Classifications in Periodontics
- A Compilation

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Dr. Srishti Shankar

Preface

A classification scheme is necessary for clinicians to properly diagnose and treat patients as well as for scientists to investigate the etiology, pathogenesis, natural history, and treatment of diseases and conditions. The concept of classification systems is considered as uninteresting by many, but it provides us with a framework to come to a diagnosis.

The intricacy of periodontal diseases can be understood by classifying various diseases which can be considered as old as mankind. There are several pieces of evidence in history shedding light on the evaluation of pathology and therapeutics - such as the anatomical findings from well preserved skeletons, details observed in Egyptian mummies, various instruments and equipment recovered during archeological surveys and excerpts from manuscripts. Almost all early historical records that involve dental topics have several chapters dealing with periodontal disease and the need for treatment.

Three dominant paradigms that reflected the understanding of the nature of periodontal diseases were noticed during the evolution and classification of periodontal diseases - Clinical characteristics paradigm(1870-1920) - based on clinical features of the diseases, Classical pathology paradigm(1920-1970) - based on concepts of classical pathology and Infection / host response paradigm(1970-present) - based on infectious etiology of diseases.

Classification systems in the modern era represent a blend of all three paradigms since there is a certain amount of gravity to some of the earliest thoughts about the nature of periodontal diseases. History reveals that people in the past opposed the modification of these entities. They were adamant to accept a particular classification in spite of it having many flaws. But in the true sense, they need to be periodically modified and updated based on modern thinking and concepts.

Therefore this book serves as a compilation of the various classifications present in Periodontics to guide the undergraduate and postgraduate students in dentistry as well as healthcare professionals to arrive at an apt diagnosis. The authors and contributors have worked hard in compiling the classifications, sincerely hoping that it would benefit dentists across all age groups.

Contents

Introduction

1 Chapter

A classification scheme is necessary for clinicians to properly diagnose and treat patients as well as for scientists to investigate the etiology, pathogenesis, natural history, and treatment of diseases and conditions.

Need for a classification system:

The concept of classification systems is considered as uninteresting by many, but it provides us with a framework to come to a diagnosis. The intricacy of periodontal diseases can be understood by classifying various diseases. Its goals are:

- To provide a foundation to study the etiology, susceptibility traits, pathogenesis, and treatment of diseases in an organized manner.
- To give clinicians a way to organize the health care needs of their patients.
- Assemble similar disease phenotypes in more homogeneous syndromes.

History:

The diseases of the periodontium are considered as old as the recorded history of mankind. The historical evaluation of pathology and therapeutics can be traced through a variety of sources: anatomical findings from more or less well-preserved skeletal parts, details observed in mummies, instruments and equipments collected during archaeological investigations and evidence from engravings and various manuscript[2]. Studies in paleopathology have indicated that a destructive periodontal disease, as evidenced by bone loss, accompanied early human beings in diverse cultures[16].

Almost all early historical records that involve dental topics have several chapters dealing with periodontal disease and the need for treatment. The development of dentistry can be conveniently divided into three periods[2] : magico-religious medicine (5000– 400 BC); empirico-rational medicine (400 BC–1500 AD) and scientific medicine (1500 AD – until today).

Magico-religious medicine[7]:

Hesy-Ra(2686 –2613 BC)	He has also been credited as being the first man to recognize periodontal diseases
Susruta (6th century BC)	▪ Susruta Samhita, contains four descriptions of periodontal disease ▪ Charaka Samhita, discusses proper oral hygiene and toothbrushing
Huang-ti (2500 BC)	Huang-ti Nei Ching (The Canon of Internal Medicine) describes various conditions affecting the oral cavity, including periodontal disease (detailed description of gingival inflammation, periodontal abscess and gingival ulceration).
Hippocrates of Cos (460–377 BC)	▪ Corpus Hippocrati cum (The Hippocrati c Collection) He believed that inflammation of the gums could be caused by accumulations of "pituita" or calculus
Galen of Pergamon (129–200/216 AD)	▪ Wrote the first article about dentistry with a mention of periodontal diseases.

Empirico-rational medicine[7]:

Abu al-Qasim, also known as Albucasis (936 –1013)	▪ Understanding of the major etiologic role of calculus deposits, ▪ He invented and proposed the use of many elevators and scalers, described the techniques of scaling the teeth and splinting loose teeth with gold wire.
Guy de Chauliac (1290–1368)	Chirurgia Magna (The Great Surgery) loose teeth are the result of different causes,one of them being corrosion of the gums"

Scientific medicine[7]:

A. Renaissance:

Leonardo da Vinci (1452–1519)	Presents the earliest accurate drawings of the teeth and associated structures .
Girolamo Cardano (1501–1576)	De Dentibus (About the Teeth), he mentioned a type of disease that occurred with the advancing age and led to progressive loosening and the loss of teeth, as well as a second very aggressive type that occured in younger patients.
Ambroise Paré (1510–1590)	Understood the etiologic significance of calculus and used a set of scalers to remove the hard deposits on the teeth. He developed many oral surgical procedures, such as gingivectomy for hyperplastic gingival tissues
Bartholomeus Eustachius/Bartolomeo Eustachi (1520– 1574)	Concerning the treatment of periodontitis, Eustachius had very modern ideas and recommended both the scaling of calculus and curettage of granulation tissue to promote reattachment of the gingival and periodontal tissue.
Anton van Leeu wenhoek (1632–1723)	First described oral bacterial flora, and his drawings offered a reasonably good presentation of oral spirochetes and bacilli. He even performed antiplaque experiments.

B. The 18th century:

Pierre Fauchard (1678–1761)	▪ Le Chirurgien Dentiste (The Surgeon Dentist) ▪ Fauchard described the scaling technique using instruments he invented, in order to,, detach hard matter or tartar from the teeth "& many remedies to „strengthen the gums".
John Hunter (1728–1793)	▪ The Natural History of the Human Teeth and Practical Treatise on the Diseases of the Teeth. he described the features of periodontal diseases.

C. The 19th century:

Levi Spear Parmly (1790–1859)	▪ Was a New Orleans, Louisiana, dentist who is considered the father of oral hygiene and the inventor of dental floss.
John M. Riggs (1811–1885)	▪ Leading authority on periodontal disease and at the time, periodontitis was known as **"Riggs' disease"**. ▪ He developed the concept of oral prophy laxis and prevention,
Willoughby D. Miller (1853–1907)	▪ "Father of dental prevention", who described the features of periodontal disease and their contribution in the disease development in his classic **The Microorganisms of the Human Mouth**. ▪ He believed that periodontal disease was not caused by one, but many bacterial species present normally in oral cavity ("non-specific plaque hypothesis").
Leon J. Williams (1852–1932)	▪ First described dental plaque as "gelatinous accumulation of the bacteria adherent to the enamel surface".
Vincent (1862–1950)	▪ Described the spirillum and fusiform bacilli associated with what later became Vincent's angina.

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Dominant Paradigms

Chapter 2

Three dominant paradigms that reflected the understanding of the nature of periodontal diseases were noticed during the evolution of periodontal diseases.

The classification of periodontal diseases can be placed in 3 dominant paradigms primarily based on :-

1. Clinical characteristics paradigm(1870-1920) - based on clinical features of the diseases
2. Classical pathology paradigm(1920-1970) - based on concepts of classical pathology
3. Infection / host response paradigm(1970-present) - based on infectious etiology of diseases

A. Clinical characteristics paradigm (~1870–1920)[1]:

- For the period from approximately 1870 to 1920, the researchers had insufficient information about the etiopathogenesis of periodontal diseases. There was a dispute about the nature of periodontal diseases; whether they were caused by local or systemic factors. Opinions were divided. Many of the advocates for the etiological role of local factors also acknowledged that in some cases both local and systemic factors are responsible. Various researchers depended on case descriptions and personal interpretations of clinical cases to classify periodontal diseases.

C.G. DAVIS classification (a paper published in 1879 in Dental cosmos):- who believed that there were three distinct forms of destructive periodontal disease:

- Gingival recession with minimal or no inflammation.
- Periodontal destruction secondary to 'lime deposits'. "
- Riggs' Disease'.

Similarly, in 1886 G.V. Black published his thoughts on the classification of periodontal diseases based on their clinical characteristics and his understanding of their cause into five separate groups.

- Constitutional gingivitis; including mercurial gingivitis, potassium iodide gingivitis and scurvy.
- A painful form of gingivitis. Black described a clinical condition that resembled what is now termed necrotizing ulcerative gingivitis (NUG), but he never used the term.
- Simple gingivitis. This was associated with the accumulation of debris that eventually led to 'calcic inflammation of the peridental membrane.'

- Calcic inflammation of the peridental membrane. This was associated with 'salivary' and/or 'serumal' calculus. Usually there was an even or generalized pattern of destruction of alveolar bone. The destruction usually occurred slowly. Black's description best fits the periodontal disease that is now known as chronic periodontitis.
- Phagedenic pericementitis (phagedenic Ω spreading ulcer or necrosis). This condition shared many features with 'calcic inflammation of the peridental membrane' but there was an irregular pattern of destruction and not much dental calculus. Destruction of the alveolar bone can occur slowly or rapidly. In a later publication Black replaced the term 'phagedenic pericementitis' with 'chronic suppurative pericementitis'.

Drawbacks : -

- Little or no scientific evidence was used, it was only based on their personal interpretation of what they saw clinically as the primary basis for classifying periodontal diseases.
- No generally accepted terminology or classification system for periodontal diseases was adopted during this era. As a result in the latter part of 19th century periodontitis went under numerous names including: 'pyorrhea alveolaris' , 'Riggs' disease' , 'calcic inflammation of the peridental membrane' , 'phagedenic pericementitis' , and 'chronic suppurative pericementitis' . During this period, the dominant term used for destructive periodontal disease was pyorrhea alveolaris.

B. Classical pathology paradigm (~1920–1970):

· During this time a new concept developed that periodontal diseases can be of 2 types- inflammatory and non-inflammatory ('degenerative' or 'dystrophic'). This was based on the observation that certain forms of periodontal diseases were due to degenerative changes in the periodontium such as cementopathia. As a result most of the classification systems in this era included disease categories such as 'dystrophic', 'atrophic' or 'degenerative'. Around 1970, a different paradigm had begun to dominate thoughts about the nature of periodontal diseases. Also observation that a patient with hypophosphatasia who had premature loss of anterior deciduous teeth, also harbored Porphyromonasgingivalis in the sub gingival flora[2-5], suggested that something other than hypoplasia of cementum might have contributed to periodontal destruction.

C. Infection/ host response paradigm (~1970–present):

After the publication of Robert Koch's postulates (1876), researchers stressed upon the infectious nature of periodontal diseases. W.D. Miller[6], in particular, was an early proponent of the infectious nature of periodontal diseases. He stated three factors which were to be taken into consideration in every case of pyorrhea alveolar is:

1) Predisposing circumstances,
2) Local irritation,
3) Bacteria.

Miller also recognized that certain systemic conditions (e.g. diabetes, pregnancy) could modify the course of the disease. The next major discovery in periodontal microbiology was the preliminary demonstration in 1976–1977 of microbial specificity at sites with perio-dontosis.[7,8] This finding, coupled with the demonstration in 1977– 1979 that neutrophils from patients with juvenile periodontitis (periodontosis) had defective chemotactic and phagocytic activities[9,10], marked the beginning of the dominance of the Infection/Host Response paradigm

Classification systems in the modern era represent a blend of all three paradigms since there is a certain amount of gravity to some of the earliest thoughts about the nature of periodontal diseases. History reveals that people in the past opposed the modification of these entities. They were adamant to accept a particular classification in spite of it having many flaws. But in the true sense, they should be periodically modified based on modern thinking and concepts.

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3 Chapter

Classifications of:- Antioxidants

Definition :

In 1992 Halliwell and Gutteridge defined antioxidants as, "those substances which when present at low concentrations, compared to those of an oxidizable substrate, will significantly delay or inhibit oxidation of that substrate" [1].

Sics in 1996 defined antioxidants as, "the substances that neutralize free radicals or their actions"[2].

Ternay and Sorokin in 1997 defined antioxidant as, "any substance that hinders a free radical reaction"[3].

Azzi and Davies in 2004 defined antioxidant as, "the substances which counteract free radicals and prevent the damage caused by them.

Antioxidants by virtue of scavenging the oxidants prevents chain reactions or activation of oxygen into highly reactive products before they affect the cells"[4].

Classified As :

A. According to mode of action It can be classified as[1]:

A. Intracellular: Superoxide dismutase enzyme 1 and 2, catalase, glutathione peroxidase, DNA repair enzymes e.g. poly (ADP-ribose) polymerase, others, reduce glutathione, ubiquinone (reduced form).

B. Extracellular: Superoxide dismutase enzyme3, selenium, glutathione peroxidase, lactoferrin, transferring, ascorbate, uric acid, carotenoids, ceruloplasmin.

B. According to the location of action[1]:

1. Preventive antioxidants Enzymes:

Superoxide dismutase enzymes (1,2 and 3), catalase, glutathione peroxidase, DNA repair enzymes. Metal ion sequestrators: Albumin, lactoferrin, transferrin, haptoglobin, ceruloplasmin, hemopexin, carotenoids, superoxide dismutase, catalase, glutathione peroxidase, glutathione reductase, uric acid, polyphenolic flavonoids.

2. Scavenging antioxidants:

Ascorbate, carotenoids, uric acid, α – tocopherol, polyphenols, bilirubin, albumin, ubiquinone, reduced glutathione and other thiols.

C. According to solubility[1]:

A. Lipid soluble: Haptoglobin, ceruloplasmin, albumin, ascorbate, uric acid, polyphenolic flavonoids, reduce glutathione, and other thiols.

B. Water soluble: α- tocopherol, carotenoids, bilirubin, quinines.

D. According to structures they protect [1]:

A. DNA protective antioxidants:
Superoxide dismutase enzyme 1 and 2, glutathione peroxidase, DNA repair enzymes [poly (ADP) ribose polymerase], reduced glutathione, cysteine.

B. Protein protective antioxidants:
Sequestration of transition metals by preventative antioxidants. Scavenging by competing substrates. Antioxidant enzymes.

C. Lipid protective antioxidants:
α- tocopherol, ascorbate, carotenoids, reduced glutathione, glutathione peroxidase, bilirubin.

E. According to their origin [1]:

A. Exogenous antioxidants: Carotenoids, ascorbic acid, tocopherols (a,b,c,d), polyphenols, folic acid cysteine.

B. Endogenous antioxidants: Catalase, superoxide dismutase, glutathione peroxidase, glutathione –S- transferase, reduce glutathione, ceruloplasmin, transferrin, ferritin, glycosylases.

C. Synthetic: N-acetylcysteine, penicillamine, tetracyclines.

F. RH Liu in 2004 classified antioxidants in to two major groups[5] :

A. Enzymatic antioxidants.

B. Non enzymatic antioxidants.(Figure:1)

- 10 Antioxidants are grouped into two namely:
- Primary or natural antioxidants: Antioxidants minerals - Selenium, copper, iron, zinc and manganese. Anti oxidants vitamins – They include- Vitamin C, Vitamin E, Vitamin B. Phytochemicals – Flavonoids
- Secondary or synthetic antioxidants : Butylated hydroxyanisole (BHA). Butylated hydroxytoluene (BHT). Propyl gallate (PG) and metal chelating agent (EDTA). Tertiary butyl hydroquinone (TBHQ). Nordihydroguaiaretic acid (NDGA)[6].

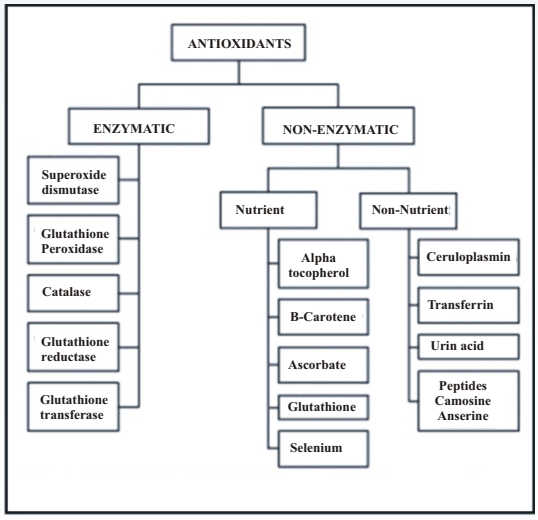

Fig 1. Antioxidants classified as enzymatic and non enzymatic

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4 Chapter Antiplaque Agents

Definition :

Chemicals that have an effect on plaque, sufficient to benefit gingivitis and/or caries[1].

Classified As :

A. According to Kormann (1986) [2]

Anti-plaque agents based on their mechanism of action

First generation anti plaque agents: They are capable of reducing plaque up to 20-50%.They exhibit poor retention within the mouth. Eg: Triclosan, antibiotics, phenols, quaternary ammonium compounds and Sanguinarine.

Second generation anti plaque agents: They produce over all plaque reduction of up to 70-90%. These are better retained than first generation agents. Eg: bis biguanides (chlorhexidine)

Third generation anti plaque agents: They block binding of microorganisms to the tooth or to each other. They have poor retention capacity when compared to the second generation agents. Eg: Delmopinol

B. Classification by Mandel[2]

a. Antiplaque enzymes
Amyloglucosidase, glucose oxidase, dextranse, fungal enzymes, mucinase, mutanase, pancreatin, proteinase-amylase, zendium
b. Plauqe- modifying agents
Ascoxal (astra zeneca), urea peroxide

C. They can also be classified as [3]

Group A agents- antiplaque Chlorhexidine, acidified sodium chlorate, salifluor and delmopinol

Group B agents- plaque inhibitory cetyl pyridinium chloride, essential oil and triclosan rinses used as adjuncts to mechanical cleaning

Group C agents- Have a low to moderate activity and are used for cosmetic purposes like breath freshening Sanguinarine , oxygenating agents, saturated pyrimidine, hexetidine

D. Based on chemical composition[4]

Bisbiguanides- chlorhexidine, alexidine
Bispyridines- octenidine hydrochloride
Halogens- iodine, iodophores, fluorides
Heavy metal salts- silver, mercury, zinc, copper, tin
Herbal extracts- sanguinaria extract
Oxygenating agents- peroxides, perborate
Phenolic compounds- phenol, thymol, triclosan, 2-phenylphenol, hexylresorcinol, Listerine
Pyrimidines- hexetidine
Quaternary ammonium compounds- cetylpridinium chloride, benzethonium chloride, domiphen bromide

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5 Chapter

Biofilm

Definition :

Costerton et al in 1995 defined biofilm as "a structured community of bacterial cells enclosed in a self produced polymeric matrix and adherent to an inert or living surface"[1].

Percival et al in 2000 defined biofilm as "microbial cells immobilised in a matrix of extracellular polymers acting as an independent functioning ecosystem, homeostatically regulated"[2].

Marsh P D 2005 defined biofilm as "Orientated aggregations of microorganisms attached to each other or to a surface and enclosed in extracellular polymeric substance (EPS) produced by themselves"[3].

Classified As :

A. On basis of its location[4]

1. Supra gingival - Present coronal to the gingival margin.

2. Sub gingival - Present apical to the gingival margin.

B. On basis of pathogenicity[4]

1. Cariogenic - Generally acidogenic and gram positive.

2. Periopathogenic- Mostly basophilic and gram negative

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• **Internalized Stains :** Internalised discoloration is the incorporation of extrinsic stains within the tooth substance following dental development [Fig. 4]. It occurs in enamel defects and in the porous surface of exposed dentine. The routes by which pigments may become internalised are:

1. Developmental defects.
2. Acquired defects.

a) Tooth wear and gingival recession.
b) Dental caries.
c) Restorative materials[4]

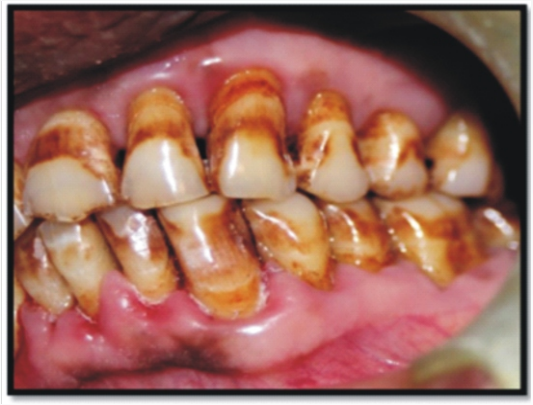


Fig 4. Internalised tobacco stains

B. Depending on colour[3]

Colour	Characteristics	Location	Etiology
Brown Stain	Stain is thin and bacteria free.	Most commonly found on the buccal surface of the maxillary molars & the lingual of the mandibular incisors, & least on the labial surfaces of maxillary anterior teeth.	Found in individuals with poor oral hygiene due the deposition of tannin found in tea, coffee and other beverages and those who use a dentifrice with inadequate clearing and polishing action. (Hattab et al.; 1999)
Black Stain	Thin or wide black line firmly attached to the tooth surface that is difficult to remove with a tooth brush & dentifrice. It mostly tends to recur after removal	Found on the facial and lingual surfaces of the teeth near the gingival margin and extends on the proximal surfaces.	It is caused by colour producing (chromogenic) bacteria, primarily Actinomycetres. The black stain is due to ferric sulphide formed by the reaction between hydrogen sulphide produced by bacteria & iron found in saliva & gingival exudaters. (Hattab et al.; 1999)
Green Stain	These are tenacious and thick stains common in children.	Found on the facial surface of the maxillary anterior teeth near the gingival third.	It is considered to be remnants of the primary enamel cuticle that has been stained. the discoloration is considered to be due to fluorescent bacteria and fungi like Penicillium and aspergillurs. (Hattab et al;1999) It has also been seen in people exposed to copper salts in mouth washes. (Manuel et al; 2010)
Orange Stain	These are easily removable. it is rare and found in only 3% of the population.	Usually occurs on the labial surface of maxillary & mandibular anterior teeth at the gingival teeth.	Found in individual with poor oral hygiene. Chromogenic bacteria of orange stain, such as Serratia marcescens and Flavobacterium lutescens are considered the main cause. (Hattab et al.; 1999)
Metallic Stain	This type of staining evident in industrial workers exposed to metal.	This staining is mostly evident is exposed tooth surface or outer environment.	The metals combine with acquired pellicle to produce a surface stain, or penetrate the tooth substance to cause permanent discoloration. Different metal producers a different colour of staining. (Manuel et al; 2010)
Yellow	Stains easily removed by proper brushing.	Mostly found on the cervical interproximal areas of teeth, dorsum of the tongue.	Caused by use of essential oil and phenolic mouthrinsers (Manual et al; 2010)
Red-Black	Thick, hard staining not easy to remove.	Found on the facial, lingual and occlusal surface of both anterior and posterior teeth.	Found in people who are habitual betal palm leaf and nut chewer. (Sruthy et al; 2013)
Violet Black	Easily removable by proper brushing.	Found on the cervical interproximal areas of teeth, dorsum of the tongue.	Due to the presence of potassium permanganate in mouthrinsers. (Sruthy et al; 2013)

Table 2

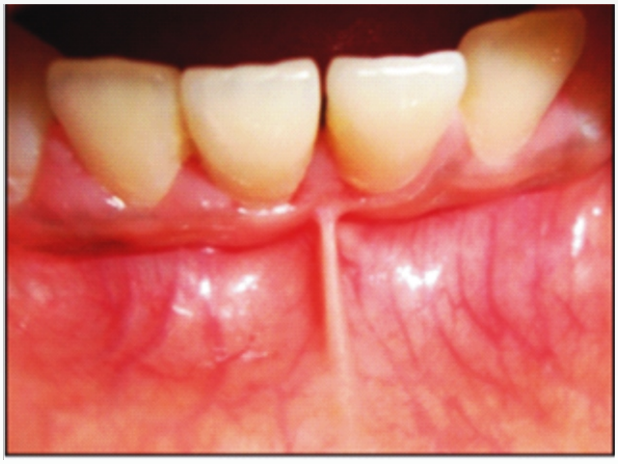


Figure 3: Papillary frenal attachment

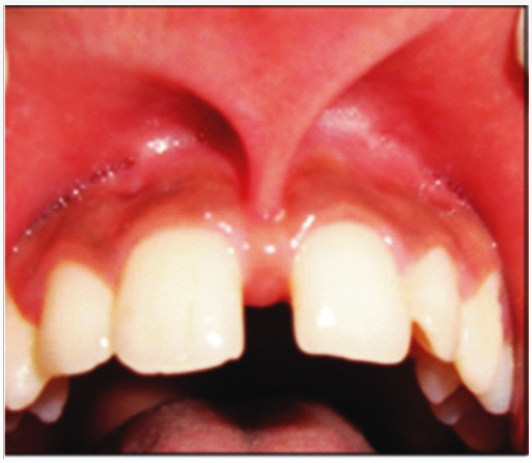


Figure 4: Papilla penetrating frenal attachment

B. Other variations of normal frenal attachment include (Swerin et al. 1971)[4] :

- Simple frenum with a nodule [Figure 5]
- Simple frenum with appendix [Figure 6]
- Simple frenum with nichum [Figure 7]
- Bifid labial frenum
- Persistent tecto labial frenum
- Double frenum
- Wider frenum [Figure 8]

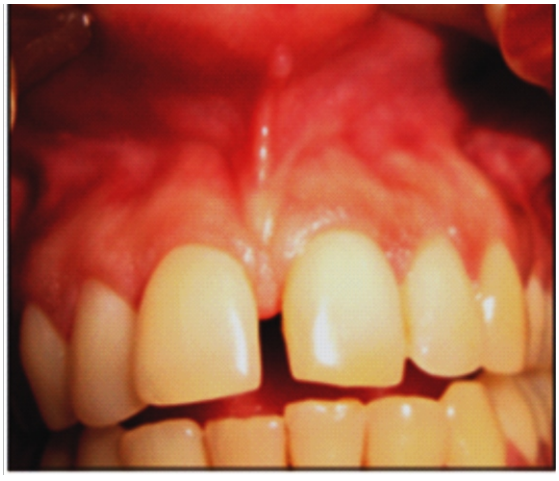


Figure 5: Simple frenum with a nodule

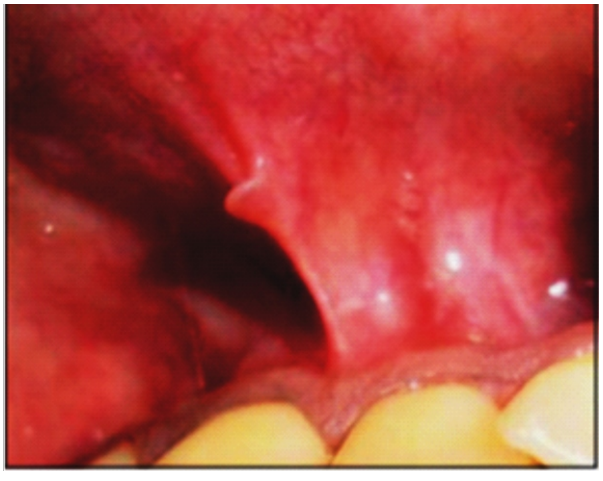
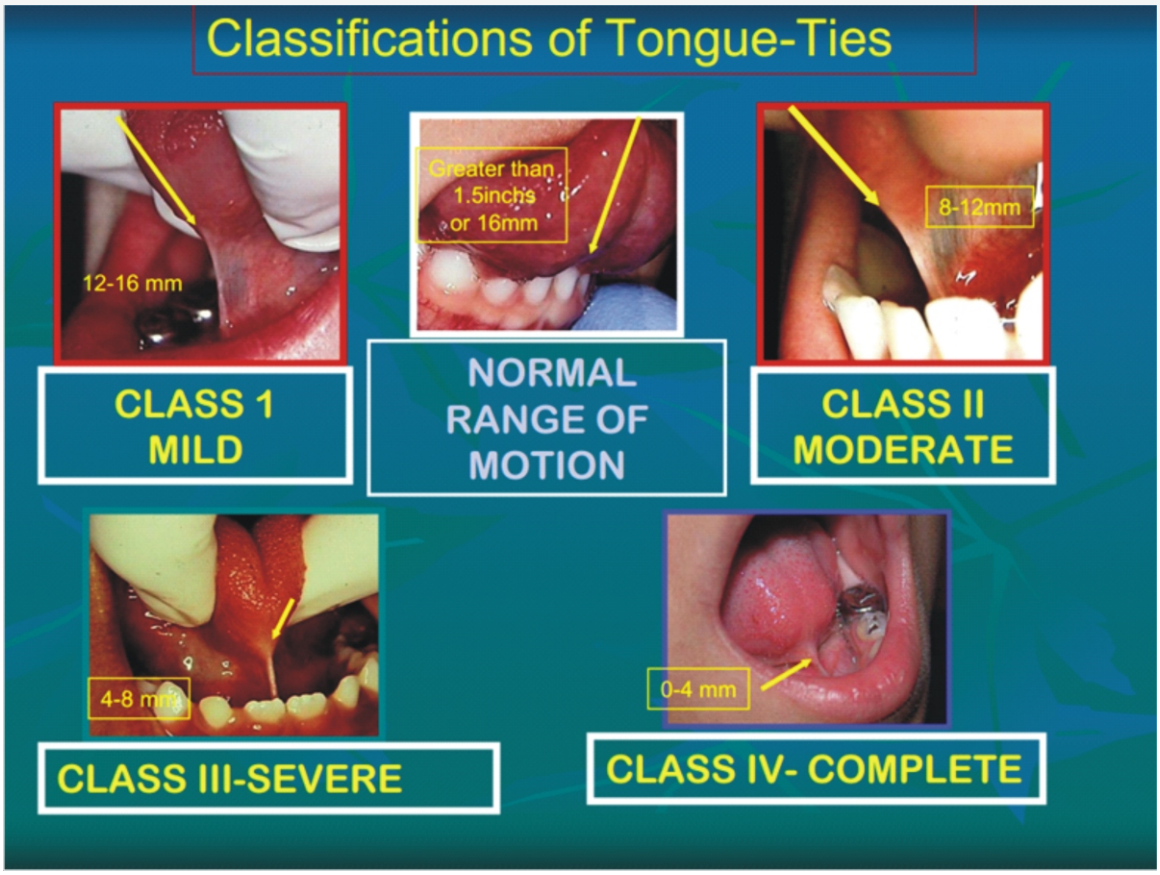


Figure 6: Simple frenum with appendix



D. A set of clinical criteria to classify the position of the labial frenulum [Kotlow, 2010][6]

Maxillary frenulum	
Class I	Little or no tissue attachment of the lip to the gingival tissue
Class II	Frenulum attaches above or at the border between free and attached gingival tissue
Class III	Frenulum attaches at or into the interproximal area between the upper central incisor
Class IV	Frenulum attaches into the palatal tissue

Considering the presence of cervical discrepancies (step), measured with a periodontal probe perpendicular to the long axis of the:

Class (+), presence of cervical step (>0.5 mm) involving the root or the crown and the root Class (-), absence of cervical step

M.Nagappa and Mukta's Classification (2018)[11]

Proposed Classification System for Gingival Recession

It is classified into four classes with subdivisions a and b.

Class I – Apical shift in the crest of marginal gingiva 1–2 mm from CEJ

- I a – without any interproximal tissue loss clinically [Fig. 2]
- I b – with interproximal tissue loss coronal to interproximal CEJ clinically [Fig. 3].

Class II – Apical shift in the crest of marginal gingiva >2 mm <3 mm from CEJ

- II a – without any interproximal tissue loss clinically [Fig. 4]
- II b – with interproximal tissue loss coronal to interproximal CEJ clinically [Fig. 5].

Class III – Apical shift in the crest of marginal gingiva ≥ 3 mm from CEJ

- III a – without any interproximal tissue loss clinically [Fig. 6]
- III b – with interproximal tissue loss apical to interproximal CEJ clinically [Fig. 7]

Class IV – Apical shift in the crest of marginal gingiva >3 mm from CEJ with severe malposed tooth

- IV a – without any interproximal tissue loss clinically [Fig. 8]
- IV b – with interproximal tissue loss apical to interproximal CEJ clinically [Fig. 9]

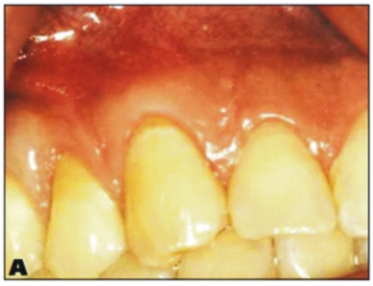


Figure 2: Class I - apical shift in the crest of marginal gingiva 1-2 mm from cemento-enamel junction. I A- without any interpro- ximal tissue loss clinically.

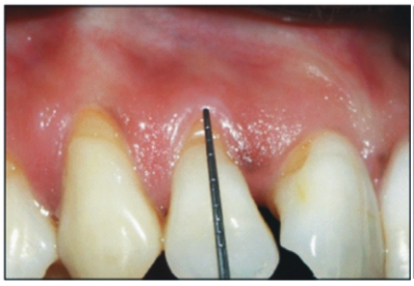


Figure 3: Class I b- with interproximal tissue loss coronal to interproximal cementoenamel junction.

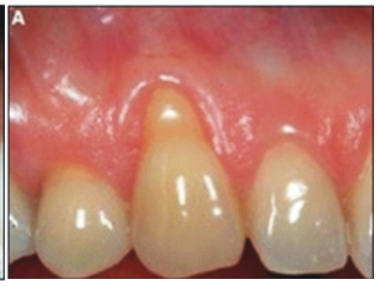


Figure 4: Class II - apical shift in the crest of marginal gingiva 2-3 mm from cemento-enamel junction. II a- without any interpro-ximal tissue loss.

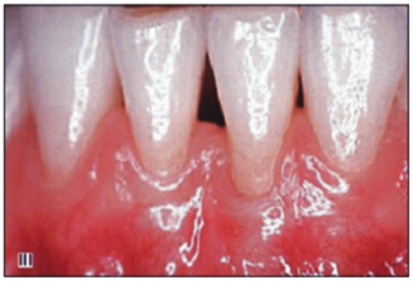


Figure 5: Class II b- with interproximal tissue loss coronal to interproximal cementoenamel junction.

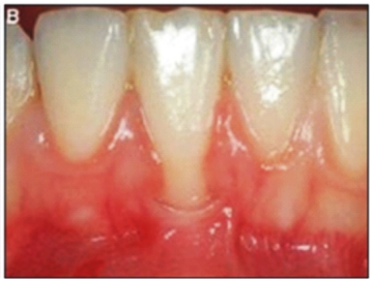


Figure 6: Class III - apical shift in the crest of marginal gingiva > 3 mm from cemento-enamel junction. IIIa- without any interpro-ximal tisssue loss.

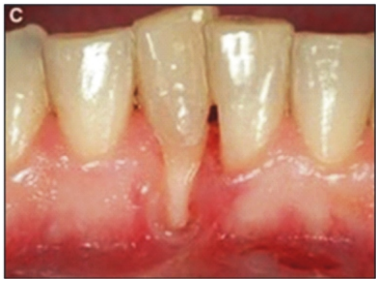
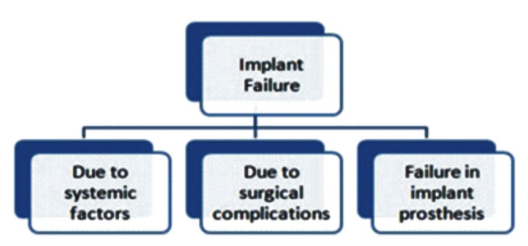


Figure 7: Class III b- with interproximal tissue loss apical to interproximal cementoenamel junction.

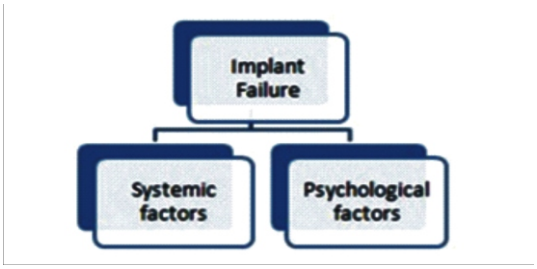
ii) Psychological Factors:
Lack of support
Cognitive difficulty
- Mental retardation
- Dementia
- Psychosis

Emotional problems
- Interpersonal problems
- Behavioral problems
- Problematic attitudes and beliefs.

8. Nallaswamy[6]

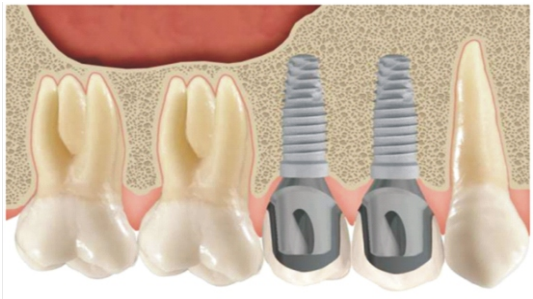


9. Matukas[3]



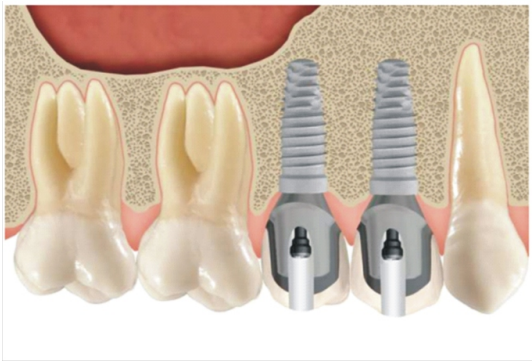
E. Adjacent dental implants classification based on restorative design.[7]

Class I prosthetic design: Individual crowns are cemented on the abutments

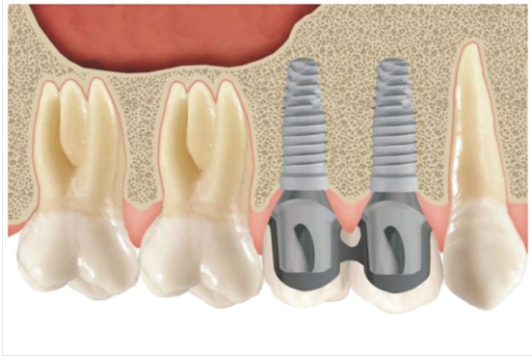


Class II prosthetic design: Individual crowns are screw-retained on the implants

Class III prosthetic design: Individual screw-retrievable cement-retained are made



Class IV prosthetic design: Splinted crowns are cemented on the abutments



2. Suturing Needle [1] :

A. According to eye: Eye less needles.
Needles with eye.

B. According to shape: Straight needles.
Curved needles.

C. According to cutting edge: Round body.
Cutting: a-conventional.
b-reverse cutting.

D. According to its tip: Triangular tip.
Round tip
Blunt tip.

E. Others: Spatula needles.
Micro point needles.
Cuticular needles.
Plastic needles.

3. Suturing Techniques [1] :

A. Simple loop modification of interrupted suture technique.

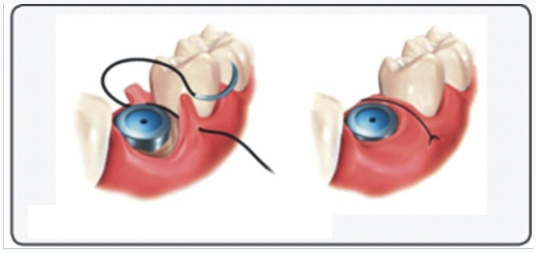


Figure 2: Interrupted simple suture

B. Continuous non-interlocking suture technique.

C. Continuous locking suture technique.

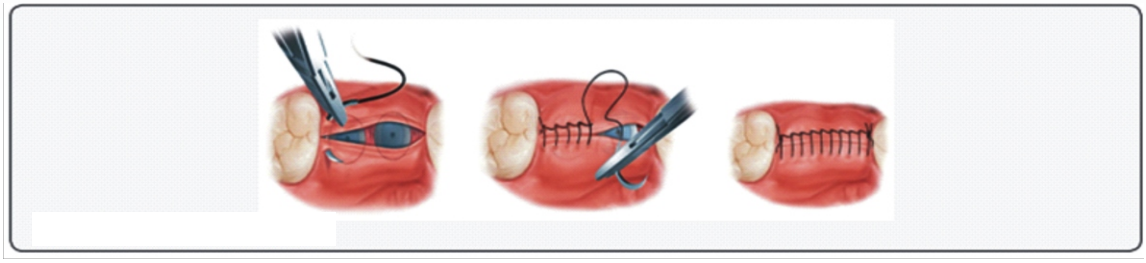


Figure 3: Continuous locking suture.

D.Vertical mattress suture technique.

I. internal vertical mattress :

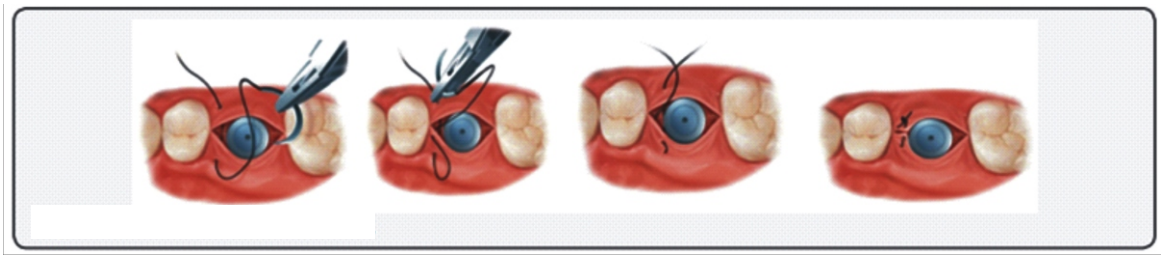


Figure 4: Internal vertical mattress suture.

II. External vertical mattress :

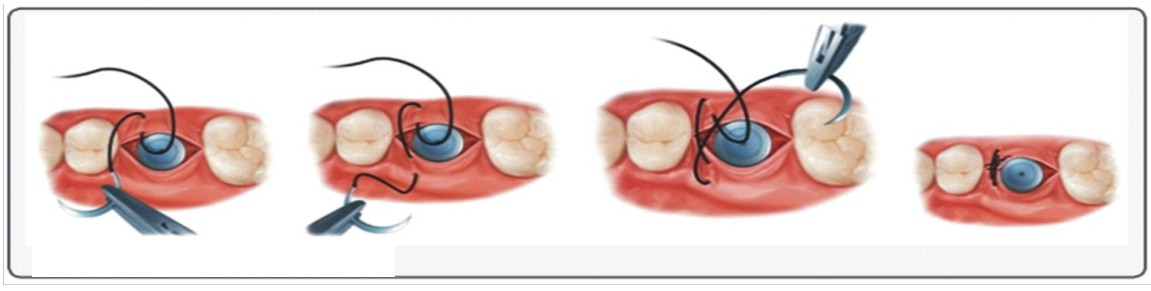


Figure 5: External vertical mattress suture.

E.Horizontal mattress suture technique.

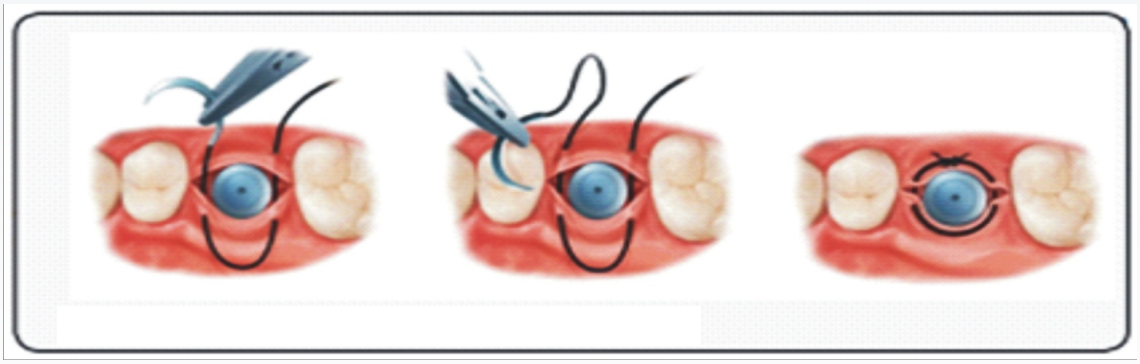


Figure 6: Horizontal mattress suture.

F.Continuous horizontal mattress suture technique.

G.Modification of interrupted suture technique.

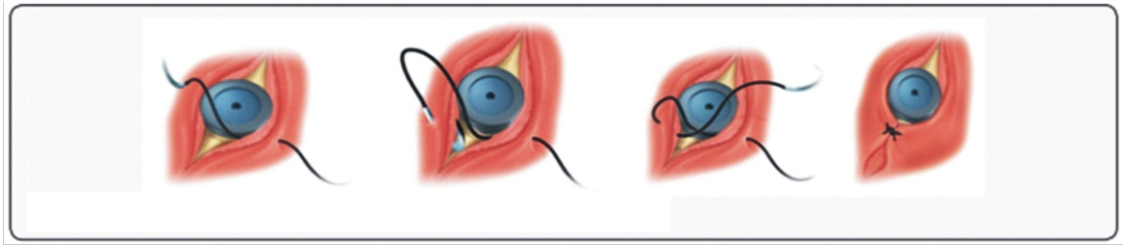


Figure 7: Suture

H.Cross (Crisscross) suture technique.

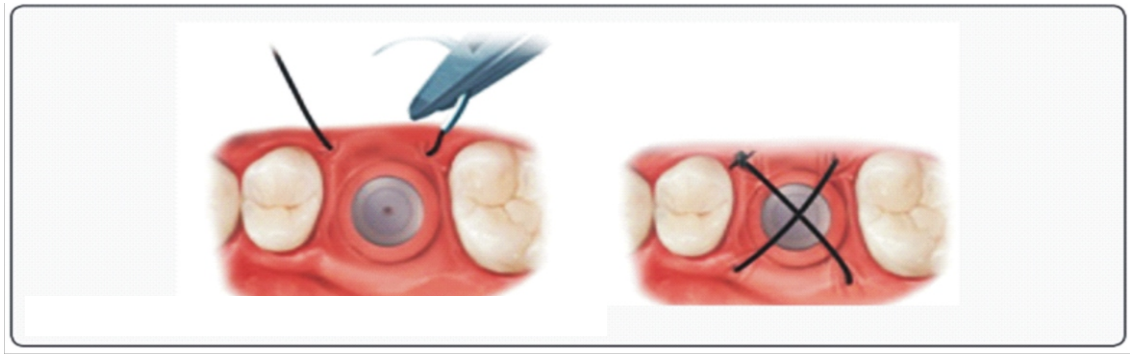


Figure 8: Cross Suture

I.Periosteal Suturing Technique.

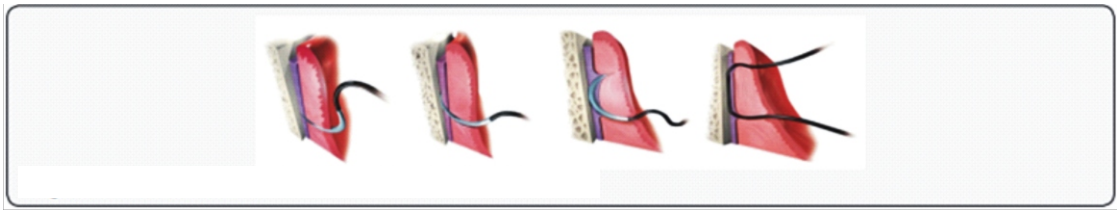


Figure 9: Periosteal Suture