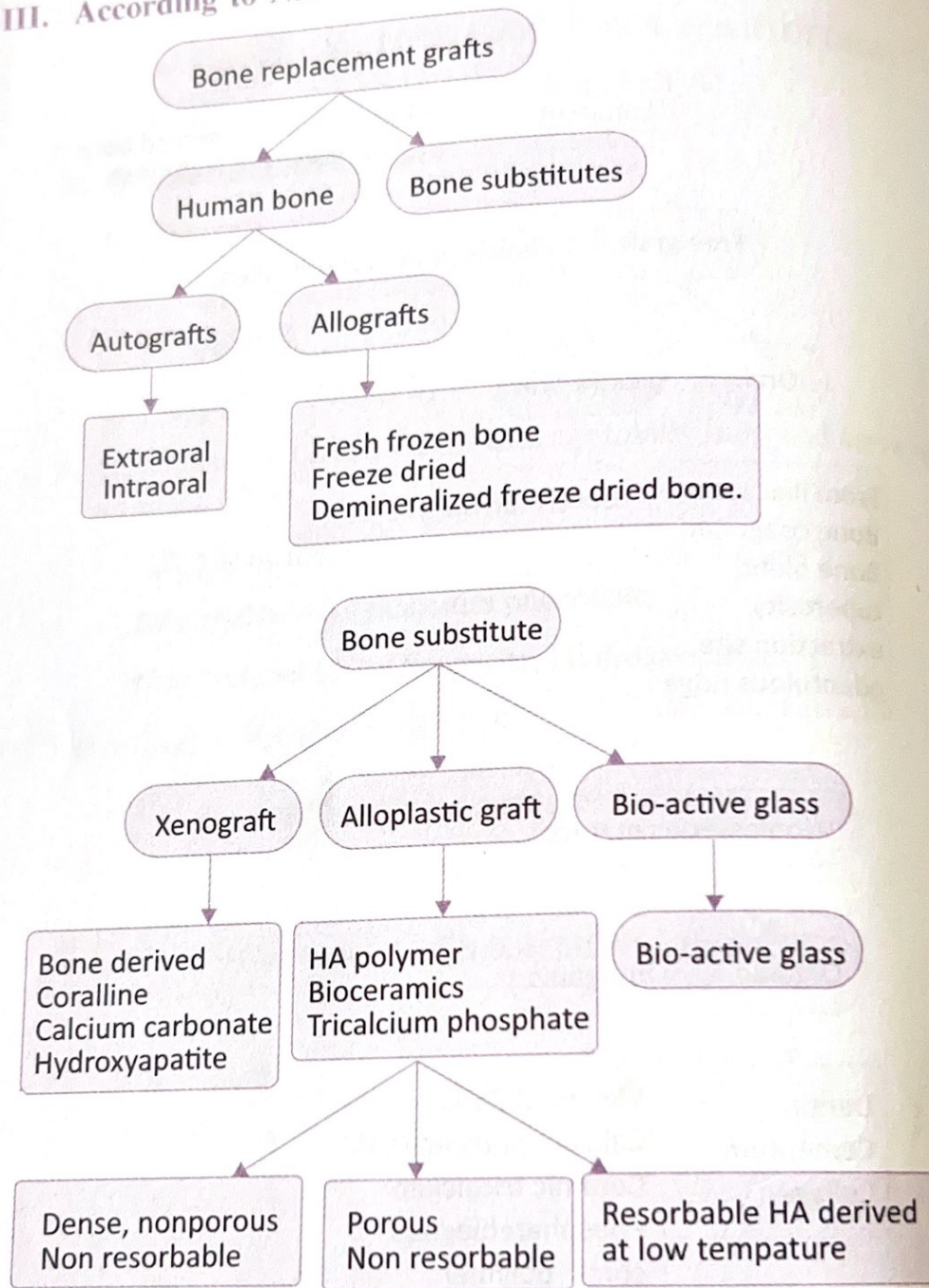
Resorbable –  $\beta$  Tricalcium phosphate

Non resorbable – Durapatite, Hydroxyapatite

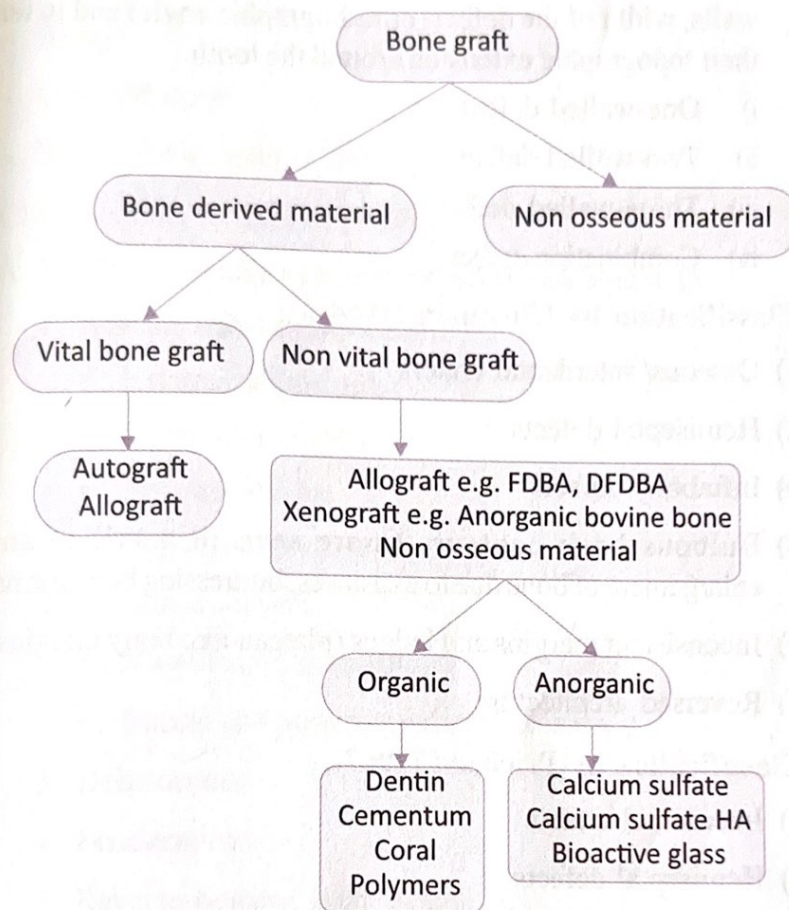
**II. According to Conge et al 1978**



III. According to Nasr et al. 1999



## IV. According to Rosenberg and Rose et al. 1998



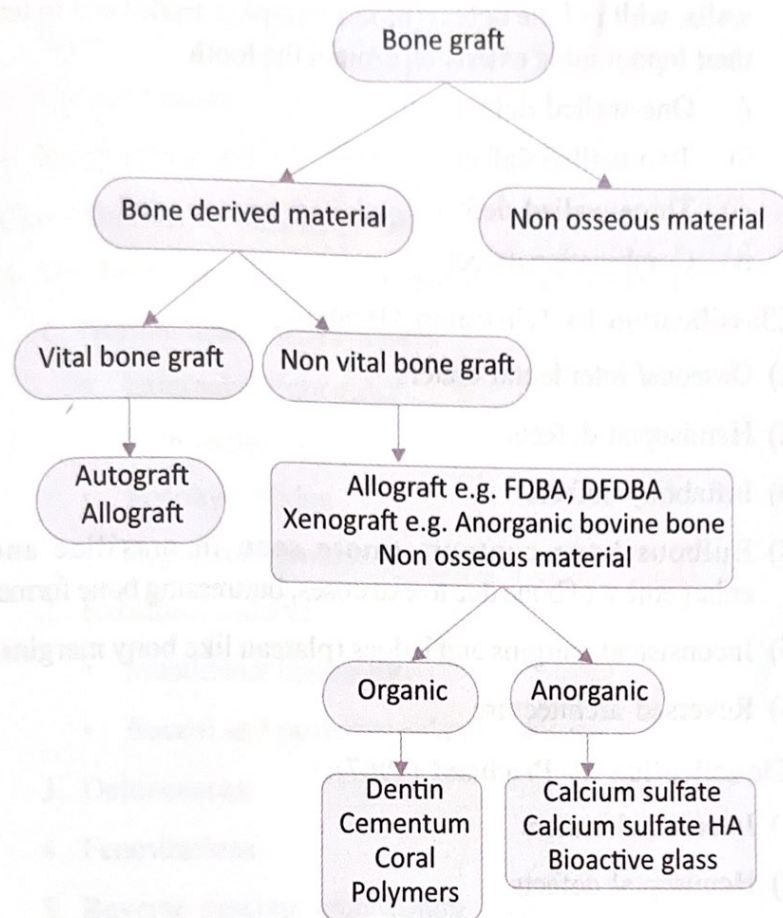
## BONE DESTRUCTION PATTERNS

## I. According to Goldman &amp; Cohen (1958):

- a) Suprabony defects: Where the base of pocket is located coronal to the alveolar crest.
- b) Infrabony defects: Apical location of the base of the pocket with respect to the residual alveolar crest
  - 1) Intrabony defects: Bony defects whose infrabony component affects primarily one tooth.
  - 2) Craters: The defect affects two adjacent root surfaces to a similar extent.



## IV. According to Rosenberg and Rose et al. 1998



## BONE DESTRUCTION PATTERNS

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  - 2) Craters: The defect affects two adjacent root surfaces to a similar extent.

c) Intra-bony defects:

Classified according to their morphology in terms of residual bony walls, width of the defect (or radiographic angle) and in terms of their topographic extension around the tooth.

- i) One-walled defect
- ii) Two-walled defect
- iii) Three-walled defect
- iv) Combination defect

**II. Classification by Glickman (1964):**

- 1) Osseous/ interdental craters
- 2) Hemiseptal defects
- 3) Intra-bony defects
- 4) Bulbous bone contours (more seen in maxillae and are enlargement of bone due to exostoses, buttressing bone formation).
- 5) Inconsistent margins and ledges (plateau like bony margins)
- 6) Reversed architecture

**III. Classification by Prichard (1967):**

- 1) Interdental craters
- 2) Hemiseptal defects
- 3) Intra-bony defects
- 4) Inconsistent margins
- 5) Furcation involvement
- 6) Anatomic aberrations of the alveolar process
  - Thick marginal ledges
  - Exostosis and tori
  - Dehiscence and fenestrations

**IV. Classification by Karn et al (1983):**

- Horizontal bone loss
- Crater

- Trench
- Moat
- Ramp
- Cratered ramp
- Ramp into a crater or trench

#### V. Classification by Grant (1988):

##### A. Vestibular, lingual or palatal defects associated with:

###### 1. Normal anatomic structures

- External oblique ridge
- Retromolar triangle
- Mylohyoid ridge
- Zygomatic process

###### 2. Exostosis and tori

- Mandibular lingual tori
- Buccal and posterior palatal exostosis

###### 3. Dehiscences

###### 4. Fenestrations

###### 5. Reverse osseous architecture

##### B. Vertical defects:

###### 1. Three walls

###### 2. Two walls

###### 3. One wall

###### 4. Combination with a different number of walls at the various levels of the defect.

##### C. Furcation defects:

###### 1. Class I or incipient

###### 2. Class II or partial

###### 3. Class III or through and through

VI. Classification by Panos and Tonetti (2000):

A. Suprabony defects

B. Infrabony defects - Intrabony defects -

- 1 wall
- 2 walls
- 3 walls
- Combinations
- Craters

C. Interradicular defects

1) Horizontal classification

- Class I
- Class II
- Class III

2) Vertical classification

- Subclass A
- Subclass B
- Subclass C

BIOMARKERS CLASSIFICATION

I. Based on the source:

GCF

Saliva

Serum

II. GCF biomarkers (Taba M - 2005):

- a) Microbial plaque: Endotoxins (lipopolysachrides), enzymes, metabolic end products, DNA probes, cultures of putative periodontal pathogens.
- b) Host derived: IL- $\beta$ , aspartate, aminotransferase, transferrin, matrix proteins, lactoferrin, lysozyme etc...
- c) Connective tissue breakdown products: Collagen-telopeptides, osteocalcin, proteoglycans, breakdown products, fibronectin fragments.

- d) Inflammatory mediators: Complement, cytokines, interleukins, tumor necrosis factor- $\alpha$ , interferon- $\alpha$ , antibacterial antibodies IgG, IgM, IgA, substance P, prostaglandin E2, acute-phase proteins, transferrin, C-reactive protein.

### III. Salivary biomarkers (Miller C S - 2006)

- 1) Enzymes: Alkaline phosphatase, amino peptidase, trypsin,  $\alpha$  glucosidase,  $\alpha$  galactosidase,  $\alpha$  glucuronidase, gelatinase, esterase, collagenase, kininase
- 2) Immunoglobulins: Ig A, Ig G, Ig M, sIg A
- 3) Proteins: Cystatin, fibronectin, lactoferrin, vascular endothelial growth factors, platelet activating factors, epidermal growth factors
- 4) Phenotypic markers: Epithelial keratin
- 5) Host cells: Leukocytes (PMN's)
- 6) Ions: Calcium
- 7) Hormones: Cortisol
- 8) Bacteria: A.a, P. gingivalis, P. intermedia, P. micros, C. rectus, T. denticola, B. forsythus, P. micros, mycoplasma
- 9) Volatile compounds: Hydrogen sulphide, methyl mercaptan, picolines, pyridines

### IV. Specific salivary biomarkers for periodontal disease (Lei Zhang - 2009)

Table 1:

Proteomic biomarkers	Genetic markers	Microbial markers	Other markers
Alpha glucosidase	Cathepsin C	Aggregatibacteractinomycesemcomitans	Calcium
Acid phosphatase	Gene mutation	Campylobacter rectus	Cortisol
Alkaline phosphatase	Collagen gene Mutation	Mycoplasma Porphyromonas gingivalis	Hydrogen sulphide
Amino peptidase		Prevotella intermedia	Methyl mercaptans
Aspartate aminotransferase	IL-1 Polymorphism	Peptostreptococcus micros	Picolines
Beta glucosidase	IL-10	Prevotella nigricans	Polymorpho nuclear leukocytes
Beta galactosidase	Polymorphism	Treponema denticola	Pyridine
Beta glucuronidase	Tumour	Tanerella forsythus	
Calprotectin	Necrosis factor	Treponema socranskii	
Caprylate esterase lipase	Polymorphism		
Cathepsin B			
CD 14			
Cystatins			
Esterase			
Fibronectin			
Gelatinase			
IgA			
IgG			
IgM			
Kallikrein			
Kininase			
Lactoferrin			
Lactotransferrin			
Lactate			
Dehydrogenase			
Lysozyme			
MMP-13			
MMP-8			
MMP-9			
Myeloperoxidase			
Osteocalcin			
Osteonectin			
Osteopontin			
Platelet activating factor			
PDGF			
Trypsin			
VEGF			
IgA			

## CLASSIFICATION OF JAW BONES: (Carranza)

- I. 1. Basal bone
2. Alveolar process -
  - a. Cortical bone (external plate)
  - b. Compact bone (alveolar bone proper)
  - c. Cancellous bone.

## BONE QUALITY SCHEMES RELATED TO IMPLANT DENTISTRY

### I. Linkow's Classification of Bone Density (1970)

1. Class 1 bone structure: Ideal bone type consists of evenly spaced trabeculae with small cancellated spaces.
2. Class 2 bone structure: Slightly larger cancellated spaces with less uniformity of the osseous pattern
3. Class 3 bone structure: Large marrow filled spaces exist between bone trabeculae.

### II. Lekholm and Zarb (1985)

1. Quality 1: Composed of homogeneous compact bone
2. Quality 2: Thick layer of compact bone surrounding a core of dense trabecular bone
3. Quality 3: Thin layer of cortical bone surrounding dense trabecular bone of favorable strength.

### III. Misch Bone Density Classification (1988)

1. D1 - Dense cortical bone
  2. D2 - Thick dense to porous cortical bone on crest and coarse trabecular bone within
  3. D3 - Thin porous cortical bone on crest and fine trabecular bone within
  4. D4 - Fine trabecular bone
  5. D5 - Immature, non-mineralized bone
-

**BONE MORPHOGENIC PROTEINS****Table 2:** According to Even J. Journal of American Academy of Orthopaedic Surgeons 2012.

S. No	Name of the BMP	Characteristic
1.	BMP 1	Not part of TGF $\beta$ family
2.	BMP 2	Osteoinductive, osteoblast differentiation, apoptosis
3.	BMP 3(osteogenin)	Most abundant BMP in bone, inhibits osteogenesis
4.	BMP 4	Osteoinductive, lung and eye development
5.	BMP 5	Chondrogenesis
6.	BMP 6	Osteoblast differentiation, chondrogenesis
7.	BMP 7 (osteogenic Protein 1)	Osteoinductive, development of kidney and eye
8.	BMP 8 (osteogenic Protein 2)	Osteoinductive
9.	BMP 9	Nervous system, hepatic reticuloendothelial system
10.	BMP 10	Cardiac development
11.	BMP 11 (growth/ differentiation factor 8)	Neuronal tissues
12.	BMP 12 (growth/ differentiation factor 7)	Tendon iliac tissue formation
13.	BMP 13 (growth/ differentiation factor 6)	Tendon and ligament like tissue formation
14.	BMP 14 (growth/ differentiation factor 5)	Enhances tendon healing and bone formation.
15.	BMP 15	Follicle stimulating hormone activity



**BISPHOSPHONATES** According to Wheeler (Textbook of Orthopaedics)

**1. Based on the generation:**

- i) First generation: with alkyl side chains  
E.g., Etidronate
- ii) Second-generation: amino-bisphosphonates with an amino-terminal group  
E.g., Alendronate and pamidronate
- iii) Third-generation: with cyclic side chains  
E.g., Risedronate.

**2. Based on route of administration:**

- i. Orally administered bisphosphonates:  
E.g. Risedronate, ibandronate, alendronate, tiludronate, etidronate
- ii. Intravenously administered bisphosphonates  
E.g. Pamidronate, zoledronic acid, clodronate

**3. Based on presence of nitrogen:**

- i. Non-N-containing bisphosphonates:  
E.g: Etidronate, clodronate, tiludronate
- ii. N-containing bisphosphonates:  
E.g: Pamidronate, neridronate, olpadronate, alendronate, ibandronate, risedronate, zoledronate

**CLASSIFICATION OF BLEEDING DISORDERS**

**According to Burket's**

**I) Vessel Wall Disorders**

Scurvy

Cushing's syndrome

Ehlers - Danlos syndrome

Rendu-Osler Weber syndrome

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**II) Platelet Disorders****a) Congenital**

- i) Thrombocytopenic—quantitative platelet deficiency
  - May-Hegglin anomaly
  - Wiskott-Aldrich syndrome
  - Neonatal alloimmune thrombocytopenia
- ii) Nonthrombocytopenic—qualitative or functional platelet defect
  - Glanzmann's thrombasthenia
  - Platelet-type von Willebrand's disease
  - Bernard-Soulier syndrome

**b) Acquired**

- i) Thrombocytopenic—quantitative platelet deficiency
  - Autoimmune or idiopathic thrombocytopenia purpura
  - Thrombotic thrombocytopenia purpura
  - Cytotoxic chemotherapy
  - Drug-induced (e.g., quinine, quinidine, gold salts, trimethoprim/sulfamethoxazole, rifampicin)
  - Leukemia
  - Aplastic anemia
  - Myelodysplasia
  - Systemic lupus erythematosus
  - Associated with infection: HIV, mononucleosis, malaria
  - Disseminated intravascular coagulation
- ii) Nonthrombocytopenic—qualitative or functional platelet defect
  - Drug-induced (e.g., by aspirin, NSAIDs, penicillin, cephalosporins)
  - Uremia
  - Alcohol dependency
  - Liver disease
  - Myeloma, myeloproliferative disorders, macroglobulinemia
  - Acquired platelet-type von Willebrand's disease

## BIOMATERIALS OF IMPLANTS

According to Carranza

a) **Metallic:**

Titanium

Titanium alloys

Aluminium

Vanadium

Co Cr alloys

Stainless steel

Precious metals such as gold and platinum.

b) **Ceramic:**

Aluminium oxide

Ceramics

Carbon

Carbon-silicon compounds.

c) **Polymers and composite:**

Cross linked polymers such as polymethyl methacrylate, silicone rubber and polyethylene.

## CERVICAL ENAMEL PROJECTIONS

According to Master & Hoskins (1964)

Grade I: Short CEP from CEJ

Grade II: Longer CEP and approaches the furcation area

Grade III: CEP that extends directly into the furcation.

## CYTOKINES

1) **According to Carranza**

a) **Innate immunity cytokines**

TNF- $\alpha$ , IL-1, IL-12, IF- $\alpha$  IF- $\beta$ , IL-6, IL-8, IL-18, IL-23, IL-27

- b) Adaptive immunity cytokines  
IL-2, IL-4, IL-5, IF- $\gamma$ , IL-6, IL-11, IL-13, IL-17
- c) Hematopoietic cytokines  
CSF, IL-7, IL-3 (multi CSF) 3 (multi CSF), IL-1, IL-5, IL-6,  
Erythropoietin
- d) Immunosuppressive cytokines  
TGF- $\beta$ , IL-10, IL-4, IL-13

**II) Jan Lindhe's Classification:** According to Jan Lindhe

- A) Pro inflammatory cytokines  
E.g. IL -1, 6, TNF  $\alpha$  – stimulates bone resorption
- B) Chemotactic cytokines  
E.g. IL-8, 16
- C) Lymphocyte signaling cytokines
  - I) Released by Th 1- IL 2, IFN
  - II) Released by Th 2- IL4, 5, 10, 13

**III) According to Charles A. Dinarello.** Historical insights into cytokines, Eur. J. Immunol. 2007; 37: S34–45

**Categories of cytokines**

- a) Mediators of natural immunity
  - i) TNF- $\alpha$
  - ii) IL 1
  - iii) IL 10
  - iv) IL 12
  - v) Type I interferons
  - vi) INF- $\gamma$
  - vii) Chemokines
- b) Mediators of adaptive immunity
  - i) IL 2
  - ii) IL 4
  - iii) IL 5
  - iv) TGF- $\beta$

- c) Stimulators for hematopoiesis
  - i) GM - CSF
  - ii) M - CSF

## CLASSIFICATION OF CLOTTING DISORDERS

### According to Burket's

Coagulation disorders may be congenital or acquired secondary to drugs or disease.

#### a) **Congenital:**

- Hemophilia A
- Hemophilia B
- Factor XI deficiency
- Factor XII deficiency
- Factor V deficiency
- Factor X deficiency
- Factor XIII & I deficiencies
- Von - Willebrand's disease.

#### b) **Acquired:**

- i) Anti-coagulant related coagulopathies:
  - Heparin
  - Coumarin
- ii) Disease related coagulopathies:
  - Liver disease
  - Vitamin K deficiency
  - Disseminated intravascular coagulation (DIC)

**CEMENTUM****I) Berkovitz et al 2002****a) Based on the location on teeth**

- i) Coronal cementum
- ii) Radicular cementum

**b) Based on cellularity**

- i) Acellular cementum (primary)
- ii) Cellular cementum (secondary)

**c) Based on presence or absence of collagen fibrils in organic matrix**

- i) Fibrillar cementum
- ii) Afibrillar cementum

**II) Classification system devised by Owens in 1970 and summarized recently by Schroders and Page**

- a) Acellular afibrillar cementum. (AAC)
- b) Acellular extrinsic fiber cementum. (AEFC)
- c) Cellular mixed stratified cementum. (CMSC)
- d) Cellular intrinsic fiber cementum. (CIFC)
- e) Intermediate cementum.

**MODELS OF DISEASE PROGRESSION**

1. *Continuous model* (Socransky et al 1984):  
Slow, steady, progressive disease process.
2. *Episodic burst theory* (Goodson et al; 1982; Zimmerman 1986):  
Irregular periods of exacerbation and remission.
3. *Asynchronous burst theory* (Socransky et al 1984):  
Periods of exacerbation and remission during defined period
4. *Epidemiologic model* (Cohen et al 1988)  
Consistent with continuous disease aging process that depends only on the duration of the process

5. *Brownian motion or stochastic model* (Manji et al 1989)

Random periods of sharp bursts and/or remission can occur, but underlying disease activity remains constant.

6. *Random walking model* (Manji et al 1989)

When observed at regular intervals, model is similar to Brownian motion model.

7. *Fractal model* (Landini et al 1991)

Multifactorial model; simulates disease advancing with age in bursts and remissions

8. Sterne et al., presented three models representing different possibilities for disease progression:

- i. Constant progression,
- ii. Instantaneous increments in progression model
- iii. Varying non-instantaneous progression model.

9. Yang et al.

A. The constant model in which there is no detectable site-specific attachment level change, with the mean of the replicative relative attachment loss values being constant at each visit;

B. The random fluctuation model, in which the mean of the replicative relative attachment loss values changes at each visit but appears to fluctuate around a constant;

C. The gradual model, with a constant gradual increase in visit mean relative attachment loss;

D. The single burst model;

E. The multiple burst model, which included the epidemiological model;

F. The random walk model based on the Brownian motion model; and

G. The autoregressive time series model, a statistical model.

H. The autoregressive model produced a better result than the other models, suggesting that predictive capability is more of a whole-mouth rather than a site-specific phenomenon.

organic

1970 and

mmerman 1986):

1984):

defined period

rocess that depends only

**DISCLOSING AGENTS****I) Soben Peter - Preventive and Community Dentistry 3<sup>rd</sup> Edition**

- A. Iodine preparations:
  - Skinner's iodine solution
  - Diluted tincture of iodine
- B. Mercurochrome preparations
  - Mercurochrome solution 5%
  - Flavoured mercurochrome disclosing solution
- C. Bismark Brown
- D. Merbromin
- E. Fast green
- F. Fluorescein
- G. Two tone solutions
- H. Basic fuchsin

**ENDO-PERIO LESION****I) According to Walker et al. 2000**

- a) Endodontic lesion
- b) Periodontal lesion
- c) True combined lesion
- d) Iatrogenic lesion

**II) Classification by Franklin. S. Weine (1972):**

The classification is based on the etiology of the disease, which determines the type of therapy required and the probable prognosis.

- a) Class I - Tooth in which symptoms clinically and radiographically simulate periodontal disease but are in fact due to pulpal inflammation and/or necrosis.
- b) Class II - Tooth that has both pulpal or periapical disease and periodontal disease concomitantly.



- c) Class III- Tooth that has no pulpal problem but requires endodontic therapy plus root amputation to gain periodontal healing.
- d) Class IV- Tooth that clinically and radiographically simulates pulpal or periapical disease but in fact has periodontal disease.

**III) Simon J.H, Glick and Frank (1972)** classified endo-perio lesions based on etiology, diagnosis, prognosis and treatment:

- a) Primary endodontic lesion
- b) Primary endodontic lesion with secondary periodontal disease
- c) Primary periodontal lesion
- d) Primary periodontal lesion with secondarily endodontic involvement
- e) True combination of endodontic-periodontal lesions.

**IV) Stock (1988) modified Simon's classification.**

Omitted Class V of the classification.

He argued that both Class II and Class IV lesions in advanced stages can become combined lesions and therefore a separate class to describe these lesions was not necessary.

**V) Classification according to Mutschelknauss (1975) and Guldener(1975)**

- a) Lesions of endodontic origin with periodontal involvement.
    - (a) Expansion of the pulp lesion, either periapically or interradiarily via the accessory and lateral canals
    - (b) Iatrogenic: by perforations
  - b) Lesions of periodontal origin with endodontic involvement
    - (a) Iatrogenic: Periodontal therapy requires hemisection/ apicectomy
    - (b) Retrograde pulp infection
  - c) Combined endodontic-Periodontal lesions where two independent defects have merged in to one.
-

- I) According to Oliet, Pallock and Grossman 1968 – Based on treatment procedures:
- Lesions that require endodontic treatment procedures only.
  - Lesions that require periodontal treatment procedures only.
  - Lesions that require combined endodontic – periodontic treatment procedures.
- II) Classification according to Geurtsen et al. (1985)
- Combined lesions requiring only a single root-canal treatment (favorable prognosis)
  - Combined lesions requiring both endodontic and periodontal treatments (less favorable prognosis)
  - Combined lesions with little hope of successful treatment (poor prognosis)
- III) Clinical classification was provided by Torabinejad and Trop in 1996, based on the origin of the periodontal pocket:
- Endodontic origin
  - Periodontal origin
  - Combined endo-perio lesion
  - Separate endodontic and periodontal lesions
  - Lesions with communication
  - Lesions with no communication.
- IX) Based on endodontic therapy (Rateitschak et al 1988):
- Type I: It is primarily of endodontic origin and the pulp is usually dead.
  - Type II: It is basically periodontal disease, sometimes affects the pulp, and the pulp is usually normal, sometimes damaged by ascending pulpitis.
  - Type III: It is a combined case of a root canal problem, periodontal disease, and the pulp is usually dead.

- X) Based on the primary disease with its secondary effect (Khalid S. Al-Fouzan 2014)
- a) Retrograde periodontal disease:
    - 1) Primary endodontic lesion with drainage through the periodontal ligament
    - 2) Primary endodontic lesion with secondary periodontal involvement
  - b) Primary periodontal lesion
  - c) Primary periodontal lesion with secondary endodontic involvement
  - d) Combined endodontic-periodontal lesion
  - e) Iatrogenic periodontal lesions.

## FURCATION

### FURCATION CLASSIFICATION

- 1) Glickman (1958)
  - 2) Staffileno (1969)
  - 3) Goldman and Cohen (1980)
  - 4) Heins & Canter (1968)
  - 5) Easley & Drennan classification (1969)
  - 6) Hamp (1975)
  - 7) Lindhe & Nyman (1975)
  - 8) Ramjford & Ash (1979)
  - 9) Riccheti (1982)
  - 10) Lindhe (1983)
  - 11) Eskow and Kapin (1984)
  - 12) Tarnow & Fletcher (1984)
  - 13) Fedi (1985)
  - 14) Hamp and Nyrnan (1989)
  - 15) Basaraba (1990)
  - 16) Hou et al (1998)
-

**I) Glickman's Classification (1958)**

- a) **Grade-I:** It is the incipient or early stage of furcation involvement. The pocket is suprabony and primarily affects the soft tissue. Early bone loss may have occurred with an increase in probing depth, but radiographic changes are not usually found.
- b) **Grade-II:** Can affect one or more of the furcations of the same tooth. The furcation lesion is a cul-de-sac, with a definite horizontal component. If multiple defects are present, they do not communicate with each other. Vertical bone loss may be present. Radiographs may or may not depict the furcation involvement, especially in the maxillary molars due to the radiographic overlap of roots.
- c) **Grade III:** The bone is not attached to the dome of the furcation. In early Grade III, the furcation opening may be filled with soft tissue. Properly exposed and angled radiographs display the defect as radiolucency in the furcation area.
- d) **Grade IV:** The interdental bone is destroyed, and the soft tissues have receded apically so that the furcation opening is clinically visible. The periodontal probe passes readily from one aspect of the tooth to another.

**II) Tarnow and Fletcher (1984)**

Based on vertical component, each grade of furcation was divided into 3 sub-groups depending on the distance between the bottom of the defect to roof of the furcation.

- a) Subgroup A: 1-3 mm
- b) Subgroup B: 4-6 mm
- c) Subgroup C: >7 mm

**III) Goldman's Classification (1958)**

Grade I: Incipient.

Grade II: Cul-de-sac

Grade III: Through and through

**IV) Easley and Drenan Classification (1969)**

Grade I : Normal furca.

Grade II :Furca opening wide, only horizontal component present.

Grade III: Both vertical as well as horizontal components are present

**V) Hamp's Classification (1975)**

Degree I: Horizontal loss of periodontal tissue support less than 3 mm.

Degree II: Horizontal loss of support > 3 mm but not encompassing the total width of the furcation.

Degree III: Horizontal through and through destruction of the periodontal tissue in the furcation.

**VI) Ramfjord and Ash classification (1979)**

Class I: Beginning involvement. Tissue destruction < 2 mm [less than 1/3<sup>rd</sup> the tooth width] into the furcation.

Class II: Cul-de-sac. Tissue destruction > 2 mm [ $> 1/3^{\text{rd}}$  the tooth width] but not through and through.

Class III: Through and through destruction.

**VII) Rosenberg's Classification (1980)**

1: Denotes grade I horizontal component of bone loss.

2: Denotes grade II horizontal component of bone loss.

2A: Denotes grade II horizontal component of bone loss with shallow vertical component of bone loss.

2B: Denotes grade II horizontal component of bone loss with deep component of vertical bone loss.

3: Denotes grade III horizontal component of bone loss

3A: Denotes grade III horizontal component of bone loss with shallow vertical component of bone loss.

3B: Denotes grade III horizontal component of bone loss with deep vertical component of bone loss.

---

**VIII) Ricchetti classification (1982)**

Class I: 1 mm of horizontal measurement

Class Ia: 1-2 mm of horizontal invasion; earliest damage.

Class II: 2-4 mm of horizontal invasion.

Class IIa: 4-6 mm of horizontal invasion.

Class III: >6 mm of horizontal invasion

**IX) Lindhe's Classification (1983): Based on horizontal bone loss.**

Grade I: Loss of inter radicular bone less than or equal to 1/3rd the horizontal tooth width

Grade II: Loss of inter radicular bone greater 1/3<sup>rd</sup> tooth width but not through and through.

Grade III: Through and through loss of inter radicular bone.

**X) Eskow and Kapin Classification (1984)**

Similar to Tarnow & Fletcher but used thirds instead of mm

Class A: Vertical osseous defect upto 1/3<sup>rd</sup> of root

Class B: Vertical osseous defect upto 2/3<sup>rd</sup> of root

Class C: Vertical osseous defect > 2/3<sup>rd</sup> of root

**XI) Fedi JR Classification (1985)**

He combined the Glickman and Hamp classification.

The Grades I through IV have been retained.

But grade II furcation involvement is sub divided into degree I (<3 mm) and degree II (>3 mm).

**XII) Basarba's Classification (1990)**

Class I: Incipient – moderate and uniform horizontal bone loss with a soft tissue pocket extending into the furcal lesion.

Class II: The potent furcal invasion. This type of invasion creates deep pockets and varying degree of bone destruction into the region of the furca. There is no through and through communication. The path of destruction into the furca is in a horizontal direction and extends several millimeters.

Class III: Communicating furcal lesion. It is a patent exposure that communicates with a second or third furcal opening.

**XIII) Hou Classification (1998)**

Hou proposed a classification of molar furcation involvement based on the root trunk and horizontal and vertical probing attachment levels.

**Root Trunk**

**Furcation Class**

- |   |  |
|---|--|
| <ul style="list-style-type: none"> <li>• Type A<br/>(Cervical 1/3rd)</li> </ul> | <ul style="list-style-type: none"> <li>Class I (&lt;3 mm)</li> <li>Class II (&gt;3 mm)</li> <li>Class III (Through and through)</li> </ul> |
| <ul style="list-style-type: none"> <li>• Type B<br/>(Middle 1/3rd)</li> </ul>   | <ul style="list-style-type: none"> <li>Class I (&lt;3 mm)</li> <li>Class II (&gt;3 mm)</li> <li>Class III (Through and through)</li> </ul> |
| <ul style="list-style-type: none"> <li>• Type C<br/>(Cervical 2/3rd)</li> </ul> | <ul style="list-style-type: none"> <li>Class I (&lt;3 mm)</li> <li>Class II (&gt;3 mm)</li> <li>Class III (Through and through)</li> </ul> |

**XIV) Staffilonos Classification 1969**

Based on location and number of bony walls and degree of furcal exposure

Class 1: A soft furcation lesion with minimal osseous destruction

Class 2: Soft tissue lesion with variable degree of osseous destruction but not through and through

Class 2F: Osseous destruction on facial aspect

Class 2L: Osseous destruction on lingual aspect

Class 2M: Osseous destruction on mesial aspect

Class 2D: Osseous destruction on distal aspect

Class 3: Through and through

**XV) Eskow and Kapin (1984)**

Class A: Vertical osseous defect 1/3<sup>rd</sup> of root

Class B: Vertical osseous defect up to 2/3<sup>rd</sup> of root

Class C: Vertical osseous defect beyond 2/3<sup>rd</sup> of root

**FRENAL ATTACHMENTS****I. Depending upon the extension of attachment of fibers, frenal attachments have been classified as:**

(Ref: Mirko P, Miroslav S, Lubor M. Significance of the labial frenum attachment in periodontal disease in man. Part I. Classification and epidemiology of the labial frenum attachment. J Periodontal. 1974; 45:891-4.)

1. Mucosal – when the frenal fibers are attached up to mucogingival junction
2. Gingival – when fibres are inserted within attached gingiva
3. Papillary – when fibres are extending into inter dental papilla;
4. Papilla penetrating – when the frenal fibres cross the alveolar process and extend up to palatine papilla

**II. Other variations in frenal attachments**

(Ref: Anubha N, Chaubey KK, Arora VK, Narula IS. Frenectomy combined with a laterally displaced pedicle graft. Indian J Dent Sci. 2010; 2:47-51.)

1. Simple frenum with a nodule
2. Simple frenum with appendix
3. Simple frenum with nichum
5. Bifid labial frenum
6. Persistent tecto labial frenum
7. Double frenum
8. Wider frenum



**III. Kotlow infant and newborn maxillary lip-tie diagnostic classifications** (based upon insertion location of the frenum to the upper jaw) (Kotlow L. Oral diagnosis of abnormal frenum attachments in neonates and infants. J Pediatr Dent Care. 2004; 10(3):26-28.)

1. Class I: Minimal visible attachment
2. Class II: Attachment into the area where the free and attached gingival tissue meet
3. Class III: Inserts just in front of anterior papilla
4. Class IV: Attachment just into the hard palate or anterior papilla area

**IV. House classification (Nallaswamy)**

**Classification of broader frenal attachments**

Class I: Attachments are placed away from the crest of the ridge. There is atleast 0.5 inches distance between the attachment and the crest of the ridge.

Class II: Distance between the attachment and the crest of the ridge is 0.25 to 0.5 inches.

Class III: Distance between the attachment and the crest of the ridge is less than 0.25.

**V. House frenal attachment classifications**

Class I: The frenum is located away from the crest of the ridge

Class II: The frenum is located nearer to the crest of the ridge

Class III: The frenum is encroached the ridge.

**CLASSIFICATION OF FLAPS (Carranza)**

I) Based on bone exposure after flap reflection

a) Full thickness flap

b) Partial thickness flap

II) Based on placement of flap after surgery

a) Nondisplaced flaps

b) Displaced flaps

- III) Based on management of papilla
- a) Conventional flap
  - b) Papilla preservation flap

### FIBROBLASTS

According to Weissmanshomer 1975

1. Depending on the activity
  - a) Active fibroblasts
  - b) Inactive or resting fibroblasts
2. Depending on the shape
  - a) Round
  - b) Spindle

### FREMITUS TEST GRADES

Grade I: Mild vibrations – Mild perceptible

Grade II: Moderate vibrations – Moderate definitive

Grade III: Visible movement of teeth

### GINGIVAL RECESSION

#### I) Sullivan and Atkins (1960)

- a) Shallow-narrow
- b) Shallow-wide
- c) Deep-narrow
- d) Deep-wide

#### II) P. D. Miller (1985)

- a) **Class I:** Marginal tissue recession that does not extend to the mucogingival junction. There is no loss of bone or soft tissue in the interdental area. This type of recession can be narrow or wide.

- b) **Class II:** Marginal tissue recession that extends to or beyond the mucogingival junction. There is no loss of bone or soft tissue in the interdental area. This type of recession can be subclassified into wide and narrow
- c) **Class III:** Marginal tissue recession that extends to or beyond the mucogingival junction; in addition, there is bone and/or soft tissue loss interdentally or there is malpositioning of the tooth.
- d) **Class IV:** Marginal tissue recession that extends to or beyond the mucogingival junction with severe bone and soft tissue loss interdentally and/or severe tooth malposition.

### III) Mahajans classification (2010)

Class I: Gingival recession defects not extending to the muco gingival junction.

Class II: Gingival recession defects extending to the muco gingival junction/beyond it.

Class III: Gingival recession defects with bone or soft-tissue loss in the interdental area up to cervical 1/3<sup>rd</sup> of the root surface and/or malpositioning of the teeth.

Class IV: Gingival recession defects with severe bone or soft-tissue loss in the interdental area greater than cervical 1/3<sup>rd</sup> of the root surface and/or severe malpositioning of the teeth.

### IV) Mlinek et al, 1973

- a) Shallow- narrow defect <3 mm in both vertical and horizontal defect
- b) Deep-wide defect >3 mm in both vertical and horizontal defect

### V) Bengueet al (1983) classified recessions according to the coverage prognosis:

- a) "U" type—poor prognosis
- b) "V" type—fair prognosis
- c) "I" type—good prognosis

### VI) Index of recession (Roger G. Smith)

A new two-figure index of recession (IR) (e.g., F2-4 asterisk) was described, in which the 1st digit relates to the proportional evaluation of the horizontal extent of GR at the level of CEJ, and the 2nd digit is the vertical extent of GR from CEJ in millimetres.

The asterisk denotes involvement of the muco gingival junction.

The prefixed F (or L) denotes whether GR is facial (or lingual) to the involved root.

### VII) Recession of interdental papilla – Norland and Tarnow (1998)

Class I: There is no loss of interdental bone or soft-tissue.

Class I-A: Gingival margin on F/L aspect lies apical to CEJ, but coronal to MGJ with attached gingiva present between marginal gingiva and MGJ.

Class I-B: Gingival margin on F/L aspect lies at or apical to MGJ with an absence of attached gingiva between marginal gingiva and MGJ. Either of the subdivisions can be on F or L aspect or both (F and L).

Class II: The tip of the interdental papilla is located between the interdental contact point and the level of the CEJ mid-buccally/mid-lingually. Interproximal bone loss is visible on the radiograph. This is sub-classified into three categories:

Class II-A: There is no marginal tissue recession on F/L aspect

Class II-B: Gingival margin on F/L aspect lies apical to CEJ but coronal to MGJ with attached gingiva present between marginal gingiva and MGJ

Class II-C: Gingival margin on F/L aspect lies at or apical to MGJ with an absence of attached gingiva between marginal gingiva and MGJ.

Either of the subdivisions can be on F or L aspect or both (F and L).

Class III: The tip of the interdental papilla is located at or apical to the level of the CEJ mid-buccally/mid-lingually. Interproximal bone loss is visible on the radiograph. This is sub-classified into two categories.

Class III-A: Gingival margin on F/L aspect lies apical to CEJ, but coronal to MGJ with attached gingiva present between marginal gingiva and MGJ.

Class III-B: Gingival margin on F/L aspect lies at or apical to MGJ with an absence of attached gingiva between marginal gingiva and MGJ.

Either of the subdivisions can be on F or L aspect or both (F and L).

### VIII) Classification of palatal gingival recession. Proposed classification of gingival recession (Ashish Kumar and Sujata Surendra Masamatti 2013)

The position of interdental papilla remains the basis of classifying gingival recession on palatal aspect. The criteria of sub-classifications have been modified to compensate for the absence of MGJ.

PR-I deals with marginal tissue recession on palatal aspect with no loss of interdental bone or soft-tissue.

PR-II and PR-III deal with the loss of interdental bone/soft-tissue with marginal tissue recession on palatal aspect.

#### **Palatal recession-I**

There is no loss of interdental bone or soft-tissue. This is sub-classified into two categories:

PR-I-A: Marginal tissue recession  $\leq 3$  mm from CEJ

PR-I-B: Marginal tissue recession of  $>3$  mm from CEJ

#### **Palatal recession-II**

The tip of the interdental papilla is located between the interdental contact point and the level of the CEJ mid-palatally. Interproximal bone loss is visible on the radiograph. This is sub-classified into two categories:

PR-II-A: Marginal tissue recession  $\leq 3$  mm from CEJ

PR-II-B: Marginal tissue recession of  $>3$  mm from CEJ

#### **Palatal recession-III**

The tip of the interdental papilla is located at or apical to the level of the CEJ mid-palatally. Interproximal bone loss is visible on the radiograph. This is sub-classified into two categories:

PR-III-A: Marginal tissue recession  $\leq 3$  mm from CEJ

PR-III-B: Marginal tissue recession of  $>3$  mm from CEJ.

### IX) Jemts classification (1997)

Grade 0: No papilla present

Grade 1: Less than half of the height of the papilla present

Grade 2: Half or more of the papilla present

Grade 3: The papilla fills up the entire papillary space

Grade 4: The papilla are hyperplastic

X) **Cardapolis classification** – Based on the positional relationship among the papilla, CEJ and adjacent teeth

Papilla presence index score 1 - When the papilla is completely present and coronal to the contact.

Papilla presence index score 2 - point and at the same level as adjacent teeth.

Papilla no longer completely present and lies apical to the contact point and not at the same level as adjacent papilla.

Papilla presence index score 3 - Papilla has moved apically and the interproximal CEJ becomes visible.

Papilla presence index score 4 - Papilla lies apical to both the interproximal CEJ and buccal CEJ.

## GINGIVAL ENLARGEMENT

I) According to etiopatogenesis (Carranza 11<sup>th</sup>ed, pg-118)

### 1. Inflammatory enlargement

A. Chronic

B. Acute

### 2. Drug-induced enlargement

### 3. Enlargements associated with systemic diseases/ conditions

#### A. Conditioned enlargement

1. Pregnancy

2. Puberty

3. Vitamin C deficiency

4. Plasma cell gingivitis

5. Nonspecific conditioned enlargement  
(Granuloma pyogenicum)

**B. Systemic diseases causing gingival enlargement**

1. Leukemia
2. Granulomatous diseases (Wegener's granulomatosis, sarcoidosis etc.)

**4. Neoplastic enlargement (gingival tumors)**

- A. Benign tumors
- B. Malignant tumors

**5. False enlargement**

**II. According to location and distribution (Carranza 11<sup>th</sup> ed., pg-118)**

- 1. Localized:** Limited to the gingiva adjacent to a single tooth or group of teeth
- 2. Generalized:** Involving the gingiva throughout the mouth
- 3. Marginal:** Confined to the marginal gingiva
- 4. Papillary:** Confined to the interdental papilla
- 5. Diffuse:** Involving the marginal and attached gingiva and papillae
- 6. Discrete:** An isolated sessile or pedunculated tumor like enlargement

**III. Grading of gingival enlargement**

The degree of gingival enlargement can be scored as follows:

**1. Bokenkamp A and Bonhorst B(1994) (Buchner A, Hansen AS 1979)**

**Grade 0:** No sign of gingival enlargement

**Grade 1:** Enlargement confined to interdental papilla

**Grade 2:** Enlargement involving papillary and marginal gingiva

**Grade 3:** Enlargement covering three quarters or more of the crown

**2. Aas et al. (1963)**

Developed a gingival index using **4 grades**

From **slight** to **very severe** gingival hyperplasia.

---

### 3. Angelopoulos and Goaz (1972)

**Grade 0:** No gingival enlargement

**Grade 1:** No more than **one third** of the clinical crown covered

**Grade 2:** Any part of the **middle third** of the crown covered

**Grade 3:** Greater than **two-thirds** of the crown covered

### 4. Barak et al. (1987)

Complex classification system based on **length of rete pegs** (acanthosis) as determined by **histological examination**.

**Grade 1:** Normal width of epithelium: 0.30 to 0.50 mm

**Grade 2:** Slight hyperplasia: 0.50 to 1.5 mm

**Grade 3:** Moderate hyperplasia: 1.50 to 3.0 mm

**Grade 4:** Severe hyperplasia: 3 to 4 mm

### 5. Babcock's index (1965)

- **Mild – Minimal** gingival enlargement
- **Moderate – Slight but definite** gingival enlargement not interfering with function
- **Severe –** Gingival enlargement interfering with **function**

### 6. Conard et al. (1974)

**Conard et al.** grouped the teeth in **segments** and classified each segment by **degree of hyperplasia** on scale of 0-4.

- 0- No clinical signs of hyperplasia.
- 4-Teeth were completely covered with hyperplastic tissue.

### 7. Eva Ingles (1999)

**Grade 0:** **No overgrowth**, knife-edge papilla and no increase in the density of gingiva.

**Grade 1:** **Early overgrowth**, tip of papilla rounded and increase in density of gingiva.

**Grade 2:** **Moderate overgrowth**, size of papilla increased and is retractable.



**Grade 3: Marked overgrowth**, gingival contour is convex

**Grade 4: Severe overgrowth**, large parts of clinical crown covered.

**8. Ingle et al., (1959)**

Utilized a mm scale to obtain measurement on **study casts**.

Distance was measured between **height of the tissue and incisal edge of all six anteriors**.

Three points were recorded on each tooth.

- Incisal edge to mesial papilla
- Incisal edge to distal papilla
- Incisal edge to marginal gingiva

**Average measurement** was obtained that could be compared with **additional casts** for each patients.

These average were converted to **percentage** and **charted** as an increase or decrease in tissue level.

**9. Harris Walt index (1942)**

A) Grade 0: No clinical evidence of overgrowth.

B) Grade 4: Enlargement covering at least 3/4<sup>th</sup> crown.

**10. Mc Gaw et al. (1987)**

Assessed gingival overgrowth using **modification of semiquantitative index** developed by Aas et al.

He divided patients in to two groups, **responders and non-responders**.

Those patients with score of grade 1 or less were considered non-responders.

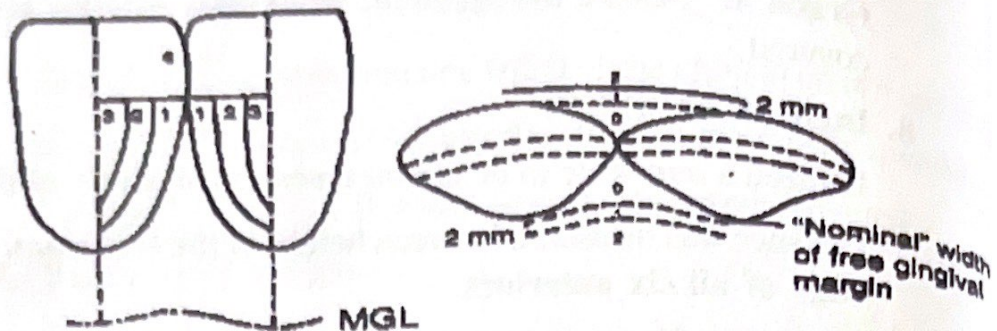
**11. King et al (1993)**

Classified and graded each gingival unit from the **study cast of 12 anterior tooth** using a hyper plastic index consisting of 2 components.

I) **Vertical dimention**—as used by Aas, conard, Mcgaw

II) **Horizontal dimention** as used by Seymour.

## 12. Seymour et al (1985)



The increase in size of the papilla was measured from the enamel surface, at the inter-dental contact point, to the outer papillary surface.

Two scores were obtained, one for the buccal papilla and another for the lingual/ palatal papilla, according to the following criteria:

Mesiodistal enlargement is measured as shown in the diagram:

Included both thickening and encroachment. GO assessed on a plaster model in upper and lower segments.

Grade 0: Normal

Grade 1: Thickening from normal upto 2 mm,

Grade 2: Thickening >2 mm

## 13. Miranda and Brunet -2001

Measured GO in buccolingual direction both for buccal and lingual/ palatal papilla.

0 – Papillary thickness < 1 mm

1 – Papillary thickness 1-2 mm

2 – Papillary thickness > 2 mm

## GROWTH FACTORS

### I. Extracellular matrix

- a. Enamel matrix protein
- b. Fibronectin
- c. Citric acid
- d. Adhesion factors like – Osteopontin and bone salioproteins

### II. Polypeptide growth factors

#### A. Mitogenic polypeptides

1. Platelet derived growth factor
2. Insulin-like growth factor
3. Fibroblast growth factor
4. Transforming growth factor
5. Epidermal growth factor
6. Cementum-derived growth factor

#### B. Differentiation polypeptides

- Bone morphogenic proteins

### III. Arachidonic acid metabolites

- Prostaglandin E1

## GTR MEMBRANES

### I. Minabe classification (1991)

- a) Non resorbable membranes
- b) Resorbable membranes

### II. Gottlow's classification (1993)

- a) First generation (Nonresorbable)
  1. Millipore filter
  2. Polytetrafluoroethylene membrane
  3. Expanded polytetrafluoroethylene membrane (e-PTFE)
  4. Nucleopore membrane.
  5. Rubber dam.

## b) Second generation (resorbable)

1. Collagen membrane.
2. Polylactic acid membrane. (guidor)
3. Vicryl mesh
4. Cargile membrane.
5. Oxidised cellulose membrane
6. Hydrolyzable polyester

## c) Third generation (resorbable with growth factors)

- Inion launched the first 3rd generation Dental Membrane System in Europe [06.08.2003]

**III. Types of barrier membranes**

## a. Non-absorbable barriers

- Millipore filters
- Rubber filters
- Rubber dam
- e- PTFE membranes
- Ethyl cellulose
- Bio – brane knitted nylon fibre

## b. Absorbable barriers

## 1. Natural products

**Collagen:**

- Bio – gide- paro
- Bio – mend
- Paraguide
- Avitene
- Collistat
- Dura mater
- Cargile membrane
- Laminar bone
- Connective tissue graft

2. Synthetic products

i) Polylactic acid derivatives:

- Atrisorb
- Guidor
- Osmed
- Epiguide

ii) Combination of PLA and PGA:

- Ethisorb
- Resolut
- Vicryl mesh.



**HABITS:**

I. James (1923)

- a. Useful habits
- b. Harmful habits

II. Morris and Bohanna (1969)

- a. Pressure habits, non pressure habits
- b. Biting habits

III. Klein (1971)

- a. Empty habits
- b. Meaningful habits

IV. Finn (1987)

- a. Compulsive habits
- b. Non compulsive habits

V. Kingsley

- Functional
- Muscular
- Postural
- Combined

## VI. Soren (1935)

**Neurosis** - Lip, cheek, pencil, tooth pick, nail biting, Tongue thrusting, occlusal.

**Occupational** - Holding nails or needles in the mouth by carpenters and cobblers and pressure of the reed while playing musical instruments.

**Miscellaneous** - Pipe and tobacco chewing, smoking, incorrect brushing method, mouth breathing and thumb sucking.

## HALITOSIS

## I. Based on etiology, halitosis can be divided into the following categories (Dominic et al 1982):

- a) Local factors of pathological origin
- b) Local factors of non-pathological origin
- c) Systemic factors of non-pathological origin
- d) Systemic factors of pathological origin.

## II. Based on causes it can be also classified as (Bogdasarian 1986):

- a) Normal breath and physiologic mouth odour
- b) Odors from oral conditions
- c) Odors from nasopharynx, pharynx and lungs
- d) Odors excreted via the lungs.

## III. Dayan et al divided foul odour into 3 groups :

- a) Odor emanating within oral cavity.
- b) Odor emanating from regions immediately adjacent to oral cavity.
- c) Odor emanating from lungs.

## IV. Glickman 1894

- a) Local causes
  - Pathologic, non pathologic
- b) Systemic causes
  - Pathologic, non pathologic

V. Classification of halitosis with corresponding treatment needs (Miyazaki et. al)

Table 3:

Classification	Treatment Need	Description
I. Genuine halitosis		Obvious malodour, with intensity beyond socially acceptable level, is perceived.
I.A. Physiologic halitosis	TN-1:	<ol style="list-style-type: none"> <li>1. Malodour arises through putrefactive process within the oral cavity. Neither specific disease, nor pathologic condition that could cause halitosis is found.</li> <li>2. Origin is mainly the dorsoposterior region of the tongue.</li> <li>3. Temporary halitosis due to dietary factors (e.g., garlic) should be excluded.</li> </ol>
I.B. Pathologic halitosis		
(i) Oral	TN-1 and TN-2:	<ol style="list-style-type: none"> <li>1. Halitosis caused by disease, pathologic condition, or malfunction of oral tissues.</li> <li>2. Halitosis derived from tongue coating, modified by pathologic condition (e.g., periodontal disease, xerostomia) is included in this subdivision.</li> </ol>
(ii) Extraoral	TN-1 and TN-3:	<ol style="list-style-type: none"> <li>1. Malodour originates from nasal, pernasal, and/or laryngeal regions.</li> <li>2. Malodour originates from pulmonary tract or upper digestive tract.</li> <li>3. Malodour originates from disorders anywhere in the body, whereby, the odour is blood-borne and emitted via the lungs (e.g., diabetes, hepatic cirrhosis, uremia, internal bleeding).</li> </ol>
II. Pseudo-halitosis	TN-1 and TN-4:	<ol style="list-style-type: none"> <li>1. Others do not perceive obvious malodour although the patient stubbornly complains of its existence.</li> <li>2. Condition is improved by counseling (using literature support, education, and explanation of examination results) and simple oral hygiene measures.</li> </ol>
III. Halitophobia	TN-1 and TN-5:	<ol style="list-style-type: none"> <li>1. After treatment for genuine halitosis or pseudo-halitosis, the patient persists in believing that he/she has halitosis.</li> <li>2. No physical or social evidence exists to suggest that halitosis is present.</li> </ol>

Table 4:

Category	Description
TN-1	Explanation of halitosis and instructions for oral hygiene (support and reinforcement of patients own self care for further improvement of their oral hygiene)
TN-2	Oral prophylaxis, professional cleaning and treatment for oral diseases, especially periodontal diseases.
TN-3	Referral to a physician or medical specialist.
TN-4	Explanation of examination data, further professional instruction, education and reassurance.
TN-5	Referral to a clinical pshychologist, psychiatrist or other psychological specialist.

## HOST MODULATION AGENTS

### I. According to Kenneth Kornman 1999

- a. Blocking direct effects of bone and connective tissue destruction  
bisphosphonates
- b. Blocking host mechanism that influence clinical outcome
  1. Nonsteroidal anti-inflammatory drugs
  2. Inhibitors of IL-1 and TNF
- c. Host mechanisms that influence bacterial control

### II. According to Ryan ME 2002

- a. Inhibition of matrix metalloproteinases (MMPs): This is achieved by chemically modified tetracyclines (CMTs)
- b. Inhibition of arachidonic acid metabolites: Through NSAIDs
  1. COX-1 inhibitors: Indomethacin, flurbiprofen, naproxen
  2. COX-2 inhibitors: Rofecoxib
  3. COX and LOX inhibitors: Triclosan, topical ketoprofen
  4. LOX inhibitors: Lipoxins
- c. Modulation of bone metabolism
  1. Bisphosphonates



2. Hormone replacement therapy (HRT)
3. Calcium supplementation
- d. Regulation of immune and inflammatory response
  1. Suppressing proinflammatory cytokines: IL-1 and TNF- $\alpha$  receptor antagonist.
  2. Nitric oxide inhibition
  3. Generation of protective antibodies through vaccination
  4. Infusion/supplementary anti-inflammatory cytokines: IL-4 and IL-10

**III. Reddy MS et al, 2003**

- a. Antiproteinases: Tetracyclines
- b. Anti-inflammatory: NSAIDs
- c. Bone sparing agents

**IV. According to Salvi GE and Lang NP 2005**

- a. Modulation of arachidonic acid metabolites
- b. Modulation of MMPs
- c. Modulation of bone remodeling
- d. Modulation of NOS activity

**V. According to Kantarci et al, 2006**

- a. Pro-resolution agents: lipoxins, resolvins
- b. Anti-inflammatory agents: TIMS, tetracyclines, bisphosphonates

**VI. According to Carranza 10<sup>th</sup> edition**

- a. Systemically administered agents
    1. NSAIDs
    2. Bisphosphonates
    3. Subantimicrobial dose doxycycline
  - b. Locally administered agents
    1. NSAIDs
    2. Enamel matrix proteins, growth factors and bone morphogenic proteins
-

## HEMOSTATIC TECHNIQUES

### 1. Mechanical techniques

- Direct pressure
- Sutures
- Staples
- Ligating clips
- Fabric pads
- Gauzes
- Sponges
- Blood component/replacement

### 2. Thermal techniques

- Electrocautery
- Hemostatic scalpel
- Laser

### 3. Chemical techniques

#### a. Pharmacotherapy

- Hypotensive anesthesia
- Epinephrine
- Vitamin K
- Protamine
- Desmopressin
- Aminocaproic acid
- Tranexemic acid

#### b. Topical hemostats

- Collagen
- Cellulose
- Gelatin
- Thrombin-2

- c. Topical sealants and adhesives
  - Fibrin sealants
  - Synthetic glues

## INSTRUMENTS

### I. Periodontal Instruments: Jill S. Nield-Gehrig

- a. Assessment instruments – Periodontal probes  
Explorers
- b. Calculus removal instruments – Sickle scalers  
Periodontal files  
Curettes

### II. Glickman

According to the purpose:

- A. Periodontal probe
- B. Explorer
- C. Scaling, root planing and curettage instruments - Sickle scalers, curettes, hoe, chisel and files, ultrasonic and sonic instruments
- D. Periodontal endoscope
- E. Cleaning and polishing instruments

### III. According to Use:

- a) Examination /diagnostic instruments
- b) Scaling instruments
- c) Cleaning and polishing instruments
- d) Periodontal surgical instruments
- e) Electrosurgical instruments

### IV. Surgical Instruments: Larry J Peterson

- a) Incise
- b) Elevating mucoperiosteum
- c) Controlling hemorrhage

- d) Removing bone
- e) Grasp tissues
- f) Renewal of soft tissue form
- g) Removal of bone defect
- h) Providing suction
- i) Transfer sterile instruments
- j) Irrigation
- k) Suturing- needle holder, needles, sutures, scissors

## INDICES

According to Essentials of Preventive and Community Dentistry  
(Soben Peter, 2<sup>nd</sup> edition)

### Classification of Indices

**I. Based upon the direction in which their scores can fluctuate, indices are classified as either:**

**A. Reversible:** Index that measures conditions that can be changed. Reversible index scores can increase or decrease on subsequent examinations.

Eg: Indices that measure periodontal conditions.

**B. Irreversible:** Index that measures conditions that will not change. Irreversible Index scores, once established cannot decrease in value on subsequent examinations.

Eg: An Index that measures dental caries.

**II. Depending upon the extent to which areas of oral cavity are measured, indices are classified into**

**A. "Full mouth"** These indices measure the patient's entire periodontium or dentition.

E.g., Russell's Periodontal Index (PI).

**B. "Simplified":** These indices measure only a representative sample of the dental apparatus.

E.g., Greene & Vermillion's Oral Hygiene Index-Simplified (OHI-S).

III. Indices may be classified in certain general categories according to the entity which they measure like:

- A. Disease index
- B. Symptom index
- C. Treatment index

IV. Dental indices can also be classified under special categories as

A. Simple index - Index that measures the presence or absence of a condition.

Eg: An index that would measure the presence of dental plaque without an evaluation of its effect on gingiva.

B. Cumulative index - Index that measures all the evidence of a condition, past and present.

Eg: DMF index for caries

### INTERDENTAL AIDS

According to Carranza 10<sup>th</sup> edition

A. Dental floss

- 1. Nylon / plastic
- 2. Waxed / unwaxed
- 3. Thin / thick
- 4. Braided

B. Interdental brushes

- 1. Conical
- 2. Cylindrical
- 3. Tapered wooden toothpicks
- 4. Single tufted brushes
- 5. Multiple tufted brushes

C. Wooden or rubber tips

- 1. Wooden tip
- 2. Plastic tip

**IMPLANT COMPLICATIONS****I. According to Stuart J et al.****a. Surgical complications**

1. Haemorrhage and haematoma
2. Neurosensory disturbances
3. Implant malpositions

**b. Biological complications**

1. Inflammation and proliferation
2. Dehiscence and recession
3. Peri-implantitis and bone loss
4. Implant loss and failure

**c. Complications related to augmentation procedure**

1. Autogenous bone harvesting / grafting
2. Guided bone regeneration
3. Sinus bone augmentation

**d. Complications related to placement of loading protocols**

1. Immediate implant placement
2. Immediate loading after implant placement
3. Implant placement using a flapless approach

**e. Prosthetic or mechanical complications**

1. Screw loosening and fracture
2. Implant fracture
3. Fracture of restorative materials

**f. Esthetic and phonetic complications**

1. Esthetic complications
2. Phonetic problems

II. According to Lippincott and Williams

a. Treatment plan related complications

1. Wrong angulation
2. Improper implant location
3. Too close
4. Too apart
5. Lack of communication

b. Anatomy related

1. Nerve injury
2. Bleeding
3. Cortical plate perforation
4. Sinus perforation
5. Deviation of adjacent teeth

c. Procedure related

1. Lack of primary stability
2. Mechanical complications
3. Mandibular fracture
4. Ingestion / aspiration

d. Others

1. Iatrogenic
2. Human error

IMPLANT SHAPES (Carranza)

a) Shape – according to the area of application

1. Endosteal
  - i) Ramus plate
  - ii) Root form
  - iii) Blade form

2. Sub periosteal
3. Trans osteal
4. Intra mucosal

**b) Based on macroscopic body design**

1. Cylinder
2. Thread
3. Plateau
4. Perforated
5. Solid
6. Hollow/vented

**IMPLANT SURFACE**

**I) Based on microscopic design (dental implant prosthetic-MISCH)**

1. Smooth
2. Machined
3. Coated
4. Textured

**II) Based on implant surface treatment (Lindhe)**

1. Grit blasted
2. Blasted and etched
3. Etched
4. Hydroxyapatite coated
5. Oxidized surface
6. Titanium plasma sprayed
7. Turned surface



**LOCAL DRUG DELIVERY CLASSIFICATION**

- 1) Jain. N; Jain. G; Javed. S and Iqbal. Z (2008), "Recent approaches for the treatment of periodontitis", Drug Discovery Today. 13, 21-22.  
 K Schwach-Abdellaoui, N and Vivien-Castioni, R (2000), "Gummy Local Delivery of Antimicrobial agents for the treatment of periodontal diseases", European Journal Of Pharmaceutics And Biopharmaceutics, 50, 83 – 99

Table 5:

System	Polymer matrix	Drug incorporate
Fibres	Cellulose acetate	Tetracycline HCl
	Ethylene vinyl acetate	Chlorhexidine
	Poly(e-caprolactone)	Tetracycline HCl
	Polyethyl metha acrylate (acrylic)	Tetracycline
	Hydroxypropyl cellulose	Tetracycline HCl
	HPC + methacrylic acid	Metronidazole
	Ofloxacin	Chlorhexidine, tetracycline
	Polyhydroxybutyric acid	Doxycycline
Strip	Poly lactide-co-glycolic acid (PLGA)	Tetracycline HCl
	Ethyl cellulose	Chlorhexidine
Films	Ethyl cellulose	Metronidazole
	Cross-linked atelocollagen	Minocycline
	Gelatin (BycoW protein)	Tetracycline HCl
	Cross-linked gelatin + glycerine	Tetracycline
	Chitosan	Chlorhexidine diacetate
	Chitosan + PLGA	Chlorhexidine digluconate
	Chitosan + PCL	Taurine
	PLGA	Iproflavone
	Poly(ortho ester),	Metronidazole
	Eudragit LW and Eudragit SW	Tetracycline
	PCL	Metronidazole Clindamycine Minocycline

System	Polymer matrix	Drug incorporate
Gels	Chitosan HEC + polyvinyl pyrrolidone HEC + polycarbophil Poloxamer 407 + Carbopol 934P Glycerol monooleate + sesame oil PLGA	Metronidazole Tetracycline Metronidazole Propolis Metronidazole Tetracycline
Microparticles	Pluronic F 127 PLGA PLGA + PCL	Tetracycline Tetracycline Histatin peptides Doxycycline
Nano particles	PLGA Chitosan Cellulose acetate phthalate PLGA	Harungana madagascariensis leaf extract Antisense oligonucleotide Triclosan
Vesicular system	Phosphatidylinositol Immunoliposomes	Triclosan anti-oralis
Other systems	Poly(ethylene-co-vinyl acetate)	Acyclovir chlorhexidine

II) Carranza, GM, Fermin, AC (2004), "Clinical Periodontology", 9th ed., Saunders, 676-683.

Table 6:

Product	Antimicrobial Agents	Dosage Form	Manufacturer
Actiside®	Tetracycline	Non resorbale fibre	Alzacorp
Arestin®	Minocycline	Biodegradable powder in syringe	Oropharmacorp Warminster
Atridox®	Doxycycline	Biodegradable mix in syringe	AtrixLabs, Ft, Collins, Co
Dentamycin®	Minocycline	Biodegradable mix in syringe	Sunstar Corp., Tokyo, Japan
Elyzol®	Metronidazole	Biodegradable mix in syringe	Dumex Corp.Co Denmark

Periochip® Jerusalem	Chlorhexidine	Biodegradable device	Dexcel Pharma Inc
Periochip®	Chlorhexidine/ Tetracycline	Film	Perioproduts Ltd.
Periochip®	Gluconate	Inserts	Perioproduts Ltd.
Gluconate®	Metronidazole	Inserts	Perioproduts Ltd.
Elyzol®	Minocycline	Gel	Dumex pharma
Atrigel®	Doxycycline	Gel	Atridox (atridox lab)

**LASERS** (Textbook of Principles and Practice of Laser Dentistry;  
Robert A. Convissar)

**I) Based on Application:**

- i. Soft tissue – CO<sub>2</sub>, diode, NdYAG
- ii. Hard tissue – ErbYAG
- iii. Resin covering laser - Argon

**II) Based on mode of contact/Action:**

- i. Contact –Nd YAG
  - ii. Non-contact – CO<sub>2</sub>
- Focused
- Defocused

**III) Based on the form used:**

- i. Solid – Ruby, Nd YAG
- ii. Gas – He, Ne, CO<sub>2</sub>
- iii. Excimers
- iv. Dye
- v. Diode

**IV) Based on radiation generation:**

- i. Continuous
- ii. Discrete
- iii. Multiple timed pulse

**V) Based on emission:**

- i. Low level - Stimulates cellular activity
- ii. High level - Hard lasers- ErYAG, NdYAG

**LOCAL ANESTHESIA** (Handbook of Local Anaesthesia Malamed,,  
6th Edition)**a. Injectable**

## 1. Low potency, short duration

- Procaine

## 2. Intermediate potency and duration

- Lignocaine (lidocaine)
- Prilocaine

## 3. High potency, long duration

- Tetracaine (amethocaine)
- Bupivacaine
- Ropivacaine
- Dibucaine (cinchocaine)

**b. Surface anesthetic**

## 1. Soluble

- Cocaine
- Lignocaine
- Tetracaine
- Benoxinate

## 2. Insoluble

- Benzocaine
- Butylaminobenzoate (butamben)

**II. Based on Chemical Structure****a. Ester group**

## 1. Benzoic acid esters

i) Benzocaine

ii) Cocaine

iii) Butacaine

iv) Tetracaine, piperocaine

2. Para amino benzoic acid esters

- i) Procaine
- ii) Chlorprocaine
- iii) Propoxycaine

**b. Amide group**

- i) Lignocaine
- ii) Bupivacaine
- iii) Mepivacaine
- iv) Prilocaine
- v) Articaine
- vi) Dibucaine
- vii) Etidocaine
- viii) Ropivacaine

**c. Quinolone**

Centbucridine

**III. Based on the Biological Site and Mode of Action**

- a) **Class A:** Acting on receptor site located on external + surface of nerve membrane - Tetrodotoxin
- b) **Class B:** Acting on receptor site located on internal + surface of nerve membrane – Quaternary ammonium analogues of lidocaine
- c) **Class C:** Acting by a receptor-independent physico-chemical mechanism - Benzocaine
- d) **Class D:** Acting by combination of receptor and receptor-independent mechanism - Lidocaine, mepivacaine

**LYMPH NODES**

- 1. In 1991 American Academy of Head and Neck Surgery, classified lymph nodes into levels following a system originally proposed by Memorial Sloan-Kettering Cancer Group, New York, also called Robins classification:

- ia. Submental lymph node
- ib. Submandibular lymph node
- ii. Upper jugular lymph node
- iii. Middle jugular lymph node
- iv. Lower jugular lymph node
- v. Posterior triangle group
- vi. Anterior compartment (central) group

## 2. Based on location in facial spaces

Ref: Essentials of Human Anatomy – A.K. Dutta

### a) Outer circle group

- i) Sub mandibular
- ii) Sub mental
- iii) Pre auricular
- iv) Post auricular
- v) Occipital
- vi) Superficial cervical

### b) Inner circle group

- i) Pre tracheal
- ii) Para tracheal
- iii) Retro pharyngeal
- iv) Waldeyer's ring

### c) Deep cervical group

- i) Upper group
- ii) Lower group

3. Based on pathological consistency

Reference - S. Das - Clinical Manual of Surgery

- a) Normal
- b) Enlarged
  - i) Soft
  - ii) Elastic, rubbery
  - iii) Firm
  - iv) Stony hard
  - v) Variable

**TOOTH MOBILITY**

**I) W D Millers: Classification 1853**

- a) Grade 1: Tooth moves from 0.2 mm to 1 mm in the buccolingual direction
- b) Grade 2: Tooth moves more than 1 mm in the buccolingual direction, but does not move in the apicocoronal direction
- c) Grade 3: Tooth moves in both buccolingual and apicocoronal direction

**II) Carranza, 11<sup>th</sup> edition**

- a) Normal mobility
- b) Grade I: Slightly more than normal
- c) Grade II: Moderately more than normal
- d) Grade III: Severe mobility faciolingually and/or mesiodistally, combined with vertical displacement.

**III) Lindhe et. al, 1983**

- a) Degree 1: Movability of the crown of the tooth 0.2 - 1 mm in horizontal direction.
- b) Degree 2: Movability exceeding 1 mm in a horizontal direction.
- c) Degree 3: Movability in a vertical direction as well.

**IV) Fleszar et. al 1980**

- a) Class 0: Physiologic mobility; firm tooth
- b) Class I: Slightly increased mobility
- c) Class II: Definite to considerable increase in mobility, but impairment of function.
- d) Class III: Extreme mobility; a loose tooth that would be uncomfortable in function (a plus sign can be used for intermediate values, e.g., I+)

**V) Walter B Hall et al, 1984**

- a) Class 0: Minute or no mobility
- b) Class 1: Movement less than 1mm
- c) Class 2: Movement equal to or greater than 1 mm but less than 2 mm
- d) Class 3: Movement equal to or greater 2 mm and or depressible

**VI) Bartolucci et al, 1981**

- a) Degree 0: Absent.
- b) Degree 1: Tooth mobility in a vestibular- lingual direction up to 1 mm.
- c) Degree 2: Tooth mobility in a vestibular- lingual direction by more than 1 mm.
- d) Degree 3: Tooth mobility in a vestibular- lingual direction by more than 1 mm and/or depressibility in the alveolus.

**VII) RM Palmer and PD Floyd -1992**

- a) No detectable movement classically mobility less than 0.2 mm
- b) Horizontal mobility greater than 0.2 mm and less than 0.5 mm
- c) Movement of 0.5 mm-1 mm.
- d) Movement greater than 1 mm or vertical displacement.

**VIII) Muhelman H R 1975 (Degree of tooth mobility)**

- 0 - Normal physiological mobility



- 1 - Detectable mobility
- Elevated mobility
- 2 - Visible mobility up to 0.5 mm
- 3 - Severe mobility up to 1 mm
- 4 - Extreme mobility, tooth no longer functional

### TOOTH MOBILITY INDICES

#### I) Miller's Index (1950)

- a) Class I – first detectable sign of movement
- b) Class II – movement of > 1 mm in any direction
- c) Class III – movement of > 1 mm in any direction and vertical depression or rotation.

#### II) Miller's Modified Scale of Mobility Index (1975)

- a) 0-Physiological mobility
- b) 1-< than 1 mm in faciolingual direction
- c) 2-> than 1 mm faciolingual direction
- d) 3-> than 2 mm faciolingual direction and mesiodistal and also compressible in the socket.

#### III) Glickman's Index (1972)

- Normal mobility
  - Pathologic mobility
- a) **Grade I:** Slightly more than normal
  - b) **Grade II:** Moderately more than normal
  - c) **Grade III:** Severe mobility faciolingually

#### IV) The mobility index, developed by Grace and Smales, can be useful to track the amount of mobility in teeth over a period of time.

- a) Grade 0 indicates no apparent mobility.
- b) Grade 1 is assigned to a tooth in which mobility is perceptible, but less than 1 mm buccolingually.

- c) Grade 2 mobility is between 1-2 mm, and
- d) Grade 3 mobility exceeds 2 mm buccolingually or vertically.

V) **Wasserman's Index (1973)**

- a) **Grade I:** Normal
- b) **Grade II:** Slight mobility less than 1 mm of bucco-lingual movement
- c) **Grade III:** Moderate mobility - up to approximately 2 mm of bucco-lingual movement
- d) **Grade IV:** Severe mobility - more than 2 mm of movement.

VI) **Nyman's Index (1975)**

- a) **Zero degree:** Normal - less than 0.2 mm
- b) **Degree 1:** Horizontal/ Mesiodistal mobility of 0.2 - 1 mm
- c) **Degree 2:** Horizontal/ Mesiodistal mobility of 1-2 mm
- d) **Degree 3:** Horizontal/ Mesiodistal mobility exceeding 2 mm and or vertical mobility

VII) **Flezar's Index (1980) (modification of Miller scale)**

- a) **M0** - Firm tooth
- b) **M1** - Slight increased mobility
- c) **M2** - Definite to considerable increase in mobility, but not impairment of function.
- d) **M3** - Extreme mobility, a loose tooth that would be incomparable in function

VIII) **Lovdal Index (1959)**

- a) **First degree:** Teeth those were somewhat more mobile than normal.
- b) **Second degree:** Teeth showing conspicuous mobility in transversal but not axial direction.
- c) **Third degree:** Teeth being mobile in axial as well as in transversal direction.

**IX) Prichard's Index (1972)**

- a) **Grade I:** Slight mobility
- b) **Grade II:** Moderate mobility
- c) **Grade III:** Extensive movement in a lateral or mesio-distal direction combined with vertical displacement in the alveolus.
- d) **Grade IV** - signs can be used for added refinement

**X) Clinical Implant Mobility (IM) Scale (Carl E Misch 2nd edition)**

- a) 0: Absence of clinical mobility with 500 g in any direction.
- b) 1: Slight detectable horizontal movement.
- c) 2: Moderate visible horizontal mobility upto 0.5 mm.
- d) 3: Severe horizontal movement more than 0.5 mm.
- e) 4: Visible moderate to severe horizontal and any visible vertical movement.

**XI) Ramfjord (1959, 1967). Tooth mobility was scored as follows:**

- 1. MO: Physiologic mobility; firm tooth
- 2. M1: Slightly increased mobility
- 3. M2: Definite to considerable increase in mobility, but no impairment of function
- 4. M3: Extreme mobility; a "loose" tooth that would be uncomfortable in function

**XII) Wasserman, Geiger and Turgen modification of the Miller Index, 1973 which is used at the Columbia Dental School. This method utilizes a 1 to 5 scoring system**

- 1 - Normal tooth mobility.
- 2 - Slight mobility—less than approximately  $\frac{3}{4}$  mm of movement bucco-lingually.
- 3 - Moderate mobility—up to approximately 2 mm of movement bucco-lingually.
- 4 - Severe mobility—more than 2 mm of movement.

## CLASSIFICATION OF BACTERIA (Frank Lowy)

### I. Gram Positive Bacteria

- a. Cocci
  - 1. Aerobe - Staphylococci, Streptococci, Enterococci
  - 2. Anerobe - Peptostreptococci
- b. Rod
  - 1. Aerobe - Bacillus, Listeria, Nocardia
  - 2. Anerobe - Actinomyces, Clostridium

### II. Gram Negative Bacteria

- a. Cocci
  - 1. Aerobic - Facultative anaerobe
  - 2. Anerobe - Veillonella
- b. Rods
  - 1. Aerobe - Lactose fermenters - Enterobacteriaceae  
Non lactose fermenters - Pseudomonas, Hemophilus, vibrio
  - 2. Anerobe - Bacterioids, fusobacterium

- a. Intracellular bacteria - Chlamydia  
Rickettsia  
Borelia

- b. Poorly staining - Mycoplasma  
Legionella  
Helicobacter

- c. Acid fast stain - Mycobacteria  
Nocardia

Classification of Matrix Metallo Proteinases  
Golub et al 1995  
Table 6:

Number	Enzyme	KDaproforma	Preferred substrate
MMP-1	Interstitial collagenase	57/52	Helical collagen, proMMP-2, proMMP-9
MMP-2	Gelatinase A	72/66	ProMMP-9, gelatin, fibronectin, elastin, collagen IV, V, VII X
MMP-3	Stromelysin-1	60/55	Fibronectin, laminin, elastin, proteoglycan, collagen IV, V, IX, X, proMMP-1, 7, 8, 9, 13
MMP-7	Matrilysin	28/19	Fibronectin, elastin, collagen IV
MMP-7	Matrilysin-1		Pro- $\alpha$ -defensins, latent TNFSyndecan-1, E-cadherin, Elastin
MMP-8	Neutrophil collagenase	85-64	Helical collagen
MMP-9	Gelatinase B	92/80	Gelatin, fibronectin, elastin, collagen IV, V, VII e X
MMP-10	Stromelysin-2	60/55	Fibronectin, Laminin, elastin, proteoglycan, collagen IV, V, IX, X
MMP-12	Matrilysin-2	54/22	Elastin
MMP-12	Matrilysin-2		Latent TNF
CMMP-13	Collagenase-3	52-42	Helical collagen
MMP-14	MT1-MMP	66/54	ProMMP-2,-13, Helical collagen
MMP-15	MT2-MMP	72/60	
MMP-16	MT3-MMP	64/53	ProMMP-2
MMP-17	MT4-MMP	57/53	
MMP-20	Enamelysin	54/22	Dental enamel organic matrix
MMP-23	CA-MMP		
MMP-24	MT5-MMP		
MMP-25	MT6-MMP		
MMP-26	Matrilysin-2		

## MAST CELLS

- a. Connective tissue mast cells / T mast cells
  1. Found around blood vessels
  2. Contains neutral proteases (tryptase and chymotryptic proteinase)
- b. Mucosal mast cells / TC mast cells
  1. Found in mucosal tissues
  2. Contains tryptase

## MICROSURGERY

Acc. to Carranza

Micro surgical loupe

1. Simple loupes
2. Compound loupes
3. Prism or telescopic loupes

## MUCO GINGIVAL PROBLEMS

### 1) Deformity and condition around the teeth

- a) Gingival or soft tissue recession
  1. Facial or lingual surface
  2. Interproximal surface
- b) Lack of keratinized gingiva
- c) Decreased vestibular depth
- d) Aberrant frenum or muscle position
- e) Gingival excess
  - 1) Pseudo pocket
  - 2) Inconsistent gingival margin
  - 3) Excessive gingival display
  - 4) Gingival enlargement
  - 5) Abnormal colour

II) Deformity and contour on the edentulous ridge

- a) Vertical or horizontal ridge deficiency
- b) Lack of gingiva or keratinized tissue
- c) Gingival or soft tissue enlargement
- d) Aberrant frenum or muscle position
- e) Decreased vestibular depth
- f) Abnormal color

NSAIDs

I) According to K.D.Tripathi

1. Drugs which have potent analgesics and anti-inflammatory action.
2. Drugs which have good analgesic but poor anti-inflammatory action.

II) Analgesic and anti inflammatory

- Salicylates - Aspirin, salicylamide, diflunisal pyrazolone derivatives- Phenyl butazone
- Indole derivatives - Indomethacin, sulindac
- Propionic acid derivatives - Ibuprofen, naproxen, ketoprofen, flurbiprofen
- Anthranilic acid derivatives - Mefenemic acid
- Aryl acetic acid derivatives - Diclofenac, tolmetin
- Oxicam derivatives- Piroxicam, meloxicam
- Pyrrolo-pyrrole derivatives- Ketorolac
- Sulfonanilide - Nimuselide
- Alkanones - Nabumetone

III) Analgesic but poor anti inflammatory action

- Para amino phenol derivative - Paracetamol(acetaminophen)
- Pyrazolone derivatives - Metamizole
- Benzoxazocine derivatives - Nefopam

**IV) According to Goodman and Gillman:****1. Non selective Cox inhibitor**

- Salicylates- Aspirin, salicylamide
- Indole and indene acetic acid derivatives - Indomethacin
- Aryl proprionic acid derivatives - Ibuprofen, naproxen, ketoprofen, flurbiprofen
- Anthranilic acid derivatives - Mefenemic acid
- Hetero aryl acetic acid derivatives - Diclofenac, tolmetin
- Enolic acid (oxicam derivatives) - Piroxicam, meloxicam
- Alkanones - Nebumatone
- Para amino phenol derivative-Paracetamol(acetaminophen)

**2. Selective Cox-2 inhibitor**

- Di aryl substituted furanones - Rofecoxib
- Di aryl substituted pyrazoles - Celecoxib
- Indole acetic acids - Etodolac
- Sulfonanilides - Nimesulide

**NEEDLES** (According to Edward Cohen; Atlas Cosmetic and Reconstructive Periodontal Surgery; 1st Edition)

**I. Based on needle shape:**

- a) Straight
- b) 3/8 Circle 135°
- c) 1/2 Circle 180°
- d) 5/8 Circle 225°

**II. Based on point design:**

- a) Round bodied
- b) Curved cutting
- c) Reverse cutting
- d) Reverse cutting prime
- e) Taper cutting



- f) Micro point reverse cutting
- g) Micro point spatula cutting
- h) CSU spatula
- i) SBR spatula

**III. Based on end type:**

- a) Rolled end
- b) Drilled end
- c) Regular eye
- d) Spring eye
- e) Spring double eyes

**IV. Based on curvature:**

- a) Straight needle
- b) Curved needle

**V. Based on presence or absence of eye:**

- a) Eyed needle
- b) Swaged needle (eyeless needle)

**NEUTROPHIL DEFECTS**

Neutrophils disorders may be:

**I. QUANTITATIVE**

a) **Neutrophilia** - Increased count of circulating neutrophils

- i. Acute infections
- ii. Non infectious inflammation
- iii. Acute hemorrhage
- iv. Surgical trauma
- v. Malignant neoplasms
- vi. Metabolic
- vii. Poisoning

- viii. Physiologic neutrophilia
- ix. Other causes: In association with convulsions and paroxysmal tachycardia, Cushing's disease. Acute or chronic administration of corticosteroids causes neutrophilia.

Neutrophilia may be present without an identifiable cause; in this case, it is known as chronic idiopathic neutrophilia.

**b) Neutropenia:** Decreased count

Based on the absolute neutrophil count (ANC), measured in cells per microliter of blood:

- Mild neutropenia ( $1000 < \text{ANC} < 1500$ ) — minimal risk of infection
- Moderate neutropenia ( $500 < \text{ANC} < 1000$ ) — moderate risk of infection
- Severe neutropenia ( $\text{ANC} < 500$ ) — severe risk of infection

## II. QUALITATIVE

There have been disorders described in the neutrophil function of adherence, chemotaxis, phagocytosis, and bactericidal activity.

Table 7:

Adherence	Chemotaxis	Phagocytosis & Bactericidal Activity.
Leukocyte adhesion deficiency disease	Actin dysfunction syndrome	Actin dysfunction syndrome
	Chediak-Higashi syndrome	Diabetes mellitus
	Diabetes mellitus	Rheumatoid arthritis
	Down's syndrome	Systemic lupus erythematosus
	Hyperimmunoglobulinemia	Chronic granulomatous disease,
	Icthyosis	Myeloperoxidase deficiency
	Crohn's syndrome	
	Ulcerative colitis	
	Mannosidosis	
	Malnutrition	
	Otitis media	
	Otitis and diarrhea syndrome	
	Felty's syndrome	
	Rheumatoid arthritis	
	Pegler Huet anomaly	
	Chronic pulmonary disease	

**OCCCLUSION** (According to Gurkeerat Singh, Textbook of Orthodontics, 2nd Edition)

**I. Depending on the type of occlusion**

- a) Normal occlusion
- b) Ideal occlusion
- c) Physiologic occlusion
- d) Traumatic occlusion
- e) Therapeutic occlusion

II. According to E H Angle (1880) based on 1<sup>st</sup> permanent molar relation it is classified into

- a) Class I
- b) Class II
  - i. Div I subdivision
  - ii. Div II subdivision
- c) Class III - Pseudoclass III
  - i. Subdivision
  - ii. Skeletal class III

III. Simon's classification used to describe malocclusion in antero-posterior direction

- a) Protraction
- b) Retraction
- c) Contraction
- d) Distraction

IV. Baume's classification

- a) Flush terminal plane
- b) Distal step terminal plane
- c) Mesial step terminal plane

V. Ballard's classification based on incisor relation, occlusion is classified into

- a) Class I
- b) Class II
- c) Class III

**PERI IMPLANTITIS CLASSIFICATION**

I. Stuart J Froum, Paul S Rosen (2012)

- a) **Early** - Probing depth  $\geq 4$  mm, bleeding on probing and suppuration, bone loss  $< 25\%$  of implant length.

b) Moderate - Probing depth  $\geq 6$  mm, bleeding on probing and suppuration, bone loss 25%-50% of implant length.

c) Advanced - Probing depth  $\geq 8$  mm, bleeding on probing and suppuration, bone loss  $>50\%$  of implant length.

II. Class 1: Slight horizontal bone loss with minimal peri-implant defects

Class 2: Moderate horizontal bone loss with isolated vertical defects

Class 3: Moderate to advanced horizontal bone loss with broad, circular bony defects

Class 4: Advanced horizontal bone loss with broad, circumferential vertical defects, as well as loss of the oral and/or vestibular bony wall.

**PROGNOSIS**

**I. Hirschfeld and Wasserman - 1978**

- a) Favorable
- b) Questionable - deep probing depths, furcation involvement, bone loss, and mobility.

**II. Becker et al - 1984**

- a) Good
- b) Questionable - 50% bone loss, 6 to 8 mm probing depth, Class 2 furcation, or anatomic variables such as a deep palatal groove on the maxillary incisors or a mesial furcation involvement of the maxillary first premolar
- c) Hopeless - more than 75% bone loss, more than 8 mm probing depth, Class 3 furcation involvement, Class 3 mobility, poor crown-root ratio, unfavorable root proximity, or repeated periodontal abscess formation.

**III. Mc Guire - 1991**

- a) Very good - Less than 25% attachment loss
- b) Good - 25% attachment loss and /or class I furcation involvement
- c) Fair - 25- 50 % attachment loss and /or easily accessible class II furcation involvement.

- d) Poor – 50 - 75 % attachment loss and /or class II inaccessible furcation involvement, class III furcation involvement, class II mobility
- e) Hopeless – > 75% attachment loss, class III mobility.

**IV. McGuire and Nunn-1996**

- a) Good prognosis (one or more of the following) Control of etiologic factors and adequate periodontal support as measured clinically and radiographically to ensure that the tooth would be relatively easy to maintain by the patient and clinician, assuming proper maintenance.
- b) Fair prognosis (one or more of the following) 25% attachment loss as measured clinically and radiographically and /or class I furcation involvement. The location and depth of furcation would allow proper maintenance with good patient compliance.
- c) Questionable prognosis (one or more of the following): 50 % attachment loss with easily class II furcations. The location and depth of furcations may limit proper maintenance.
- d) Poor prognosis (one or more of the following): Greater than 50% attachment loss resulting in poor crown- root ratio; poor root form; class II furcations not easily accessible to maintenance of class III furcations; 2+ mobility or greater; significant root proximity.
- e) Hopeless prognosis: Inadequate attachments to maintain the tooth; extraction performed or suggested

**V. Kwok G & Caton J, – 2007 (With periodontal treatment and maintenance)**

Favourable .....	Likely	] Periodontal stability
Questionable .....	May be	
Unfavourable .....	Unlikely	
Hopeless .....	Extraction needed	

VI. Nicola U. Zitzmann 2010  
 Table 8: Prognostic Assessment of Potential Abutment Tooth or Dental Implant

Prognosis factors	Good	Questionable	Hopeless
<b>Periodontal</b>	PPD ≤ 3 mm, No BOP-, PAL loss ≤ 25%, FI degree ≤ I	Residual PPD ≤ 6 mm, BOP+, PAL loss approximately ≤ 50%, FI degree ≤ II or III and root proximity	Insufficient residual attachment
<b>Endodontics</b>	No clinical signs and absence of or decreasing radiolucency	No clinical signs and persisting radiolucency	Symptomatic situation and radiolucency, no further treatment feasible.
<b>Implants</b>	Absence of BOP, suppuration and bone loss	BOP with / without bone loss	Mobility
<b>Prosthetic</b>	Sufficient residual tooth substance, adequate retention and resistance forms ideally (4 mm wall height with 15-to 20-degree of convergence angle, 1.5 to 2 mm ferrule )	Reduced retention and resistance forms (less than 3 mm wall heights and/ or more than 25 degree convergence angle)	Insufficient residual tooth substance (less than 1.5 mm circular ferrule), no crown lengthening or extrusion feasible

Table 9:

Based on involvement of supporting periodontal tissue		Periodontal pocket
<b>Gingival (Pseudo pocket)</b>		There is loss of supporting periodontal tissues.
No loss of supporting tissue. Pocket is formed due to gingival enlargement + and coronal migration of the gingival margin.		
<b>Based on surfaces involved</b>		
<b>Simple</b>	<b>Compound</b>	<b>Complex/Spiral</b>
Pocket involves only one surface	Pocket involves more than one surface	Pocket starts on one surface, follows a tortuous course to involve another surface of the tooth
<b>Based on relationship with bone</b>		
<b>Suprabony (supracrestal/ supraalveolar)</b>		<b>Infrabony (subcrestal/ intraalveolar)</b>
Base of the pocket is coronal to the crest of the alveolar bone		Base of the pocket is apical to the crest of the alveolar bone
Horizontal bone loss		Angular bone loss
Interproximally, transeptal fibers are arranged horizontally		Interproximally, transeptal fibers are arranged obliquely
Facially and lingually, periodontal ligament fibers follow a horizontal course		Facially and lingually, periodontal ligament fibers follow an angular course
<b>Based on the nature of soft tissue wall of the pocket-</b>		
a) Oedematous pocket		
b) Fibrotic pocket		
<b>Based on the disease activity</b>		
a) Active pocket		
b) Inactive pocket		

CLASSIFIC

I. List

Clas

a) M

b) S

c) E

i

II. Pa

a)

b)

a)



## CLASSIFICATION OF DENTAL PLAQUE

### I. Listgarten (1976)

#### Classification according to location

- a) Marginal plaque
- b) Supra gingival plaque
- c) Sub gingival plaque
  - i. Tooth associated
  - ii. Tissue associated

### II. Pavel Godoroja and Olga Dul Ghieru 2004 (2)

- a) Supragingival
- b) Subgingival.
- a) **Supra gingival plaque**

Supra gingival plaque at and above the dento gingival junction is most commonly found at:

- Gingival third of the crown of the tooth
- Inter proximal areas
- Pits and fissures and also on other such surface with irregularities.

- b) **Sub gingival plaque**

Sub gingival plaque below the dento gingival junction is usually divided into:

- i. Tooth adherent zone
- ii. Epithelial adherent zone
- iii. Non adherent zone

CLASSIFICATION OF PERIODONTAL DISEASES

I) Periodontal disease: A simple version of 1999 International Workshop

Table 10:

Chronic periodontitis	Localized (<30%) Generalised (>30%)
Aggressive periodontitis	Localized Generalized
Periodontitis as a manifestation of systemic disease	<ul style="list-style-type: none"> <li>• Associated with genetic disorders</li> <li>• Not otherwise specified</li> </ul>
Necrotising periodontal disease	<ul style="list-style-type: none"> <li>• Necrotising ulcerative gingivitis</li> <li>• Necrotising ulcerative periodontitis</li> </ul>
Abscess of periodontium	<ul style="list-style-type: none"> <li>• Gingival abscess</li> <li>• Periodontal abscess</li> <li>• Pericoronal abscess</li> </ul>
Periodontal lesions associated with endodontic lesions	Combined periodontal endodontal lesions
Developmental or acquired deformities or conditions	<ul style="list-style-type: none"> <li>• Localised tooth related factors that modify or predispose to plaque-induced gingival disease / periodontitis</li> </ul>
	<ul style="list-style-type: none"> <li>• Mucogingival deformities and conditions around the tooth</li> </ul>
	<ul style="list-style-type: none"> <li>• Mucogingival deformities and conditions on edentulous ridge</li> </ul>
	<ul style="list-style-type: none"> <li>• Occlusal trauma</li> </ul>

II. AAP World Workshop in Clinical Periodontics, 1989

- a) Adult periodontitis
- b) Early onset periodontitis
- c) Periodontitis associated with systemic disease
- d) Necrotising ulcerative periodontitis
- e) Refractory periodontitis

**III. European Workshop in Periodontitis, 1993**

- a) Adult periodontitis
- b) Early onset periodontitis
- c) Necrotising periodontitis

**PERIODONTAL DRESSINGS**

- a) Two most widely used type of dressing materials:
  - i. Zinc oxide eugenol
  - ii. Zinc oxide non-eugenol dressings
  - iii. In addition,
    - Cyanoacrylates
    - Tissue conditioners.
- b) Periodontal dressings that contain antimicrobial agents,
  - i. Photo curing periodontal dressing materials,
  - ii. Collagen dressings,
  - iii. Intraoral adhesive bandages
  - iv. Wax packs.

**PREMALIGNANT LESIONS AND CONDITIONS**

**Premalignant Conditions:**

- a) Erosive lichen planus
- b) Actinic or solar keratosis
- c) Sideropenic dysphagia
- d) Bowen's disease or carcinoma in situ
- e) Syphilitic glossitis
- f) DLE
- g) Dyskeratosis congenita
- h) Hyperplastic candidiasis
- i) Peutz-Jegher's syndrome

- j) Oral submucous fibrosis
- k) Oral lichen planus

#### Premalignant Lesions:

- a. Leukoplakia
- b. Erythroplakia

#### PIGMENTATION

##### Endogenous Causes:

1. **Melanin is increased in the following systemic condition:**
  - Addison's disease (adrenal dysfunction)
  - Peutz Jeghers syndrome (intestinal polyposis)
  - Albright's syndrome (fibrous dysplasia)
  - Von Recklinghausen's disease (neurofibromatosis)
2. **Bilirubin (Jaundice)**
3. **Iron (hemochromatosis)**

##### Exogenous Causes:

1. Metallic pigmentation due to bismuth, arsenic, lead and silver.
2. Tobacco
3. Coloring agents in food and lozenges
4. Amalgam tattoo or implantation of amalgam into the gingiva

#### PAIN

##### I) Classification

###### a) Based on duration

1. Acute pain
2. Chronic pain
  - a) Chronic non cancer pain
  - b) Chronic cancer pain
  - c) Chronic episodic pain

b) **Based on location / origin**

1. Visceral pain
2. Somatic pain
3. Referred pain
4. Phantom limb pain
5. Emotional or psychogenic pain
6. Persistent pain
7. Abnormal pain

c) **Based on inflammation**

1. Inflammatory pain
2. Non inflammatory pain

d) **Based on intensity**

1. Mild pain
2. Moderate pain
3. Severe pain

e) **Based on etiology**

1. Nociceptive pain
  - i. Somatic pain
  - ii. Visceral pain
2. Neuropathic pain
  - i. Peripheral neuropathic pain
  - ii. Central neuropathic pain

**PLAQUE HYPOTHESIS**

**Carranza's Clinical Periodontology, 11<sup>th</sup> Edition**

- a) Traditional non-specific plaque hypothesis – Loesche 1976
- b) Specific plaque hypothesis – Locche – 1976
- c) Updated nonspecific plaque hypothesis - 1986
- d) Ecologic plaque hypothesis – Marsch 1994
- e) Key stone plaque hypothesis – Hajshingalis 2012

## FREE RADICALS

## 1. According Barry Halliwell, 2001

## a) Reactive Oxygen Species (ROS)

## 1. Radicals

Superoxide ( $O_2^-$ )

Hydroxyl (OH)

Peroxyl ( $RO_2$ )Alkoyl ( $HO_2$ )

## 2. Non Radicals

Hydrogen peroxide ( $H_2O_2$ )

Hypochlorous acid (HOCl)

Ozone ( $O_3$ )Singlet oxygen ( $^1O_2$ )

Peroxynitrite (ONOO)

## b) Reactive Nitrogen Species

## 1. Radicals

Nitric oxide (NO)

Nitrogen dioxide ( $NO_2$ )

## 2. Non radicals

Peroxynitrite (ONOO)

Alkyl peroxynitrite (ROONO)

Dinitrogen trioxide ( $N_2O_3$ )Dinitrogen tetraoxide ( $N_2O_4$ )Nitrous acid ( $HNO_2$ )Nitronium anion ( $NO_2^-$ )Nitroxyl anion ( $NO_2^-$ )Nitroxyl cation ( $NO^+$ )Nitryl chloride ( $NO_2Cl$ )

ALVEOLAR RIDGE DEFECTS

I. Selbert's classification 1983

Class I: Buccolingual loss of tissue with normal apicocoronal ridge height

Class II: Apicocoronal loss of tissue with normal buccolingual ridge width

Class III: Combination-type defects (loss of both height and width)

II. Allen nomenclature 1985

Class A: Apicocoronal loss of tissue with normal buccolingual ridge width

Class B: Buccolingual loss of tissue with normal apicocoronal ridge height

Class C: Combination-type defects (loss of both height and width)

III. Studer Zellweger et al 1996 published the first semi-quantitative classification of defects of the alveolar process according to the perceived need to reconstruct the hard and soft tissues

a. Class I: > 3 mm

b. Class 2: 3–6 mm

c. Class 3: > 6 mm

IV. The Cologne Classification of Alveolar Ridge Defects 2013 uses three-part codes to describe the effect of the alveolar ridge as comprehensively as possible with a view to existing therapeutic options:

**Part 1:** Orientation of the defect

H: Horizontal

V: Vertical

C: Combined

S (or +S): Sinus area

**Part 2:** Reconstruction needs associated with the defect

1: Low: < 4 mm

2: Medium: 4-8 mm

3: High: > 8 mm

**Part 3:** Relation of augmentation and defect region

i: Internal, inside the contour

e: External, outside the ridge contour

### RISK MODELS

- I. Periodontal risk calculator (PRC) by Page et al., 2002
- II. Periodontal risk assessment (PRA) hexagonal risk diagram model by Lang & Tonetti et al., 2003
- III. MRD/ Multifactorial risk diagram by Renvert & Persson et al.
- IV. HRD/ Hexagonal risk diagram by Persson et al.

### SCAFFOLDS

#### I. Depending on the use

- a) Naturally derived (e.g., Collagen, glycosaminoglycan, chitosan, alginate)
- b) Acellular tissue matrices (e.g., acellular dermal matrix)
- c) Synthetic polymers (e.g., Polyglycolic acid, polylactic acid, and poly lactic-co-glycolic acid)

#### II. According Shalu Bhatla 2011

##### 1. Nonresorbable

Expanded polytetrafluoroethylene (ePTFE)

Ceramic

Titanium mesh

##### 2. Resorbable

a) Alpha-hydroxy acids

Polyglycolic acid

Polylactic acid

Copolymer of poly lactic-co-glycolic acid



b) Amino acid base polymer

Collagen like proteins

Elastin like proteins

c) Natural products

Collagen

Hyaluronon

Chitosan

Gelatin

Fibrin

Alginate

**3. Synthetic hydrogels**

Polyethylene glycol

Polyethylene oxide

**4. Matrix extracts**

Matrigold

**CLASSIFICATION OF SPLINTS**

**I. According to the period of stabilization:**

**A. Temporary Stabilization: Worn for less than 6 months.**

**i. Removable**

Occlusal splint with wire

Hawley appliance with arch wire

**ii. Fixed**

**1. Intracoronal**

Amalgam

Amalgam and wire

Amalgam, wire and resin

Composite resin and wire

**2. Extracoronal**

Stainless steel wire with resins

Wire and resin with acid etching

Enamel etching and composite resin

Orthodontic soldered bands, brackets and wire

**B. Provisional Splinting:** To be used for months up to several years

E.g. Acrylic splints, metal band etc.

**C. Permanent Splints:** Used indefinitely

i. Removable/Fixed

ii. Extra/Intracoronal

iii. Full/Partial veneer crowns soldered together.

iv. Inlay/Onlay soldered together.

**II. According to the type of material:**

Bonded composite resin splint

Braided wire splint

A – Splints.

**III. According to the location on the tooth:****I. Intracoronal**

Com

Inlays

Onlays

**II. Extracoronal**

Night guard

Tooth bonded plastic and welded bands

**IV. Goldman, Cohen and Chacker Classification:****A. Temporary Splints**

i. Extra coronal type

Wire ligation

Orthodontic bands

Removable acrylic appliances

Removable cast appliances

Ultraviolet-light-polymerizing bonding materials

ii. **Intracoronal type**

Wire and acrylic

Wire and amalgam

Wire, amalgam, and acrylic

Cast chrome-cobalt alloy bars with acrylic, or both.

**B. Provisional Splints**

All acrylic

Adapted metal band and acrylic

v. **Ross, Weisgold and Wright Classification:**

**A. Temporary stabilization**

Removable extra coronal splints

Fixed extra coronal splints

Intracoronal splints

Etched metal resin-bonded splints

**B. Provisional stabilization**

Acrylic splints

Metal-band-and-acrylic splints

**C. Long-term stabilization**

Removable splints

Fixed splints and

Combinations

**SUTURES** (According to Edward Cohen; Atlas Cosmetic and Reconstructive Periodontal Surgery; 1st Edition)**I. According to the source:****a) Natural****i. Absorbable**

1. Catgut
2. Chromic catgut
3. Collagen
4. Fascia lata
5. Kangaroo tendon
6. Beef tendon
7. Cargile membrane

**ii. Non absorbable**

1. Silk
2. Silk worm gut
3. Linen
4. Cotton
5. Ramie
6. Horse hair

**b) Synthetic****i. Absorbable**

1. Polyglycolic acid
2. Polyglactic acid
3. Polyglactin 9/10 (Vicryl)
4. Polydioxanone
5. Polyglecaprone 25

**ii. Non absorbable**

1. Nylon/ polyamide

2. Polypropylene
3. Polyester
4. Polyethelene
5. Polybutester
6. Polyvinylidene fluoride/PVDF sutures

**iii. Metallic**

1. Stainless steel
2. Tantalum
3. Gold
4. Silver
5. Aluminium

**II. According to structure:**

- a) Monofilament
- b) Multifilament

**III. According to coating:**

- a) Coated
- b) Uncoated

**IV. According to fate:**

- a) Absorbable (degradation in < 60 days)
- b) Nonabsorbable

**SUTURING TECHNIQUES** (According to Edward Cohen; Atlas Cosmetic & Reconstructive Periodontal Surgery; 1st Edition)

**I. Interrupted**

- a) Figure of eight
- b) Needles
  - Circumferential direct loop
- c) Mattress-vertical or horizontal
- d) Intrapapillary

## II. Continuous

- a) Papillary sling
- b) Vertical mattress
- c) Locking

## STAINS

### I. Stains may be intrinsic or extrinsic.

#### a) Causes of intrinsic stains:

1. Fluorosis
2. Non vitality of tooth
3. Tetracycline
4. Internal root resorption (Pink tooth of mummery)
5. Porphyria
6. Amelogenesis imperfecta

#### b) Causes of extrinsic stains:

1. Tobacco
2. Food chromogens
3. Chlorhexidine mouthwash
4. Chromogenic bacteria

### II. Based on the color stains can be classified as:

a) **Brown stain:** These are tannin stains usually found on the buccal surface of the maxillary molars and on the lingual surface of the mandibular incisors. It is seen in individuals who do not brush sufficiently or who use a dentifrice with inadequate cleansing action.

b) **Black stain:** These are seen as a thin black line on the facial and lingual surfaces of the teeth near the gingival margin. This is usually seen in women with excellent oral hygiene. The chromogenic bacteria *Actinomyces* and *Prevotella melaninogenica* have been implicated. This stain is firmly attached and tends to recur after removal.

- c) **Green stain:** Usually seen on the facial surface of maxillary anteriors, more commonly in boys. It has been attributed to bacteria and fungi such as *Pencillium* and *Aspergillus*.
- d) **Orange stain:** It may occur on both facial and lingual surfaces of anterior teeth. *Serratia marcescens* and *Flavobacterium-lutescens* are the organisms implicated.

## TOOTH WEAR

### I. Smith and Knight Tooth wear index

**Score 0** – No loss of enamel surface characteristics on buccal/lingual/occlusal/incisal surfaces.

No loss of contour on cervical surface.

**Score 1** – Loss of enamel surface characteristics on buccal/lingual/occlusal/incisal surfaces. Minimal loss of contour on cervical surface.

**Score 2** – Loss of enamel exposing dentin for less than 1/3<sup>rd</sup> of buccal/lingual/occlusal surfaces.

Loss of enamel just exposing dentine on incisal surface. Defect less than 1 mm deep on cervical surface.

**Score 3** – Loss of enamel exposing dentin for more than 1/3<sup>rd</sup> of buccal/lingual/occlusal surfaces. Loss of enamel and substantial loss of dentine on incisal surface. Defect less than 1-2 mm deep cervical surface.

**Score 4** – Complete enamel loss - pulp exposure - secondary dentin exposure on buccal/lingual/occlusal surfaces. Pulp exposure or exposure of secondary dentine on incisal surface Defect more than 2 mm deep - pulp exposure - secondary dentine exposure on cervical surface.

### II. Simplified scoring criteria for tooth wear index (Bardsley PF, Taylor S, Milosevic A, 2004)

**Score 0** – No wear into dentine.

**Score 1** – Dentine just visible (including cupping) or dentine exposed.

**Score 2** – Dentine exposure greater than 1/3<sup>rd</sup> of surface.

**Score 3** – Exposure of pulp or secondary dentine.

**ATTRITION**

**Attrition index** (Seligman DA, Pullinger AG, Solberg WK)

**Score 0** – No wear

**Score 1** – Minimal wear

**Score 2** – Noticable flattening parallel to occlusal planes

**Score 3** – Flattening of cusps or grooves

**Score 4** – Total loss of contour and /or dentin exposure when identifiable

**EROSION**a) **Classification of dental erosion (Eccles JD)**

Class I – Superficial lesions involving enamel only

Class II – Localized lesions involving dentin for less than one third of the surface

Class III – Generalized lesions involving dentin for more than one third of the surfaces

- Facial surfaces
- Lingual and palatal surfaces
- Incisal and occlusal surfaces
- Severe multi surface involvement

b) **Erosion - Erosion index** (Eccles JD, Lussi A, 1996)

Facial

**Score 0** – No erosion. Surface with a smooth, silky glazed appearance, possible absence of developmental ridges

**Score 1** – Loss of surface enamel. Intact enamel cervical to the erosive lesion; concavity enamel where breadth clearly exceeds depth, thus distinguishing it from tooth brush abrasion.

Undulating borders of the lesion are possible and dentin is not involved.

**Score 2** – Involvement of dentin for less than half of tooth surface

**Score 3** – Involvement of dentin for more than half of tooth surface



**Occlusal/lingual**

No erosion.

**Score 0** – Surface with a smooth, silky glazed appearance, possible absence of developmental ridges

**Score 1** – Slight erosion, rounded cusps and edges of restorations rising above the level of adjacent tooth surface, grooves on occlusal aspects. Loss of surface enamel. Dentin is not involved.

**Score 2** – Severe erosions, more pronounced signs than in grade 1. Dentin is involved.

**Classification of TIMPs:**

**According to Mitsuya Murashige 1996**

- a) **TIMP-1** is a glycoprotein with an apparent molecular mass of 28.5 kDa which forms a complex of 1:1 stoichiometry with activated interstitial collagenase, activated stromelysin, and 92 kDa type IV collagenase.
- b) **TIMP-2** is a nonglycosylated protein with a molecular mass of 21kDa which forms a complex with both latent and activated 72 kDa type IV collagenase.
- c) **TIMP-3** is a recently identified TIMP that shows approximately 37% and 42% similarity to TIMP-1 and TIMP-2 respectively.

**TOLL LIKE RECEPTORS**

Receptor	Cell Type
TLR1	Ubiquitous
TLR2	DCs, PMLs, and monocytes
TLR3	DC and NK cells, up regulated on epithelial and endothelial cells
TLR4	Macrophages, PMLs, DCs, ECs, but not on lymphocytes
TLR5	Monocytes, immature DCs, epithelial, NK, and T cells
TLR6	High expression in B cells, lower on monocytes and NK cells
TLR7	B cells, plasmacytoid precursor DCs
TLR8	Monocytes, low in NK cells and T cells
TLR9	Plasmacytoid precursor DCs, B cells, macrophages, PMLs, NK cells, and microglial cells
TLR10	B cells, plasmacytoid precursor DCs
TLR11	Not determined

## TONGUE THRUSTING

### I. Branner and Hart Classification

- a) Type 1: Non deforming tongue thrusting
- b) Type 2: Deforming anterior tongue thrusting
  - Sub group 1: anterior open bite
  - Sub group 2: anterior proclination
  - Sub group 3: posterior cross bite
- c) Type 3: Deforming lateral tongue thrust
  - Sub group 1: posterior open bite
  - Sub group 2: posterior cross bite
  - Sub group 3: deep bite
- d) Type 4: Deforming anterior and lateral tongue thrust
  - Sub group 1: anterior and posterior open bite
  - Sub group 2: proclination of anterior teeth
  - Sub group 3: posterior cross bite

### II. Based on area of thrusting

Ref: **Shobha Tandon**

- a) Simple tongue thrusting
- b) Lateral tongue thrusting
- c) Complex tongue thrusting

### III. Classification (Ref: Shobha Tandon )

- a) Physiologic tongue thrusting
- b) Habitual tongue thrusting
- c) Anatomical tongue thrusting
- d) Functional tongue thrusting

**TMJ DISORDER** (According to Ken Olson et. al)

**I. TMJ classification**

1. Capsulitis / synovitis
2. Capsular fibrosis
3. Hypermobility
4. Articular disc displacement
  - with reduction
  - without reduction
5. Post surgical TMJ

**II. Etiological classification**

1. Developmental disorders of TMJ
2. Degenerative joint disease
3. Inflammatory disorder of joints
4. Traumatic disorder of TMJ
5. Metabolic disorder
6. Neoplastic disorder
7. TMJ disorder syndrome /myofascial pain dysfunction syndrome

**III. Radiographic classification**

- a) Developmental abnormalities
    1. Condylar hyperplasia
    2. Condylar hypoplasia
    3. Bifid condyle
  - b) Soft tissue abnormality
    1. Internal derangement
    2. Remodelling and arthritic condition
  - c) Trauma
    1. Effusion
    2. Dislocation
    3. Fracture
    4. Ankylosis
-

- d) Tumors
  - 1. Benign
  - 2. Malignant

### TOBACCO FORMS

Soben Peter - Preventive and Community Dentistry, 3<sup>rd</sup> Edition

- 1. Smoked tobacco
- 2. Smokeless tobacco

#### I. Smoked Tobacco:

- a) Beedi
- b) Chillium
- c) Chutta
- d) Cigarettes
- e) Dhumti
- f) Gudaku
- g) Hookah
- h) Hookli

#### II. Smokeless Tobacco:

- a) Khaini
- b) Manipuri tobacco
- c) Mawa
- d) Mishri/masheri
- e) Paan
- f) Snuff
- g) Zarda

Theories Related to Mineralization of Calculus Sahithya Reddy;  
Essentials of Periodontology; 1st Edition Pg No: 213-216

- 1. **Booster/Precipitation Theory:** Loss of carbon dioxide and formation of ammonia leads to increase in the pH which leads to the precipitation of calcium phosphate salts.

2. **Epitactic/Nucleation Concept:** The carbohydrate – protein complexes may initiate calcification by removing calcium from the saliva and binding with it to form nuclei that induce deposition of minerals. Seeding agents induce small foci of calcification that enlarges and unites together to form calcified mass.
3. **Inhibition Theory:** Calcification occurring only at specific site is because of the existence of an inhibiting mechanism at non-calcifying sites. Where calcification occurs, the inhibitor is apparently altered or removed. Inhibiting substance is thought to be pyrophosphate and among the controlling mechanism is the enzyme alkaline pyrophosphatase, which can hydrolyze the pyrophosphate to phosphate. The pyrophosphate inhibits calcification by preventing the initial nucleus from growing, possibly by “poisoning” the growth centers of the crystal.
4. **Transformation Theory:** Amorphous non-crystalline deposits and brushite can be transformed to octacalcium phosphate and then to hydroxyapatite.

## CLASSIFICATION OF TOOTH BRUSHES

### I. ADA Specifications of Tooth Brush (ADA number – 119)

Brush length: 1-1.25 inches

Brush width: 5/16-3/8 inches

2-4 Rows

5-12 Tufts per row

80-86 bristles/tuft

### II. Based on bristle hardness

a) Soft brush: 0.007 inches (0.2 mm)

b) Medium brush: 0.012 inches (0.3 mm)

c) Hard brush: 0.014 inches (0.4 mm)

### III. Based on nature of bristles

a) Natural: Hog

b) Artificial: Nylon which is uniform in size and elasticity, resistant to fracture and doesn't get contaminated.

## TRAUMA FROM OCCLUSION

- I. Based on the onset
  - a) Acute
  - b) Chronic
- II. Based on etiology
  - a) Primary trauma from occlusion
  - b) Secondary trauma from occlusion

## VIRUSES

### I. DNA Viruses:

#### A. Enveloped

1. Hepadna viridae
2. Herpes viridae
3. Pox viridae

#### B. Non Enveloped:

1. Parvo viridae
2. Papova viridae
3. Adeno viridae

### II. RNA Viruses:

#### A. Enveloped:

1. Togaa viridae
  2. Flavi viridae
  3. Corona viridae
  4. Rhabdo viridae
  5. Filo viridae
  6. Paramyxo viridae
  7. Orthomyxo viridae
  8. Bunya viridae
  9. Arena viridae
  10. Retro viridae
-

B. Non Enveloped:

1. Picorona viridae
2. Calci viridae
3. Reo viridae

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-



# CLASSIFICATIONS IN PERIODONTICS - AN UPDATE

## Key Features

- The book illustrates and explains several classifications of various conditions, diseases, materials, agents etc., collected and compiled from various standard textbooks and articles.
- Classifications are presented in an easy language and in extensive precise manner for clear understanding of the subject.
- Useful for both, undergraduate and post graduate dental students.
- Guides the practicing dentists in the diagnosis and treatment planning.
- Helps in public health planning and targeting of therapy
- Emphasis is given on 'extensive categorization' of each and every aspect of periodontology.
- Classifications are arranged in alphabetical order for easy grasping and are provided with references.

## About the Author

Dr. Suchetha A. Professor, Head and Ph.D Guide, Department of Periodontology, D.A.P.M.R.V Dental College, Bangalore, India. Dr. Suchetha has a teaching experience of about 20 years, guided several research projects at both under-graduate and post graduate levels. This book covers 'Classification of various diseases and conditions' in the field of Periodontology which helps the students in thorough understanding of the subject.

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