Tips & Tricks in PERIODONTOLOGY

Tips & Tricks

Foreword
SG Damle

Shalu Bathla

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Tips and Tricks in Periodontology

Tips and Tricks in Periodontology

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FOREWORD

Periodontics is a fascinating field of endeavour, arguably the most exciting, dynamic and constantly challenging area of specialization. It is indeed a happy occasion to see the publication of a comprehensive book on Periodontics in India.

The principle work of a genius is not perfection but originality, the opening of new frontier. The world makes way for people who know where they are going and also taking others. The author of this book begins from the simple things in the book and gradually takes the reader on a more difficult concepts, making them appear simple and interesting.

I congratulate **Dr Shalu Bathla** on her brilliant effort and wish this book a great success.

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Preface

"Destiny – By choice or by chance?
Hundreds of pages, but only a few to pass"

After studying many books, we finally need to write only a few pages in the exam. Hence, the objective behind the making of this book is to control our destiny by choice and leave nothing to chance.

Periodontics is a rapidly changing branch of dentistry with new scientific revelations unveiling many mysteries. Some controversies, still surrounding the basic foundation of aetiology, phenomenology, treatment methods etc., make it very difficult for a student to understand its content. The book is prepared with the objective of keeping in view the problems faced by the students during viva voce.

This maiden book of Periodontology in India has over 700 questions and answers and important points useful for the UG and PG students and also the students preparing for the PG entrance exams. This book is not a substitute for the standard textbooks available in the field of Periodontics written by the experienced periodontists. Thereby, it is suggested that the students should pursue the additional readings.

I do not claim exclusive credit for the book. Much of the material has been collected from the journals, periodicals, review articles, books, discussions, personal communications and similar sources.

Without doubt, there will be errors, few imperfections, omissions and over simplification. Hoping that the rest of the material will be enough to stimulate insight and new trains of thoughts into the subject of Periodontics. I have put a bit of heart and soul into it. Therefore, I hope that this work will be immensely educative.

Any suggestions and criticisms are most welcome.

Shalu Bathla

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Respected **(Parents)** and **(GOD)**, I lay this book at your feet.

I am forever grateful to **Dr RK Sharma,** who encouraged me as a fledgling dental student to pick up a scalpel and perform my first periodontal surgery and to Dr Rajan Gupta and Dr Shikha Tiwari, who enthusiastically supported my endeavour to develop a curriculum in Periodontics.

I particularly thank Dr Nageshwar Iyer and Dr Meenakshi Iyer for their thoughtful and creative comments. I am extremely thankful to Dr Sanjay Tiwari from whom I have learnt what dedication means. The other teachers who have influenced me tremendously are Dr Ravi Kapoor, Dr Raman Kapoor, Dr SC Narula, Dr Suresh DK and Dr Shashikant Hegde.

I am highly thankful to renowned teacher icon to Pediatric and Preventive Dentistry, Dr SG Damle for writing the foreword for this book.

I gratefully acknowledge my debt to my father-in-law, Dr JC Bathla for nurturing the seeds of this endeavour at its infancy and my brother, Mr Pankaj Chandna who has selflessly and lovingly been there for me.

I wish to express my deepest thanks to my husband, Dr Manish Bathla for his support, understanding and encouragement during long hours of forced isolation and commitment required to accomplish this feat.

I would like to thank the postgraduate students, Dr Rachna Jain, Dr Preetinder Singh, Dr Anushi Mahajan, Dr Alka Kaushik and Dr Gayatri for proofreading.

I would like to express my thanks to Shri Jitendar P Vij (Chairman and Managing Director) and Mr Tarun Duneja (Director–Publishing), Ms Bano, Mr Raman and the editorial staff of Jaypee Brothers Medical Publishers (P) Ltd, New Delhi, who have done a great job by "putting an icing on the cake" through their professional expertise to make my work, reader / friendly and reach it to your desk.

Contents

1 Normal Periodontium

A. GINGIVA

Q.1. What is Gingiva?

It is the part of oral mucosa that covers the alveolar processes of the jaw and surrounds the neck of the teeth.

Q.2. What are the parts of Gingiva?

(i) Marginal or free gingiva
(ii) Attached gingiva
(iii) Interdental gingiva.

Q.3. What are the differences between Alveolar mucosa and Attached gingiva.

		Alveolar mucosa	*Attached gingiva*
1	Colour	Red	Pink
2	Surface texture	Smooth and shiny	Stippled
3	Epithelium	• Thinner	• Thicker
		• Nonkeratinized	• Parakeratinized
		• No Rete pegs	• Rete pegs present
4	Connective tissue	• More loosely arranged	• Not so loosely arranged
		• More blood vessels	• Moderate blood vessels

Q.4. What is the width of attached gingiva?

It is the distance between mucogingival junction and the projection on the external surface of the bottom of gingival sulcus / periodontal pocket.

Width of attached gingiva on facial aspect –

- Maxillary incisor region – 3.5 to 4.5 mm
- Mandibular incisor region – 3.3 to 3.9 mm
- Maxillary first premolar – 1.9 mm
- Mandibular first premolar – 1.8 mm

Q.5. What is the significance of attached gingiva?

(i) Gives support to the marginal gingiva.
(ii) Provide attachment or a solid base for the movable alveolar mucosa for the action of lips, cheeks and tongue.
(iii) Withstand frictional stresses of mastication and toothbrushing.
(iv) Acts as a barrier for passage of inflammation.
(v) Provide resistance to tensional stress: Attached gingiva serves as a buffer to free gingival margin and mobile alveolar mucosa.

Q.6. Where stippling is normally present on gingiva?

(i) Attached gingiva.
(ii) Centre of Interdental papilla.

Q.7. What is Col?

It is a valley like depression which connects the facial and lingual papillae and conforms to the shape of the interproximal contact areas.

Q.8 What is mucogingival junction and its clinical importance?

It is the interface between the more apically located alveolar mucosa and the more coronally located attached gingiva.

Clinical importance of the mucogingival junction is in measuring the width of attached gingiva.

Q.9 Where is mucogingival junction normally present?

Mucogingival junction is present on the three gingival surfaces:

- Facial gingiva of the maxilla
- Facial gingiva of the mandible
- Lingual gingiva of the mandible.

The palatal gingiva of the maxilla is continuous with the tissue of the palate, which is bound down to the palatal bones. Because the palate is devoid of freely movable alveolar mucosa, there is no mucogingival junction.

Q.10 Write salient microscopic features of gingiva.

A. Epithelium:
  (i) Oral epithelium:
    - Stratum basale
    - Stratum spinosum
    - Stratum granulosum
    - Stratum corneum
  (ii) Sulcular epithelium
  (iii) Junctional epithelium

B. Basal lamina: Epithelium – connective tissue interface.

C. Connective tissue / Lamina propria:
  (i) Cells:
    - Fibroblasts
    - Mast cells

- Eosinophils
- Macrophages
- Adipose cells
- Inflammatory cells – neutrophil, plasma cells, lymphocyte

(ii) Fibres:
- Collagen fibers
- Reticulin fibers
- Elastic fibers

(iii) Neurovascular bundles.

Q.11 Write the cells present in the gingival epithelium.

(i) Principle cell: Keratinocytes

(ii) Non-keratinocytes / Clear cells:
(a) Langerhan cells
(b) Merkel cells
(c) Melanocytes.

Q.12 What are the functions of non-keratinocytes?

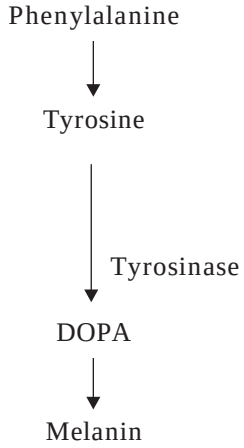
(i) Langerhan cells: are responsible for communication with immune system by acting as antigen – presenting cells for lymphocytes.

(ii) Melanocytes:
(a) Synthesize melanin which is responsible for providing color to gingiva.
(b) Are responsible for the barrier to UV damage.

(iii) Merkel cells: acts as tactile perceptors.

Q.13 Where melanin is formed and stored?

Melanin is synthesized in an organelle called, premelanosomes / melanosomes in melanocytes cells. Melanin is stored in melanophages / melanophores.



Q.14 What is the ratio of melanocytes to keratinocytes producing epithelial cells?

1:36 cells

Q.15 What is basal lamina?

Basal lamina is a structural entity of epithelial origin of 300 – 400 Å thick which is visible only under electron microscope. It consists of electrolucent zone called lamina lucida and an inner electrodense zone called lamina densa.

Q.16 What is internal basement lamina and external basement lamina?

- Internal basement lamina is junctional epithelium – tooth interface.
- External basement lamina is junctional epithelium – connective tissue interface.

Q.17 Classify various junctional complexes present in gingiva.

A. Tight junctions
B. Adhesive junctions
   (a) Cell to cell
      (i) Zonula adherens
      (ii) Desmosomes
   b. Cell to matrix
      (i) Focal adhesions
      (ii) Hemidesmosomes
C. Communicating (gap) junctions.

Q.18 What is Dentogingival unit?

The junctional epithelium and gingival fibres together forms a functional unit called as dentogingival unit.

Q.19 Which type of collagen is present in gingiva?

Type I, III, IV, V, VI.

Q.20 Write about gingival fibre's position and function.

(i) Dentogingival group: These fibres extend from the cementum apical to junctional epithelium and course laterally and coronally into lamina propria of the gingiva. Provide gingival support.

(ii) Alveologingival group: These fibres arise from the alveolar crest and insert coronally into lamina propria of the gingiva. Attaches attached gingiva to alveolar bone.

(iii) Circular group: This group of fibres encircles the teeth in a cuff or ring like fashion. Maintain contour and position of free marginal gingiva.

(iv) Transseptal fibres: These are the group of prominent horizontal fibres located interproximally that extends from cementum of one tooth to the cementum of the neighbouring tooth. Maintain relationship of adjacent teeth, protect interproximal bone.

(v) Dentoperiosteal group: On the oral and vestibular surfaces of jaws, dentoperiosteal group of fibres extends from the tooth, passing over the alveolar crest to blend with fibres of the periosteum of the alveolar bone. Anchors tooth to bone, protect periodontal ligament.

(vi) Semicircular group: Group of fibres which attach at the proximal surface of a tooth, immediately below the cementoenamel junction, go around the facial or lingual marginal gingiva of the tooth and attach on the other proximal surface of the same tooth.

(vii) Transgingival group: Fibres that attach in the proximal surface of one tooth, transverse the interdental space diagonally, go around the facial or lingual surface of the adjacent tooth, again traverse diagonally the interdental space and attach in the proximal surface of the next tooth. Secure alignment of teeth in the arch.

(viii) Intergingival group: These fibres run parallel to dentition on vestibular and oral surfaces. They provide contour and support for the attached gingiva.

(ix) Interpapillary group: They are seen in the interdental gingiva extending in a faciolingual direction. Provide support for interdental gingiva.

Dentogingival, dentoperiosteal and alveologingival fibres group provide the attachment of gingiva to the tooth and to the bony structure. Fibres of circular, semicircular, transgingival, intergingival and transseptal bundles connect teeth to one another.

Q.21 What is the clinical significance of Transseptal fibres?

The transseptal fibres collectively form an interdental ligament connecting all the teeth of the arch. This ligament, although belonging to the supra-alveolar fibre apparatus, appears to be uniquely important in maintaining the integrity of the dental arch. It is rapidly reformed after excision. Residual portions of transseptal fibres are seen, even in advanced stages of resting periodontal disease.

Q.22 What is the normal length of junctional epithelium?

0.25 - 1.35 mm

Q.23 Discuss permeability of junctional epithelium.

Junctional epithelium allows two-way movement of variety of substances:

(i) From connective tissue into crevice – Gingival fluid exudates, PMNs, Ig, complement and various cells of immune system.

(ii) From crevice to connective tissue – Foreign material such as carbon particles, trypan blue.

Q.24 Why junctional epithelium is easily penetrated?

(i) Along the junctional epithelium, sub-epithelial vessels are parallel to the surface and are made up mostly of venules rather than

capillaries. These venules have a greater disposition towards increased permeability than do capillaries and arterioles and they are more susceptible to haemorrhage and thrombosis.

(ii) Few tight junctions
(iii) Minimal cytoplasmic filaments
(iv) Higher number of intercellular spaces
(v) Lower number of desmosomes.

Q.25 What are the functions of junctional epithelium?

(i) Act as barrier
(ii) Provide attachment to tooth
(iii) Has rapid turnover rate
(iv) Secretes antimicrobial peptides—defensins, calprotectin and cathelicidin
(v) Allow GCF flow.

Q.26 What are the problems related to junctional epithelium?

(i) Permeability
(ii) Degeneration
(iii) Deattachment
(iv) Lateral and apical proliferation.

Q.27 Write peculiar feature of junctional epithelium.

Junctional epithelium is the only attachment in the body between soft tissue and a calcified tissue which is exposed to the external environment.

Q.28 What is Dentogingival plexus, Sub-epithelial plexus and Intermediate plexus?

- Dentogingival plexus: Plexus of blood vessels beneath junctional epithelium. The blood vessels in this plexus have a thickness of approximately 40 μm, which means that these are mainly venules. In healthy gingiva, no capillary loops occur in dento gingival plexus.
- Subepithelial plexus: Plexus of blood vessels beneath oral epithelium of free and attached gingiva, yield thin capillary loops, of 7 μm to each connective tissue papilla.
- Intermediate plexus (Sicher 1966): Fibers arising from cementum and bone are joined in midregion of periodontal ligament space giving rise to a zone of distinct appearance in light microscope. It was believed that the intermediate plexus provide a site where rapid remodeling of fibres occurs, allowing adjustment in the ligament to be made to accommodate small movements of tooth. However, evidence derived from electron microscope provide no support for this and was believed to be artifact.

Q.29 What is the difference between Oral, Sulcular and Junctional epithelium?

		Oral epithelium	*Sulcular epithelium*	*Junctional epithelium*
1	Keratinization	Keratinized	Non-keratinized	Non-keratinized
2	Rete pegs	Present	Absent	Absent
3	Strata granuloma and corneum	Present	Lacking	Lacking
4	Merkel cells	Present	Absent	Absent
5	Langerhan cells	Present	Few	Absent
6	Type IV collagen in basal lamina	Present	Absent	Absent
7	Tight junctions	More	Few	Few
8	Acid phosphatase activity	Present	Lacking	Lacking
9	Glycolytic enzyme activity	High	Lower	Lower
10	Intercellular space	Narrower	Narrower	Wider

Q.30 What is the arterial supply of various parts of gingiva?

(i) Free gingiva and gingival sulcus is mainly supplied by supraperiosteal arterioles.

(ii) Col is supplied mainly by vessels of periodontal ligament.

(iii) Attached gingiva is supplied by supraperiosteal arterioles, arterioles that emerge from the crest of the interdental septa and vessels of periodontal ligament.

Q.31 Describe the lymphatic drainage of periodontium.

(i) Mandibular incisors region drains into - Submental lymph nodes

(ii) Buccal gingiva of maxilla, Buccal and lingual gingiva of mandibular premolar and molar region - Submandibular lymph nodes

(iii) Third molar region - Jugulodigastric lymph nodes.

Q.32 Which factors determine the colour of gingiva?

(i) Vascular supply

(ii) Thickness of epithelium

(iii) Degree of keratinization of epithelium

(iv) Presence of pigment containing cells.

Q.**33 Classify gingival pigmentation.**

Gingival pigmentation was classified according to modification of melanin index.

Category 0: No pigmentation
Category 1: Solitary unit(s) of pigmentation in papillary gingiva without formation of continuous ribbon between solitary units.
Category 2: At least 1 unit of formation of continuous ribbon extending from two neighbouring solitary units.

Q.34 Which factors determine the contour of gingiva?

(i) Shape of the teeth
(ii) Teeth alignment in the arch
(iii) Location and size of the area of proximal contact
(iv) Dimensions of the facial and lingual gingival embrassures.

Q.35 Which factors determine the shape of interdental gingiva?

(i) Contour of the proximal tooth surface
(ii) Location and shape of the contact area
(iii) Dimensions of the gingival embrassures.

Q.36 What is the oxygen consumption of normal gingiva?

$QO_2^{(oxygen)}$ 1.6 ± 0.37

The respiratory activity of epithelium is approximately 3 times greater than that of connective tissue and the respiratory activity of the sulcular epithelium is approximately twice that of whole gingiva.

Q.37 Name various coatings present on tooth.

A. Developmental origin -
(i) Reduced enamel epithelium
(ii) Coronal cementum
(iii) Dental cuticle
(iv) Subsurface enamel matrix

B. Acquired coating -
(i) Salivary pellicle
(ii) Bacterial coating
(a) Plaque
(b) Material alba
(iii) Calculus
(iv) Subsurface pellicle
(v) Surface stains.

B. PERIODONTAL LIGAMENT

Q.1 What is the width of periodontal ligament?

0.15 to 0.38 mm

Q.2 What is the shape of periodontal ligament space?

Hour-glass shape: Periodontal ligament is thinnest at the axis of rotation in the middle and widens coronally and apically.

Q.3 What are the constituents of periodontal ligament space?

A. Periodontal ligament fibres
B. Cellular elements
C. Ground substances
   (i) Glycosaminoglycans
   (ii) Glycoproteins.

Q.4 Write functions and position of each group of periodontal ligament fibres.

1. Transseptal group-
   Functions:
   (i) Reconstructed even after destruction of the alveolar bone has occurred in the periodontal disease.
   (ii) Responsible for returning teeth to their original state after orthodontic therapy.
   Position:
   Extends interproximally over alveolar bone crest and embedded in the cementum of adjacent teeth.
2. Alveolar crest group-
   Functions:
   (i) Prevent extrusion
   (ii) Prevent lateral tooth movements
   Position:
   Extends obliquely from the cementum just beneath the junctional epithelium to the alveolar crest.
3. Horizontal group-
   Position:
   Extends at right angles to the long axis of the tooth from cementum to alveolar bone.
4. Oblique group-
   Functions:
   (i) Bear vertical masticatory stresses
   (ii) Transform vertical stress into tension on the alveolar bone.
   Position:
   Extends from the cementum in a coronal direction obliquely to the bone.
5. Apical group-
   Functions:
   (i) Prevents tooth tipping
   (ii) Resists luxation
   (iii) Protects neurovascular supply to the tooth.
   Position:
   It radiates in irregular fashion from cementum to bone at apical region of socket.

6. Interradicular group-

Functions:

(i) Prevents luxation

(ii) Prevents tooth tipping and torquing.

Position:

Found only between roots of multirooted tooth running from cementum into bone, forming crest of interradicular septum.

Q.5 Which is the largest group of periodontal ligament fibres?

Oblique group of periodontal ligament fibres.

Q.6 Which types of collagen is present in periodontal ligament?

Type I, III, V, VI, XII (FACIT).

Q.7 What are the various cells of periodontal ligament?

(i) Connective tissue cells:

(a) Synthetic cells
- Osteoblasts
- Fibroblasts
- Cementoblasts

(b) Resorptive cells
- Osteoclasts
- Fibroblasts
- Cementoclasts

(ii) Epithelial cells: Epithelial rests of malassez

(iii) Immune system cells:

(a) Neutrophils

(b) Lymphocytes

(c) Macrophages

(d) Mast cells

(e) Eosinophils

(iv) Cells associated with neurovascular elements

(v) Progenitor cells.

Q.8 What is the remarkable feature of Periodontal ligament (PDL) collagen?

There is rapid turnover rate of PDL collagen, with half life of only 10 – 15 days, which is about 5 times faster than gingival collagen, which in turn is renewed more rapidly than dermal collagen.

Q.9 What is Epithelial rests of Malassez and cementicles?

- Epithelial rests of Malassez – These are remnants of Hertwig's root sheath. They are present close to cementum through out the periodontal ligament and more in apical and cervical areas. When stimulated they proliferate and participate in the formation of periapical cysts and lateral root cysts.

- Cementicles – These are global masses of cementum arranged in concentric lamellae that lie free in the periodontal ligament or adhere to the root surface.

Q.10 Describe blood supply of periodontal ligament.

The blood supply is derived from the inferior and superior alveolar arteries and reaches the periodontal ligament from 3 sources:

(i) Apical vessels
(ii) Penetrating vessels from the alveolar bone
(iii) Anastomosing vessels from the gingiva

Q.11 What is the position of blood vessels in periodontal ligament space?

They are present in the interstitial spaces of loose connective tissue between the principal fibres which runs longitudinally connected in the net like plexus closer to the bone than cementum.

Q.12 How are the capillaries of periodontal ligament different from the capillaries of other connective tissues?

The capillaries of periodontal ligament are fenestrated while in other connective tissues they are continuous. Due to fenestration, they have greater ability of diffusion and filtration which is related to high metabolic requirements of periodontal ligament and its high rate of turnover.

Q.13 What are the functions of periodontal ligament?

(i) Supportive:
- Attaches the teeth to the bone.
- Transmit occlusal forces to the bone.
- Maintain gingival tissues in their proper relationship to the teeth.
- Resist the impact of occlusal forces acting as a shock absorber.
- Protect the blood vessels and nerves from injury by mechanical forces.

(ii) Sensory: Capable of transmitting
- Tactile
- Pressure
- Pain sensations by trigeminal pathways.

(iii) Nutritive: Supply nutrients to
- Cementum
- Bone
- Gingiva through blood vessels and lymphatics.

(iv) Homeostatic/formative: Helps in the formation and resorption of
- Cementum
- Bone during physiologic tooth movement and repair of injuries.

C. CEMENTUM

Q.1 What is cementum?

Cementum is calcified avascular mesenchymal tissue that forms the outer covering of the anatomic root.

Q.2 Who first demonstrated cementum microscopically?

Two pupils of Purkinje in 1835.

Q.3 Which types of collagen are present in cementum?

Type I – 90%, III, V, XII (FACIT), XIV

Q.4 What are the sources of collagen fibres of cementum?

(i) Fibroblasts which produce extrinsic sharpey's fibers.

(ii) Cementoblasts which produce intrinsic fibres of the cementum matrix.

Q.5 Write biochemical composition of cementum.

(i) Inorganic: 40 - 50% – Hydroxyapatite

(ii) Organic: 50%

- Collagen: Type I, III, V, XII, XIV
- Non-collagenous: Fibronectin, bone sialoprotein, osteopontin, osteocalcin, osteonectin, alkaline phosphatase
- Formative cells: Cementoblast
- Degradative cells: Cementoclast / Odontoclast
- Adhesion molecule: Cementum attachment protein
- Growth factor: Insulin like growth factor

Q.6 Classify cementum.

Schroeder classified cementum as:

(i) Acellular afibrillar cementum (AAC): 1-15 μm. It consists of only mineralized ground substance, which is a product of cementoblasts. Cells, collagen (extrinsic and intrinsic) fibres are absent. It forms coronal cementum.

(ii) Acellular extrinsic fibre cementum (AEFC): 30-230 μm. It consists of sharpey's fibers and lacks cells, which is a product of fibroblasts and cementoblasts. It is found in cervical third of the root.

(iii) Cellular mixed stratified cementum (CMSC): 100-1000 μm. It consists of extrinsic and intrinsic fibers and cells which is a product of fibroblasts and cementoblasts. It is found in apical third of roots and in furcation areas.

(iv) Cellular intrinsic fibre cementum (CIFC): It consists of cells and intrinsic fibres, lacking extrinsic collagen fibres, which is a product of cementoblasts. It fills resorption lacunae.

(v) Intermediate cementum: It consists of cellular remnants of Hertwig's sheath embedded in calcified ground substance. It is present near cementodentinal junction.

Q.7 Difference between Acellular and Cellular Cementum.

		Acellular Cementum	*Cellular Cementum*
1	Formation	Forms before tooth reaches occlusal plane	Forms after tooth reaches occlusal plane
2	Cells	Does not contain any cells	Contains cementocytes
3	Location	Coronal portion of the root	Apical portion of the root
4	Rate of formation	Slower	Faster
5	Incremental lines	More	Sparse
6	Function	Forms after regenerative periodontal surgical procedure	Contributes to the length of root during growth
7	Calcification	More calcified	Less calcified
8	Sharpey's fibres	More	Less
9	Regularity	Regular	Irregular
10	Thickness	20 - 50 μm near the cervix, 150 - 200 μm near the apex	Thickness of 1 to several mm

Q.8 What is intermediate cementum?

It is an ill defined zone near the cementodentinal junction of certain teeth that appears to contain cellular remnants of Hertwig's epithelial root sheath embedded in a calcified ground substance. It is also called as Layer of Hopewell Smith.

Q.9 What is the thickness of cementum?

In Coronal third — 16 - 60 μm (Thickness of hair)
In Apical third — 150 - 200 μm (Thickest)
Thicker in distal surfaces than mesial surfaces.

Q.10 Write normal variations in tooth morphology at CEJ.

- In about 60 - 65% — Cementum overlaps enamel.
- In about 30% — Edge to edge butt joint.
- In about 10% — Space present between cementum and enamel.

Q.11 Which type of cementum is desired following regenerative periodontal surgical procedure?

Acellular extrinsic fibre cementum.

Q.12 Name the conditions/diseases in which cementum formation is altered.

(i) Paget's disease – hypercementosis
(ii) Hypopituitarism – decrease cementum formation
(iii) Cleidocranial dysplasia – defective cementum formation
(iv) Hypophosphatasia – no cementum formation.

D. ALVEOLAR BONE

Q.1 What is alveolar process?

It is the portion of maxilla and mandible that forms and supports the tooth sockets (alveoli).

Q.2 What are the parts of alveolar process?

(i) Compact bone: External plate of cortical bone
(ii) Alveolar bone proper: Inner socket wall, Cribriform plate, Lamina dura, Bundle bone.
(iii) Trabecular bone: Cancellous trabeculae which is present between these two compact layers. It is also called as supporting alveolar bone.

Q.3 Write chemical composition of bone.

A. Inorganic: 67% Hydroxyapatite
B. Organic: 33%
- Collagen – 28%
- Non-collagenous protein – 5%.

Q.4 Which types of collagen are present in the alveolar bone?

Type I - Mainly
Type III, V, XII, XIV – Traces**.**

Q.5 Write various Non-collagenous proteins in Bone Matrix.

(i) Osteocalcin
(ii) Osteonectin
(iii) Osteopontin
(iv) Bone sialoprotein
(v) Bone proteoglycan biglycan
(vi) Bone proteoglycan II decorin
(vii) Thrombospondin
(viii) Bone morphogenetic proteins (BMPs).

Q.6 What are the functions of osteoblast?

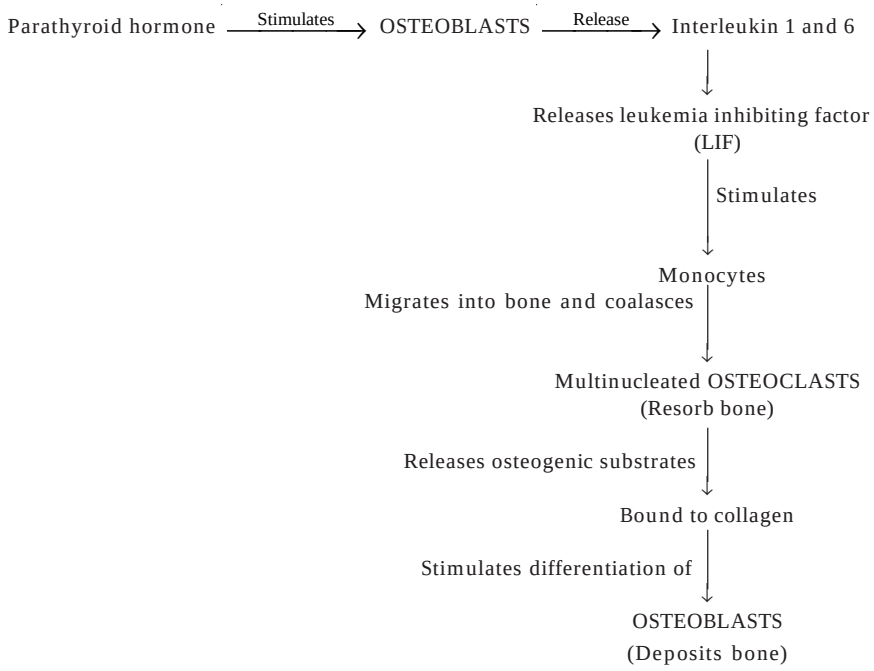
(i) Bone formation: Synthesize organic matrix of bone.
(ii) Cell to cell communication and maintenance of bone.
(iii) Bone resorption: Produces proteases, which are involved in matrix degradation and matrix maturation.
(iv) Produces:
- Type I collagen
- Non-collagenous protein
- Osteocalcin
- Osteopontin
- Osteonectin
- Various proteoglycans.

Q.7 Which are the enzymes released by Osteoclast?

(i) Acid phosphatase
(ii) Aryl sulfatase
(iii) β-glucoronidase
(iv) Several cysteine proteinase such as cathepsin B and L
(v) Tissue plasminogen
(vi) Activator TPA
(vii) MMP-I
(viii) Lysosymes.

Q.8 What is coupling?

It is interdependency of osteoblasts and osteoclasts in remodeling of the bone.



Q.9 What are various bone resorbing factors?

A. Systemic factors:
   (a) Parathyroid hormone
   (b) Parathyroid related peptide
   (c) Vitamin D_3
   (d) Thyroid hormone

B. Local factors:
   (a) Prostanoids
   (b) Lipoxygenase metabolites
   (c) Cytokines: IL – 1, IL – 4, TNF – α, TNF – β, IL – 6

C. Growth factors:
   (a) EGF
   (b) TGF – α
   (c) TGF – β
   (d) PDGF

D. Bacterial factors:
   (a) Lipopolysaccharides
   (b) Capsular material
   (c) Peptidoglycans
   (d) Lipoteichoic acids.

Q.10 Difference between woven, lamellar bone and bundle bone.

- Woven bone: The interwined collagen fibrils are oriented in many directions showing wide interfibrillar spaces. More non-collagenous matrix, proteins and cementocytes are present. It has low biomechanical strength. It is formed very quickly.
- Lamellar bone: Collagen fibres are thicker and arranged in ordered sheets consisting of aligned and closely packed fibrils. Less non-collagenous matrix, proteins and cementocytes are present. It is formed very slowly.
- Bundle bone: It is the bone adjacent to periodontal ligament, which contains great number of Sharpey's fibres. It is localized within alveolar bone proper consisting of thin lamellae arranged in layers parallel to the root, with intervening appositional lines.

Q.11 What is fenestration and dehiscence?

- Fenestration- Isolated areas in which root is denuded of bone and root surface is covered by gingiva and periosteum, where marginal bone is intact.
- Dehiscence- When the denuded areas extend through the marginal bone, the defect is called dehiscence.

Q.12 What is bone modeling and bone remodeling?

- Bone modeling: The process by which the overall size and shape of bone is established is called as bone modeling. It extends from embryonic bone development to the pre-adult period of human growth, which is continuous and covers a large surface.
- Bone remodeling: The replacement of old bone by new is called bone turnover remodeling. It does not stop when adulthood is reached, although its rate slows down, which is cyclical and usually covers a small area.

Q.13 What is periosteum and endosteum?

- Periosteum consists of an inner layer of osteoblasts surrounded by osteoprogenitor cells, which have the potential to differentiate

into osteoblasts, and an outer layer rich in blood vessels and nerves and composed of collagen fibres which penetrate the bone, binding the periosteum to the bone.

- Endosteum is composed of a single layer of osteoblasts and a small amount of connective tissue. The inner layer is the osteogenic layer and the outer is the fibrous layer.

Ageing and Periodontium

Q.1 What are the various age changes of periodontium?

A. Gingiva -
- (a) Gingival epithelium:
  - (i) Thinning and decreased keratinization
  - (ii) Rete pegs flatten
  - (iii) Migration of junctional epithelium to more apical position.
- (b) Gingival connective tissue:
  - (i) Increased rate of conversion of soluble to insoluble collagen
  - (ii) Increased mechanical strength of collagen
  - (iii) Increased denaturing temperature of collagen
  - (iv) Decreased rate of synthesis of collagen
  - (v) Greater collagen content.

B. Periodontal ligament -
- (i) Decreased number of fibroblasts
- (ii) More irregular structures
- (iii) Decrease organic matrix production
- (iv) Decrease epithelial cell rests
- (v) Increased amount of elastic fibers.

C. Cementum -
- (i) Increase in cemental width
- (ii) Decrease in permeability.

D. Alveolar bone -
- (i) Osteoporosis
- (ii) Reduction in bone metabolism
- (iii) Decreased vascularity
- (iv) Decreased healing capacity
- (v) Ability of alveolar bone to withstand occlusal forces decreases after the age of 30.

Classification of Periodontal Diseases

Q.1 Who was the first author to clearly distinguish various forms of periodontal disease?

Gottlieb

Q.2 What were the problems associated with 1989 AAP classification?

(i) It did not include gingivitis / gingival disease category.
(ii) Periodontitis categories had non-validated age dependent criteria.
(iii) There was extensive crossover in rates of progression of the different categories of periodontitis. Rapidly progressive periodontitis was a heterogenous category.
(iv) There was extensive overlap in the clinical characteristics of the different categories of periodontitis.
(v) Refractory periodontitis was a heterogenous category.
(vi) Prepubertal periodontitis was a heterogenous category.

Q.3 What are the main changes in 1999 AAP classification?

(i) The addition of a comprehensive section on gingival diseases.
(ii) The replacement of the term adult periodontitis with chronic periodontitis since epidemiological evidence suggests that chronic periodontitis may also be seen in adolescents.
(iii) The elimination of separate categories of rapidly progressive periodontitis and refractory periodontitis because of the lack of evidence that they represent separate conditions.
(iv) Replacement of the term early onset periodontitis with aggressive periodontitis largely because of the clinical difficulties in determining the age of onset in many of these cases. The author of this new classification also questions the use of the term juvenile periodontitis for the same reasons. They have replaced the term with localized and generalized aggressive periodontitis.
(v) A new classification group of periodontitis as a manifestation of systemic disease has been created and this includes those cases of prepubertal periodontitis directly resulting from known systemic diseases.

(vi) There are also new group categories of abscesses of periodontium, periodontic – endodontic lesions and developmental / acquired deformities / conditions.

Q.4 Classify periodontal diseases and conditions.

(i) Gingival diseases
- Plaque–induced gingival diseases
- Non–plaque–induced gingival diseases

(ii) Chronic Periodontitis
- Localized
- Generalized

(iii) Aggressive Periodontitis
- Localized
- Generalized

(iv) Periodontitis as a manifestation of systemic diseases

(v) Necrotizing periodontal diseases
- Necrotizing ulcerative gingivitis (NUG)
- Necrotizing ulcerative periodontitis (NUP)

(vi) Abscesses of the periodontium
- Gingival abscess
- Periodontal abscess
- Pericoronal abscess

(vii) Periodontitis associated with endodontic lesions
- Endodontic–periodontal lesion
- Periodontal–endodontic lesion
- Combined lesion

(viii) Developmental or acquired deformities and conditions
- Localized tooth–related factors that predispose to plaque–induced gingival diseases or periodontitis
- Mucogingival deformities and conditions around teeth
- Mucogingival deformities and conditions on edentulous ridges
- Occlusal trauma.

Epidemiology

Q.1 Explain various Gingival indices.

Name	*Year*	*Authors*	*Method*
Papillary – marginal attached index (PMAI)	1947	Schour and Massler	Observes; press probe against gingiva
Gingival index (GI)	1963	Loe and Silness	Observes; perform circumferential stroke against soft tissue below gingival margin

Q.2 What are the advantages of Modified Gingival Index (MGI) over Gingival Index (GI)?

(i) MGI is a non–invasive index.
(ii) MGI is more sensitive than GI.

Q.3 Explain various Bleeding indices.

Name	*Year*	*Authors*	*Method*	*Scale*
Gingival sulcus bleeding index (SBI)	1971	Muhlemann and Son	Perform sulcus probing on dry teeth	Ordinal (0 to 5; score $\geq$2 on color if bleeding occured)
Gingival bleeding index	1975	Ainamo and Bay	Perform circumferential stroke at gingival orifice; wait 10 seconds	Dichotomous
Papillary bleeding score (PBS) modified from PBI	1979	Loesche	Insert STIM – U – DENT interproximally	Ordinal (0 to 5)

Q.4 What are the advantages and disadvantages of gingival bleeding index?

Advantages:

(i) It is based on objective diagnostic sign of inflammation.
(ii) Easy reproducible assessment of gingival status.
(iii) Detect early inflammatory changes, which occur before any changes in gingival color, form and texture.

(iv) Can be used to enhance patient's motivation for plaque control as it is easily understood by the patient.

(v) Detect presence of inflammatory lesions located at the base of periodontal pocket, an area which is inaccessible to visual examination.

Disadvantages:

(i) Types of probe, angulation depth and force of probing may vary and may bring discrepancy in the results.

(ii) Bleeding from gingival sulcus may be associated with other forms of periodontal disease, not only gingivitis.

Q.5 Explain various Plaque indices.

Name	*Year*	*Authors*	*Method*	*Area evaluated*
Quigley-Hein plaque index	1962	Quigley and Hein	Disclose	Surface scored from 0 to 5, with emphasis at gingival margin
Simplified Oral Hygiene index (OHI-S)	1964	Greene and Vermillion	Perform same as OHI with different teeth selected	Facial surfaces of #3,#8,#14,#24 and lingual surfaces of #19 and #39
Plaque index (PI)	1964	Silness and Loe	Dry teeth; use mouth mirror and explorer	Gingival 1/3rd of tooth surface or tooth
Patient hygiene Performance (PHP)	1968	Podshadley and Haley	Disclose and record presence or absence of plaque	#3, #8, #14, #19, #24, #30
Turesky modification of the Quigley-Hein	1970	Turesky, Gilmore and Glickman	Disclose	Plaque assessment on facial and lingual surfaces of all teeth
Plaque control record	1972	O'leary, Drake and Naylor	Record presence of plaque to allow patient to visualize areas	4 tooth surfaces of all teeth present
Navy plaque index (modified; MN)	1972	Elliot, Bowers and Rovelstad	Disclose score 1 (plaque present) for each 9 areas	9 divisions of tooth surface with more divisions at gingival margin

Q.6 What are the advantages of patient hygiene performance index (PHP)?

(i) It is easy to use and can be performed.

(ii) It is first index developed for the sole purpose of assessing an individual performance.

(iii) Used as educational and motivational tool for patient.

Q.7 Which disclosing agent is used in plaque component of Periodontal disease index (PDI) and Turesky – Gilmore – Glickman Plaque index?

Plaque component of PDI: Bismarck brown solution

Turesky – Gilmore – Glickman plaque index: Basic fuchsin.

Q.8 Explain various Calculus indices.

Name	*Year*	*Authors*	*Method*
Calculus surface index	1961	Ennever, Sturzenberger and Radlike	Use air, mirror and explorer to detect calculus
Calculus index simplified (CI-S), part of OHI-S	1964	Greene and Vermillion	With an explorer, detect calculus on tooth surface or around cervical portion of tooth
V-M Calculus assessment	1965	Volpe, Manhold and Hazen	Measure with probe in three planes
Marginal line calculus index (MLC-I)	1967	Muhlemann and Villa	Divide tooth in half (mesial and distal); with air, visualize minute areas of supramarginal calculus next to gingiva on lingual four mandibular incisors

Q.9 Explain various Periodontal disease indices.

Name	*Year*	*Authors*	*Method*
Periodontal index (PI)	1956	Russell	Do not use probe; weight scores and combine gingival and periodontal status
Periodontal disease index (PDI)	1967	Ramjford	Select the "Ramjford" teeth (#3, #9, #12, #19, #25 and #28) and score for gingiva, attachment loss, calculus and plaque
Periodontal screening examination	1967	O'Leary	Divide mouth into 6 segments and record highest score; score gingiva by color, contour and consistency; score periodontium by mesiofacial line angle probe depth; score local irritants
Community periodontal index treatment needs (CPITN)	1982	Ainamo et al	This index is for epidemiological purposes. Use O'leary's sextants with specified index teeth or worst tooth, WHO probe, 0 to 4 codes per sextant; evaluate bleeding, deposits and pocket depth
Extent and severity index	1986	Carlos, Wolfe and Kingman	This index is for epidemiological purposes. Estimates the attachment level from probe depths-14 sites in each of 2 contralateral quadrants
Periodontal scoring and recording index (PSR)	1992	AAP, ADA	This is an individual screening exam. Divide the mouth into 6 segments; record highest score according to 4 levels, including bleeding and probe depths

Q. 10 Which epidemiologic index has true biologic gradient?
Periodontal index (PI) by Russell.

Periodontal Microbiology

A. BIOFILMS

Q.1 What is biofilm?

The term biofilm describes relatively undefinable microbial community associated with a tooth surface or any other hard non-shedding material.

Q.2 Describe the structure of biofilms.

In biofilms, microcolonies of bacterial cells are distributed in glycocalyx matrix. Water channels are present between the microcolonies which permits passage of nutrients.

Q.3 Where are biofilms found?

(i) Oral cavity
(ii) The bottom of boats and docks
(iii) Inside pipes
(iv) Rocks in streams
(v) Catheters
(vi) Hip and voice prosthesis
(vii) Contact lenses.

Q.4 What are the methods of transferring information in a biofilm?

(i) Quorum sensing
(ii) Conjugation
(iii) Transformation
(iv) Plasmid transfer
(v) Transpoon transfer.

Q.5 What is Quorum Sensing?

It involves the regulation of expression of specific genes through the accumulation of signaling compounds that mediate intercellular communication.

Q.6 What is the significance of biofilm?

(i) Organisms in a biofilm are 1000-1500 times more resistant to antibiotics than in their planktonic/unattached state.

(ii) Slower rate of growth of organisms in a biofilm makes them less susceptible to antibiotics.
(iii) Biofilm acts as a barrier to the diffusion of antibiotics.
(iv) Extracellular enzymes such as β-lactamase, formaldehyde dehydrogenase become concentrated in extracellular matrix, thus inactivating some antibiotics.

Q.7 What are the functions of biofilms?

They provide:
(i) Protection
(ii) Facilitate processing and uptake of nutrients
(iii) Cross feeding, one species providing nutrients for another species
(iv) Development of an appropriate physico-chemical environment

B. DENTAL PLAQUE

Q.1 What is dental plaque?

According to WHO in 1978, it is defined as specific but highly variable structural entity resulting from colonization and growing microorganisms on surfaces of teeth and consisting of numerous microbial species and strains embedded in an extracellular matrix.

Q.2 Write composition of plaque.

A. Microorganisms: 500 distinct species:
- Bacterial
- Non-bacterial
  (a) Mycoplasma
  (b) Yeasts
  (c) Protozoa
  (d) Viruses

B. Host cells:
- Epithelial cells
- Macrophages
- Leukocytes

C. Organic compounds:
- Polysaccharides
- Proteins
- Glycoproteins
- Lipid materials

D. Inorganic compounds:
- Calcium
- Phosphorous
- Fluoride
- Sodium
- Potassium

Q.3 How many bacteria are present in 1 mg of dental plaque?
Approximately 2×10^8 bacteria.

Q.4 Write the steps in the formation of dental plaque.
(i) Formation of dental pellicle
(ii) Initial colonization of microorganisms
(iii) Secondary colonization and maturation of microbes.

Q.6 What are the various plaque hypothesis?
(A) Non-specific plaque hypothesis: Described by Walter Loesche in 1976. According to this hypothesis, periodontal disease results from the elaboration of noxious products by the entire plaque flora.
Shortcomings:
- Some individuals with constant amount of plaque and calculus never developed destructive periodontitis.
- Some sites were not affected, whereas advanced disease was found in adjacent sites.

(B) Specific plaque hypothesis: Described by Walter Loesche in 1976. According to this hypothesis, only certain plaque is pathogenic and its pathogenicity depends on the presence of or increase in specific microorganisms, as in the case of well-known exogenous bacterial infections of man such as TB, Syphilis.
Shortcomings:
- There were occasions when either disease was diagnosed in the absence of the putative pathogens or when pathogens are present with no evidence of disease.

(C) Modern version of specific theory: Described by Socransky in 1979. According to this theory, 6-12 bacterial species may be responsible for the majority of cases of destructive periodontitis and additional species may be responsible for small number of other cases.

(D) Unified theory: Described by Theilade in 1986. It is the modern version of non–specific and specific plaque hypothesis. According to this theory all bacterial plaque may contribute to the pathogenic potential of the subgingival flora to a greater or lesser extent. This is due to its ability to colonize and evade host defenses and provoke inflammation and tissue damage.

(E) Ecological plaque hypothesis: According to this, any change in the nutrient status of a pocket, i.e.physical and chemical change to the habitat are considered the primary cause for overgrowth of pathogens.

Q.7 What are the types, components and functions of pellicle?
Types:
(a) Unstained pellicle

(b) Stained pellicle
(c) Subsurface pellicle

Components:

(a) Glycoproteins (Mucins)
(b) Proline rich protein
(c) Phosphoproteins (Statherin)
(d) Histidine–rich proteins
(e) Enzymes (Amylase)

Functions:

(a) Protective – It acts as lubricant material
(b) Attachment of calculus – One mode of calculus adhesion is by pellicle
(c) Nidus for bacteria – Participates in plaque formation by aiding in the adherence of bacteria.
(d) Salivary glycoprotein and salivary calcium phosphate are adsorbed on the enamel surface and help to reduce toothwear.

Q.8 What are the types of dental plaque?

A. Supragingival
B. Subgingival
   (a) Tooth associated
   (b) Tissue associated
C. Peri-implant

Q.9 Name tooth and tissue associated plaque microorganisms.

A. Tooth associated plaque microorganisms:
   (i) *Streptococcus mitis*
   (ii) *S. sanguis*
   (iii) *A. viscosus*
   (iv) *A. naeslundii*
   (v) *Eubacterium*
B. Tissue associated plaque microorganisms:
   (i) *S. oralis*
   (ii) *S. intermedius*
   (iii) *P. micros*
   (iv) *P. gingivalis*
   (v) *P. intermedia*
   (vi) *Tannerella forsythia*
   (vii) *F. nucleatum*

Q.10 Difference between Supragingival and Subgingival plaque.

	Supragingival plaque	*Subgingival plaque*
1 Location	Coronal to the margin of free gingiva	Apical to free gingiva
2 Origin	Salivary glycoprotein and salivary microorganisms	Downgrowth of bacteria from supragingival plaque
3 Distribution	Areas left uncleaned, cervical third and promixal surfaces.	Attached plaque covers calculus and unattached plaque extends to the periodontal attachment
4 Retention	Rough surface of teeth or restoration, malpositioned teeth and carious lesion	Overhanging margins Periodontal pockets
5 Structure	Adherent, densely packed microbial layer over pellicle on tooth surface	Tooth surface attached plaque, unattached plaque, epithelium attached plaque
6 Microorganisms	Early plaque: Gram positive cocci Older plaque: • 3–4 days: Increased no. of filaments and fusiforms • 4–9 days: More complex flora with rods, filamentous forms • 7–14 days:Vibrios, Spirochetes	Anaerobic population. Diseased pocket; Gram negative, motile, spirochetes, rods
7 Source of nutrients for bacterial proliferation	Saliva and ingested food	GCF, exudate, leukocytes
8 Significance	Causes gingivitis, supragingival calculus, dental caries	Causes gingivitis, periodontal infection, subgingival calculus

Q.11 What is the significance of site specificity of plaque?

(i) Marginal plaque is of prime importance in the development of gingivitis.

(ii) Supragingival plaque and tooth associated plaque are critical in calculus formation and root caries.

(iii) Tissue associated subgingival plaque is important in the soft tissue destruction that characterize different forms of periodontitis.

Q.12 What are the types of colonies of various periodontal pathogens?

(i) Aggregatibacter actinomycetemcomitans: Convex shaped colonies with star shaped center.

(ii) Eikenella corrodens: Corroding / pitting colonies.

(iii) Campylobacter rectus: Convex, dry spreading colonies.

(iv) Capnocytophaga: Flat, glistening colonies.

(v) Bacteroides: Black pigmented colonies.

Q.13 What are the various species of Bacteroides?

(i) Asaccharolytics: *P. gingivalis*
(vi) Intermediate level of carbohydrate fermenter: *P. intermedia*
(vii) Highly saccharolytic: *Prevotella melaninogenica.*

Q.14 Name bacteria which invade epithelial cells.

(i) *P. gingivalis*
(ii) Aggregatibacter actinomycetemcomitans
(iii) Spirochetes (*T. pallidum*)
(iv) *F. nucleatum*

Q.15 Name bacteria which are motile.

(i) Spirochetes
(ii) Campylobacter rectus
(iii) Selemonas.

Q.16 Name bacteria which produces leukotoxin.

(i) Aggregatibacter actinomycetemcomitans
(ii) Campylobacter rectus.

Q.17 What is chemokine paralysis?

According to Darveau, *P. gingivalis* inhibit the production of IL-8 by epithelial cells which is chemotaxin for PMNs, so it inhibits PMN migration and this is called chemokine paralysis.

Q.18 Who discovered Actinobacillus actinomycetemcomitans?

Actinobacillus actinomycetemcomitans was first isolated by German microbiologist Klinger, 1912 from lesion of cervicofacial actinomycosis. The microorganism was isolated together with Actinomyces *israelli***.** Hence, the species name actinomycetemcomitans means together with Actinomyces. Genus name *Actinobacillus*, "actino" referring to star-shaped internal morphology of the colonies and bacillus referring to cell shape (rod-shaped).

Q.19 Write criteria for judging periodontal microorganisms as potential pathogens.

Sigmund Socransky, a researcher at the Forsyth Dental Centre in Boston, proposed criteria by which periodontal microorganisms may be judged to be potential pathogens.

According to these criteria, a potential pathogen must have:

(a) Association – Be associated with disease, as evident by increase in the number of organisms at diseased sites.
(b) Elimination – Be eliminated or decreased in sites that demonstrate clinical resolution of disease with treatment.
(c) Host Response – Demonstrate a host response, in the form of an alteration in the host cellular or humoral immune response.
(d) Animal Studies – Be capable of causing disease.

(e) Virulence factors – Demonstrate virulence factors responsible for enabling the microorganisms to cause destruction of the periodontal tissues.

Q.20 What are the various virulence factors of Aggregatibacter actinomycetemcomitans (A.a)?

(i) Collagenase
(ii) Cytotoxin
(iii) Leukotoxin
(iv) Bacteriocin
(v) Endotoxin
(vi) Fc binding protein
(vii) Invasins
(viii) Immunosuppresive factors
(ix) Adhesins
(x) Chemotaxis inhibitor.

Q.21 Who discovered Tannerella forsythia?

Tannerella forsythia was first isolated at the Forsyth Institute from subjects with progressing advanced periodontitis in the mid 1970s and was described as Fusiform Bacteroides by Tanner et al.

Q.22 What is HACEK?

It is a group of organisms which are associated with systemic diseases distant from the oral cavity. It includes - Haemophilus sp., A.a, *Cardiobacterium hominis, Eikenella corrodens, Kingella kingae*.

Q.23 Name microbes associated with Yellow, Purple, Green, Orange and Red complexes and write their significance.

According to Socransky:

- Yellow complex: *Streptococcus* sp., *S. sanguis, S. mitis, S. intermedius, S. oralis, S. gordonii*
- Purple complex: *Veillonella parvula* and *A. odontolyticus*
- Green complex: *Capnocytophaga sp., A.a serotype a* and *Eikenella corrodens*
- Orange complex: *Campylobacter gracilus, F. nucleatum, P. intermedia, P. micros, C. rectus*
- Red complex: *P. gingivalis, Tannerella forsythia, T. denticola.*

Significance:

Early colonizers include members of yellow complexes, purple complexes and green complexes. Orange complex members are thought to bridge early colonizers. Red complex members are associated with bleeding on probing and more dominant at late stages in plaque development. Green and orange complexes include species recognized as pathogens in periodontal and non–periodontal infection.

Q.24 Name microorganisms associated with Periodontal health, Chronic Gingivitis, Chronic Periodontitis, Localized Aggressive Periodontitis, Necrotizing periodontal disease and Abscesses of periodontium?

(i) Microorganisms associated with Periodontal health:
- *S. sanguis*
- *S. mitis*
- *Gemella sp.*
- *Atopobium sp.*
- *Capnocytophaga sp.*
- *Veillonella*
- *Streptococcus*

(ii) Microorganisms associated with Chronic Gingivitis / Dental plaque induced gingivitis:

Gram-positive organisms are:
- *S. sanguis*
- *S. mitis*
- *S. intermedius*
- *S. oralis*
- *A. viscosus*
- *A. naeslundii*
- *Peptostreptococcus micros.*

Gram negative organisms are:
- *F. nucleatum*
- *P. intermedia*
- *V. parvula*
- *Haemophilus*
- *Capnocytophaga*
- *Campylobacter sp.*

(iii) Microorganisms associated with Chronic Periodontitis:
- *P. gingivalis*
- *Tannerella forsythia*
- *P. intermedia*
- *C. rectus*
- *Eikenella corrodens*
- *F. nucleatum*
- *Aggregatibacter actinomycetemcomitans (A.a)*
- *P. micros*
- *Treponema*
- *Eubacterium sp.*
- *Herpes virus group*
- *EBV-1*
- *Human CMV*

(iv) Microorganisms associated with Localized Aggressive Periodontitis:
- A. a
- *P. gingivalis*
- *E. corrodens*
- *C. rectus*
- *F. nucleatum*
- *B. capillus*
- *Eubacterium brachy*
- *Capnocytophaga* sp.
- Spirochetes
- Herpes viruses including EBV-1
- Human CMV

(v) Microorganisms associated with Necrotizing periodontal disease:
- Spirochetes
- *P. intermedia*

(vi) Microorganisms associated with Abscesses of periodontium:
- *F. nucleatum*
- *P. intermedia*
- *P. gingivalis*
- *P. micros*
- *Tannerella forsythia*.

Q.25 Which organism is believed to be important in bridging between primary and secondary colonizers during plaque maturation?

Fusobacterium nucleatum

Q.26 Give examples of coaggregation.

A. Interaction of secondary colonizers with early colonizers:
Corn–cob and test tube brush are the examples of coaggregation
   a. *Fusobacterium nucleatum* with *Streptococcus sanguis*
   b. *Prevotella loescheii* with *Actinomyces viscosus*
   c. *Capnocytophaga ochraceus* with *A. viscosus*

B. Interaction among secondary colonizers:
   a. *F. nucleatum* with *P. gingivalis*
   b. *F. nucleatum* with *Treponema denticola*.

Q.27 What is Corn–cob structures and test tube brush/ bristle brush structures?

Corn–cob structure: Structures which have inner core of rod-shaped bacterial cells such as *F. nucleatum* and over the surface of which attach the coccal cells such as Streptococci or *P. gingivalis*.

Test tube brush structures: Are composed of filamentous bacteria to which gram negative rods adhere.

Q.28 Name bacteria which are called as beneficial species of the host.

(i) *Streptococcus*
(ii) *Capnocytophaga* sp.
(iii) *Veillonella.*

Q.29 How beneficial species of the host affect the disease progression?

(i) By passively occupying a niche that may otherwise be colonized by pathogens.
(ii) By actively limiting a pathogen's ability to adhere to appropriate tissue surfaces.
(iii) By adversely affecting the vitality or growth of a pathogen.
(iv) By affecting the ability of a pathogen to produce virulence factors.
(v) By degrading virulence factors produced by the pathogens.

Q.30 Name various ecosystems/niches present in the oral cavity.

(i) Supragingival hard surfaces – Teeth, implants, restorations, prostheses
(ii) Periodontal / Peri-implant pocket
(iii) Buccal epithelium, Palatal epithelium, Epithelium of the floor of the mouth
(iv) Dorsum of the tongue
(v) Tonsils.

Q.31 What are the various subgingival ecologic niches present in the oral cavity?

(i) Tooth / implant surface
(ii) Gingival crevicular fluid medium
(iii) Surface of epithelial cells
(iv) Superficial portion of the pocket epithelium.

Q.32 Describe various synergistic/agonistic and antagonistic metabolic interactions among different bacterial species found in plaque.

A. Synergistic / Agonistic interactions:
(i) Streptococcus and Actinomyces produces lactate and formate as metabolic byproducts which are used in the metabolism of Veillonella and Campylobacter respectively.
(ii) Veillonella produces menadione which is used by *P. gingivalis and P. intermedia.*
(iii) Campylobacter produces protoheme which is used by *P. gingivalis.*
(iv) P. gingivalis produces isobutyrate which is utilized by Treponema.
(v) Treponema and Capnocytophaga produces succinate which is used by *P. gingivalis.*

B. Antagonistic interactions:
   (i) *S. sanguis* produces H_2O_2 which kills A.a.
   (ii) A.a produces Bacteriocin which kills *S. sanguis*.

Q.33 Why is the etiology of periodontal diseases difficult to determine?

(i) Technical problems – Difficulty in sample taking, cultivation and identification of isolates.
(ii) Problems associated with the complexity of the microbiota- Periodontal infections are mixed infections, in which it is difficult to distinguish between secondary invaders and true pathogens.
(iii) Problems associated with the nature of periodontal disease- Periodontal disease appears to be episodic.
(iv) Difficulty in differentiation between active and inactive sites sampled for microbiologic studies.

Q.34 Write new names of various periodontal bacteria.

	Previous name	**Current name**
(i)	Bacteroides gingivalis	Porphyromonas gingivalis
(ii)	Bacteroides intermedius	Prevotella intermedia
(iii)	Bacteroides melaninogenicus	Prevotella melaninogenica
(iv)	Bacteroides forsythus	Tannerella forsythia
(v)	Wolinella recta	Campylobacter rectus
(vi)	Actinobacillus actinomycet-emcomitans	Aggregatibacter actinomycet-emcomitans

Calculus

Q.1 What is calculus?

Calculus is an adherent, calcified or calcifying mass that forms on the surfaces of teeth and dental appliances**.**

Q.2 What is the composition of calculus?

A. Inorganic contents

(a) Elements:

- Calcium – 39%
- Phosphorous – 19%
- Carbon dioxide – 1.9%
- Magnesium – 0.8 %
- Trace amounts of sodium, zinc, strontium, bromine, copper, manganese, tungsten, gold, aluminium, silicon, iron, fluorine.

(b) Compounds:

- Calcium phosphate – 75.9%
- Calcium carbonate – 3.1%
- Magnesium phosphate – traces
- Other metals – traces

(c) Crystals: 4 main forms

- Hydroxyapatite – 58%
- Magnesium whitlockite – 21%
- Octacalcium phosphate – 12%
- Brushite – 9%

B. Organic contents

(a) Carbohydrates – 1.9 – 9.1% (galactose, glucose, rhamnose, mannose, glucoronic acid, galactosamine, arabinose, galacturonic acid, glucosamine)

(b) Proteins – 5.9 to 8.2%

(c) Lipids – 0.2% (neutral fats, fatty acids, cholesterol, phospholipids, cholesterol esters)

(d) Protein polysaccharide complexes

(e) Desquamated epithelial cells

(f) Leukocytes
(g) Microorganisms

C. Bacterial content:
At periphery – Gram negative rods and cocci predominate. filamentous organisms, diptheroids, Bacterionema and Veillonella species are also present.

Q.3 What are the differences between supragingival and subgingival calculus?

	Supragingival Calculus	*Subgingival Calculus*
1 Color	White or whitish yellow	Dark brown/genuine black
2 Shape	Amorphous, bulky, shape of calculus is determined by anatomy of teeth, contour of gingival margin and pressure of tongue, lips, cheeks	Flattened to conform with pressure from the pocket wall, may be crusty, shiny, thin, finger and fern like
3 Consistency	Hard	Flint like, brittle
4 Attachment	Easily detached from tooth	Firmly attached to the tooth surface
5 Location	Coronal to the gingival margin	Below the crest of the marginal gingiva
6 Visibility	Visible in the oral cavity	Not visible on routine clinical examination
7 Composition	More brushite and octacalcium phosphate Less magnesium whitlockite Salivary proteins are present Sodium content is lesser	Less brushite and octacalcium phosphate More Magnesium whitlockite Salivary proteins are absent Sodium content increases with the depth of pocket
8 Source	Derived from salivary secretions	Formed from gingival exudate
9 Distribution	Symmetrical arrangement on teeth, more on facial surface of maxillary molars and lingual surface of mandibular anterior teeth due to openings of salivary glands ducts	Related to pocket depth, heaviest on proximal surface

Q.4 What are various forms of submarginal and subgingival calculus?

(i) Spicules – Small isolated pieces of calculus. These are frequently located at line angles and interdental areas.
(ii) Ledge – A larger deposit that forms on a section of the tooth and is approximately parallel to the CEJ.
(iii) Ring form – A ledge like deposit that encircles the tooth, forming a ring of calculus. In addition to calculus, roughness on the tooth surface may be caused by rough restorations, carious lesion, or necrotic cementum.

Q.5 Write theories related to mineralization of calculus.

(i) 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.

(ii) Epitactic / nucleation concept – Seeding agents induce small foci of calcification that enlarge and unite together to form calcified mass. 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.

(iii) Inhibition theory – Calcification as occurring only at specific sites because of the existence of an inhibiting mechanism at noncalcifying 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.

(iv) Transformation – Amorphous noncrystalline deposits and brushite can be transformed to octacalcium phosphate and then to hydroxyapatite.

Q.6 What are the modes of attachment of calculus to the tooth surface?

(i) Attachment by means of an organic pellicle

(ii) Mechanical locking into surface irregularities such as resorption lacunae and caries

(iii) Close adaptation of calculus undersurface depressions to the gently sloping mounds of the unaltered cementum surface

(iv) Penetration of calculus bacteria into cementum.

Q.7 What is the reversal phenomenon of calculus?

Reversal phenomenon is the decline from maximal calculus accumulation. It is due to vulnerability of bulky calculus to mechanical wear from food, cheeks, lips and tongue.

Q.8 Name various anti-calculus agents.

(i) First generation anti-calculus agents:

(a) Dissolution -
- Acid
- Sodium ricinoleate

(b) Plaque attachment -
- Silicone
- Ion exchange resins

(c) Plaque inhibition -
- Antibiotics (Niddamycin)
- Antiseptics (Chloramine T)

(d) Matrix disruption -
- Enzymes
- Ascoxal
- 30% urea

(ii) 2nd generation anti-calculus agents:
Inhibition of crystal growth
(a) Victamine C (chlomethyl analogue)
(b) Pyrophosphates
(c) Diphosphonates
(d) Zinc salts (chloride, citrate)
(e) Pyrophosphates and sodium fluoride
(f) Pyrophosphates, sodium fluoride, gantrez copolymer
(g) Citroxain and sodium citrate
(h) Calcium lactate.

Q.9 Write the causes of dental stains.

(i) Chromogenic bacteria:
- Black stains - Actinomyces sp. and Prevotella melaninogenicus
- Orange stains - Serratia marcescens
- Green stains - Penicillium and Aspergillus

(ii) Antibiotics: Tetracycline - yellow stain
(iii) Antimicrobial: Chlorhexidine - brown stain
(iv) Diseases:
- Erythroblastosis foetalis
- Porphyria

(v) Iron containing medicines: Black brown stains
(vi) Various vegetables, berries, coffee, tea, cola drinks, beets, red pepper, saffron.
(vii) Breakdown products of haemoglobin.

7 Defense Mechanisms of Gingiva

A. SALIVA

Q.1 What are the normal and xerostomic values of resting and stimulated saliva?

A. Resting saliva:
   (a) Normal: 0.3 - 0.4 ml / min
   (b) Xerostomia: less than 0.1ml / min

B. Stimulated saliva:
   (a) Normal: 1-2 ml / min
   (b) Xerostomia: less than 0.5 ml / min

Q.2 Write the composition of saliva.

A. Electrolytes:
   (a) Potassium
   (b) Sodium
   (c) Chloride
   (d) Bicarbonate
   (e) Calcium
   (f) Magnesium
   (g) Phosphorous

B. Organic:
   (a) Proteins -
      (i) Acinar cell families:
         - Mucin
         - Proline rich proteins and glycoproteins
         - Histatins and statherins
         - Cystatins
         - Amylase
         - Peroxidases
         - Carbonic anhydrases
      (ii) Ductal and stromal products:
         - Lactoferrin
         - Lysozyme
         - Secretory IgA

- Kallikrein
- Fibronectin
- Lipids
- Carbohydrates
- Sulfates

Q.3 What are the functions of Saliva?

I. **Role in oral health:**

A. Provide physical protection via mucin, glycoprotein

B. Lubrication via glycoprotein, mucin

C. Antibacterial action via -
- IgA
- Salivary amylase
- Lactoferrin – act against Actinobacillus and Streptococcus
- Salivary peroxidase act against Lactobacillus, Streptococcus and Veillonella species
- Proline rich proteins
- Lysozyme causes lysis of cell wall of A.a and Veillonella

D. Aids in tooth integrity via-
- Histatin
- Statherin
- Cystatin

E. Cleansing action due to its physical flow

F. Buffering action due to presence of urea and arginine rich protein. *Carbonic anhydrases* causes the reversible hydration of carbon dioxide leading to the formation of bicarbonate, which contributes to the buffering capacity of saliva.

G. Provide valuable data for diagnostic testing

H. Hasten blood coagulation and protect wounds from bacterial invasion due to presence of some coagulating factors in saliva

I. Taste perception

J. Digestion—
- Swallowing
- Food breakdown; chewing
- Food bolus formation

K. Speech

II. **Role in oral diseases:**

A. Formation of pellicle and plaque deposition

B. Aids in plaque mineralization to form calculus

C. Affect dental caries by cleansing mechanically and by direct antibacterial activity.

Q.4 What are the functions of Secretory IgA (sIgA) present in saliva?

It provides the first line of defense via immunologic means in the oral cavity.

(i) sIgA binds to microbes which inhibit their adherence to hard and soft tissue surfaces and thus hinder microbial invasion into deeper host tissues.

(ii) It plays important role in viral neutralization, attenuation of viral growth and replication on oral surfaces.

(iii) It neutralizes and disposes the toxins and food antigens.

Q. 5 What is the function of salivary peroxidase?

Lacto-thiocyanate – peroxidase enzyme catalyses the oxidation of thiocyanate ion (SCN^-) by H_2O_2, generating highly reactive, oxidized form of thiocyanate $OSCN^-$, causing direct toxicity to Streptococcus. It neutralizes deleterious effects of H_2O_2 produced by a number of oral microorganisms.

Q.6 What are organulocytes?

Living PMNs in saliva are called as organulocytes.

Q.7 Which coagulation factors are present in saliva?

Factor VIII, IX, X, Plasma Thromboplastin Antecedent (PTA) and Hageman factor.

Q.8 Which drugs cause xerostomia?

(i) Anticholinergic
(ii) Antipsychotic
(iii) Antiparkinsonism
(iv) Antidepressant
(v) Antihistaminic
(vi) Antihypertensive

B. GINGIVAL CREVICULAR FLUID (GCF)

Q.1 Write the various defense mechanisms of oral cavity.

(i) Saliva
(ii) Sulcular fluid
(iii) Intact epithelial barrier
(iv) High tissue turnover
(v) Presence of normal flora
(vi) Local antibody production
(vii) Migrating PMNs and other leukocytes

Q.2 What is the amount of GCF secreted per day?

0.5-2.4 µl / day

Q.3 What is the mean GCF volume in proximal spaces of anterior and molar teeth?

Anterior teeth — 0.24 – 0.43 µl
Molar teeth — 0.43 – 1.56 µl

Q.4 What is the composition of GCF?

A. Cellular elements:
   (i) Epithelial cells
   (ii) Leukocytes
   (iii) Bacteria

B. Electrolytes:
   (i) Sodium
   (ii) Potassium
   (iii) Calcium

C. Organic compounds:
   (i) Carbohydrates
   (ii) Proteins
      - Immunoglobulins
      - Complement components
   (iii) Lipids

D. Metabolic and bacterial products:
   (i) Lactic acid
   (ii) Hydroxyproline
   (iii) Prostaglandins
   (vi) Urea
   (v) Endotoxins
   (vi) Cytotoxic substances
   (vii) Antibacterial factors

E. Enzymes and enzyme inhibitors:
   (i) Acid phosphatase
   (ii) Alkaline phosphatase
   (iii) Pyrophosphatase
   (iv) β - glucuronidase
   (v) Lysozymes
   (vi) Hyaluronidase
   (vii) Proteolytic enzymes
      - Mammalian proteinases
      - Bacterial proteinases
      - Serum proteinases inhibitors
   (viii) Lactate dehydrogenase.

Q.5 Which are the various drugs excreted through GCF?

(i) Tetracyclines
(ii) Metronidazole

(iii) Clindamycin
(iv) Tinidazole
(v) Erythromycin.

Q.6 What is the normal ratio of B lymphocytes to T lymphocytes in GCF?
1:3 ratio

Q.7 What are the various methods of collection of GCF?
(i) Absorbing paper strip
  (a) Intracrevicular
  (b) Extracrevicular
(ii) Preweighed twisted threads
(iii) Sampling by means of micropipettes
(iv) Gingival washings
(v) Other strips
  (a) Plastic strips
  (b) Platinum loops.

Q.8 How the amount of GCF collected is evaluated?
(i) By direct viewing and staining: Stain the strip with ninhydrin and then measured.
(ii) By weighing: Strip is weighed before and after collecting the GCF sample.
(iii) By electronic device Periotron: Sample strip paper is inserted between the two jaws, which gives reading on the screen.

Q.9 What are the problems associated with GCF collection and data interpretation?
(i) Contamination – Usually sample is contaminated with blood, saliva or plaque.
(ii) Small sample size.
(iii) Sampling time – Prolonged sampling at the site results in protein concentrations approaching those of serum.
(iv) Volume determination – Evaporation is a significant problem in accurate volume determination of GCF samples.
(v) Recovery of strips – It depends on type of paper, binding of GCF protein to the filter paper and concentration of the original protein sample.

Immunity and Inflammation

Q.1 What is immunity?

It refers to the resistance exhibited by the host towards injury caused by microorganisms and their products.

Q.2 What is inflammation?

It refers to tissue injury or irritation, initiated by the entry of pathogens or of other irritants.

Q.3 What are microphages and macrophages?

Microphages are neutrophils, which differentiate almost completely within bone marrow in 14 days and retain their small size of 10 μm when exits from bone marrow and thus, called as microphages.

Macrophages are modified monocytes, which exits from bone marrow after 2 days and increase in size to about 22 μm and thus, are called as macrophages.

Q.4 What are the various receptors which are present on Mast cells, Neutrophils, Peripheral Dendritic cell, Monocytes, Lymphocytes and NK cells?

	Mast cells	*Neutrophils*	*Peripheral Dendritic cell*	*Monocytes*	*Lymphocytes T cell*		*NK Cell B cell*
1	Fc portion of IgE and IgG (FcCR, FcYR)	Fc portion of IgG and FcYR	MHC, Class II molecule	FcYR I, FcYR II, FcYR III	TCR T cell Ag receptor	BCR B cell Ag receptor	KIR (Killer inhibitory receptor)
2	C3a	CRI, CR3, CR4	CD1	CR1, CR3, CR4	CD4	MHC	KAR
3	C5a	C5aR	ICAM-1	C5aR	CD8	CLASS II	—
4	—	—	IFA-3	CD-1	—	—	—
5	—	—	Costimulatory factors B7-1, B7-2	MHC Class II receptors	—	—	—

Q.5 What are the various microbiocidal or inflammatory mediators stored in mast cells and neutrophils?

(i) Mast cells – possesses cytoplasmic granules called as lysosomes, which stores –
- (a) Histamine
- (b) Heparin
- (c) Slow reacting substances of anaphylaxis SRS – A
- (d) Tumor necrosis factor TNF-α
- (e) Leukotriene C4
- (f) Neutrophilic chemotactic factor
- (g) Eosinophil chemotactic factor

(ii) Neutrophil – posseses 2 types of granules

A. Specific granules contains:
- (a) Lysozyme
- (b) Lactoferrin
- (c) B-12 binding protein Cobalophilin

B. Azurophilic granules:
- (a) α-defensins (HNP-1, HNP-2, HNP-3, HNP-4)
- (b) Serprocidin, elastase, proteinase 3, azurocidin
- (c) Cathepsin G
- (d) Lysozyme.

Q.6 What are the inflammatory actions of various chemical mediators?

	Action	**Mediators**
(i)	Vasoconstriction	Thromboxane A_2 Leukotrienes C_4, D_4, E_4
(ii)	Vasodilatation	PGI_2, PGE_1, PGE_2, PGD_2, Bradykinin
(iii)	Increased permeability	Leukotrienes C_4, D_4, E_4 Histamine, SRS - A Bradykinin
(iv)	Chemotaxis	Leukotriene B4, HETE, Lipoxin,
(v)	Leukocyte adhesion	Bradykinin
(vi)	Collagenase activity	α2-macroglobin

Q.7 What are the actions of various cytokines?

	Action	**Cytokines**
(i)	Pro-Inflammatory	IL-1, IL-6, IL-8, TNF-α, IFN-α
(ii)	Anti-Inflammatory	IL-4, IL-10, IL-13, TGF-α
(iii)	Scarring	IL-6, TGF-α
(iv)	Anti-scarring	IL-10
(v)	Angiogenic	IL-8, Angiogenins, VEGF
(vi)	Antiangiogenic	IL-10

Q.8 What is opsonization?

Opsonization refers to the process of coating a particle with recognizable molecules to enable phagocytic ingestion.

Two types of opsonins are:

(i) Complement metabolite; C3b
(ii) Immunoglobin (IgG)

Q.9 What is chemotaxis?

It is the directed movement of a cell along a chemical gradient. It is a receptor mediated event that is initiated by chemotactic factors forming a concentration gradient that directs the approach of phagocytic cells.

Q.10 What are the various chemotaxins for neutrophils?

(i) Tumor necrosis factor (TNF- α)
(ii) IL-8
(iii) Platelet activating factor
(iv) Leukotriene B4
(v) C5a
(vi) Neutrophilic chemotactic factor
(vii) IL-1
(viii) IFN-α
(ix) N- formyl- methionyl peptides.

Q.11 Name various chemoattractants.

A. Exogenous chemoattractant:
   (a) Bacterial products
      N - formyl methionine terminal amino acid
B. Endogenous chemoattractant:
   (a) Components of complement system C5a
   (b) Products of lipooxygenase pathways B4
   (c) Cytokines particularly IL-8

Q.12 What is Pan-receptor defect?

When all chemotaxin receptors are decreased, the defect is called Pan-receptor defect. It is seen in localized aggressive periodontitis. It is characterized by a decrease in chemotactic responses to a variety of chemotactic factors, including C5a, FMLP (formyl peptide) and leukotriene B4.

Q.13 What is complement system?

It refers to a series of factors occurring in normal serum that are activated characteristically by antigen – antibody interaction and subsequently mediate a number of biologically significant consequences. It is a complex of different protein fractions (C1 to C9).

Q.14 What are the principal biologic effects of complement system?

(i) Chemotaxis – Attracts PMNs through C3a, C5a, C5b67.

(ii) Kinin production – Induce pain, increases vascular permeability and dilatation through C2a
(iii) Anaphylaxis – Produces anaphylaxis by histamine, released by mast cells and increased permeability of capillaries.
(iv) Opsonization – Adhere Ag – Ab complexes to leukocytes, platelets etc. thereby increasing their susceptibility to phagocytosis by WBC, C3b, C5b and macrophages.
(v) Cell lysis – Causes lysis of red cells, gram negative bacteria through C6, C7, C8 and C9.
(vi) Activation of B lymphocytes – C3b
(vii) Enhancement of blood clotting – C6
(viii) Increased vascular permeability – C3a, C5a
(ix) Promotion of clot lysis – C3, C4.

Q.15 What are the sequential phases of transendothelial migration of neutrophils?

(i) Rolling
(ii) An insult to local tissue
(iii) Signalling the endothelium
(iv) Increased rolling
(v) Signal for rolling arrest
(vi) Strong adhesion
(vii) Zipper phase

Q.16 What is phagocytosis?

It is the engulfment of particulate matters or microbial parasites by the external cell membrane of the phagocyte, resulting in an intracellular membrane – delimited structure termed phagosome.

Q.17 Name various neutrophil disorders associated with periodontal disease.

(i) Diabetes mellitus
(ii) Papillon–Lefèvre syndrome
(iii) Down's syndrome
(iv) Chediak–Higashi syndrome
(v) Drug induced agranulocytosis
(vi) Cyclic neutropenia
(vii) Leukocyte adhesion deficiency.

Q.18 Name various periodontal disease with neutrophil disorders.

(i) ANUG
(ii) Localized aggressive periodontitis
(iii) Refractory periodontitis.

Q.19 What are the neutrophil defects associated with aggressive periodontitis?

(i) Abnormalities in adherence – LAD – I, LAD – II

(ii) Abnormalities in chemotaxis –
   (a) Decreased number of several receptors for chemotactic factors – Pan receptor defect.
   (b) Papillon–Lefèvre syndrome
   (c) Chediak–Higashi syndrome

(iii) Abnormalities in phagocytosis and intercellular killing.

Q.20 What are the various syndrome associated with periodontal disease?

(i) Papillon–Lefèvre syndrome:
Papillon and Lefevre in 1924 discovered this syndrome. It is a rare autosomal recessive disorder characterized by mutation in Cathepsin C gene located on chromosome 11 (11q14–q21).

Characterized by:
- Rapid generalized destruction of alveolar bone
- Affects both the deciduous and permanent dentition
- Palmar–plantar hyperkeratosis
- Intracranial calcification
- Retardation of somatic development
- Increased susceptibility to infection

(ii) Chediak–Higashi syndrome:
It is a autosomal recessive mode of inheritance disease localized to chromosome 1q43. There is a fusion of azurophil and specific granules into giant granules called Megabodies in neutrophils.

Characterized by:
- Decreased chemotaxis
- Decreased degranulation
- Decreased microbial activity
- Severe periodontitis
- Oral ulceration

(iii) Job's syndrome / Hyperimmunoglobulinemia (HIE):
Autosomal recessive disorder localized to chromosome 7q21. It is a biblical reference to Job, who was afflicted with boils from head to foot.

Characterized by:
- Marked elevation of Immunoglobulin E (IgE)
- Defects in neutrophil chemotaxis
- Chronic dermatitis
- Coarse facies include broadened nasal bridge and irregularly proportioned cheeks and mandible

- Serious life long bouts of recurrent infections with opportunistic organisms (Candida albicans).
- Cold abscesses – Abscesses can involve any organ.

Q.21 Write the microbiological aspect of host microbial interaction.

(i) Adherence
(ii) Host tissue invasion
(iii) Evasion of Host defense mechanism.

Q.22 How do the host tissue damages occur during host microbial interaction?

(i) Through metabolic by-products of microbes: Ammonia, fatty acid, peptides, enzymes (collagenase, keratinase, trypsin-like enzyme, fibronectin-degrading enzyme).
(ii) Through the release of biologic mediators from host tissue cells: IL-1, tumor necrosis factor, prostaglandins.

Gingival Diseases

Q.1 What are the various stages of gingivitis?

According to Page and Schroeder, there are 3 stages of gingivitis:

• Stage I	Initial lesion	2-4 days
• Stage II	Early lesion	4-7 days
• Stage III	Established lesion	14-21 days.

Q.2 Write the immune cells and clinical findings associated with each stages of gingivitis.

		Immune Cells	*Clinical Findings*
• Stage I —	Initial lesion	PMNs	Increase gingival fluid flow
• Stage II —	Early lesion	Lymphocytes	Erythema, bleeding on probing
• Stage III —	Established lesion	Plasma cells	Change in color, size, texture of gingiva.

Q.3 Write various etiologic factors responsible for gingival bleeding.

(A) Local factors -
- (a) Acute bleeding
  - Aggressive tooth brushing
  - Sharp pieces of hard food
  - Gingival burns from hot food or chemicals
  - Acute necrotizing ulcerative gingivitis
- (b) Chronic bleeding
  - Chronic inflammation

(B) Systemic factors -
- (a) Vascular abnormality:
  - Vitamin C deficiency
  - Allergy such as Henoch – Schonlein purpura
- (b) Vitamin K deficiency
- (c) Platelet disorder: Idiopathic thrombocytopenic purpura
- (d) Deficient platelet thromboplastic factors:
  - Uremia
  - Multiple myeloma
  - Postrubella purpura

(e) Coagulation defects:
- Haemophilia
- Christmas disease

(f) Malignancy:
- Leukemia

(g) Drugs:
- Salicylates
- Anticoagulants – dicoumarol, heparin.

Q.4 Write conditions in which gingival contour is changed.

(i) Acute and chronic gingivitis
(ii) Gingival recession
(iii) Gingival enlargement
(iv) Stillman's clefts
(v) McCall's festoons.

Q.5 Write conditions in which gingival lump is seen.

(i) Erupting teeth- third molars
(ii) Pregnancy gingivitis
(iii) Fibroepithelial polyp
(iv) Malignant conditions- Carcinoma, Kaposi's sarcoma, Lymphoma.

Q.6 What is the etiology of gingival ulcers?

(i) ANUG
(ii) Herpes simplex virus stomatitis
(iii) Aphthae
(iv) Self injury
(v) Malignant neoplasms
(vi) Drugs
(vii) Dermatoses
(viii) Systemic diseases- mucocutaneous, hematological, tuberculosis, syphilis, herpes virus, HIV.

Q.7 In which conditions gingival red lesion is seen.

(i) Desquamative gingivitis
(ii) Erythroplasia
(iii) Hemangiomas
(iv) Orofacial granulomatosis
(v) Crohn's diseases
(vi) Sarcoidosis
(vii) Wegener's granulomatosis
(viii) Neoplasm- Kaposi's sarcoma, carcinoma.

Q.8 What are McCall's festoons?

These are the enlargement of the marginal gingiva with the formation of "life–saver" like gingival prominence in relation to canine and premolar facial surfaces mostly.

Q.9 What is Stillman's cleft?

It is apostrophe-shaped identations which extend from and into gingival margin along the root surface, most frequently on the labial or buccal surfaces. The margin of the cleft are rolled underneath the linear gap in the gingiva and remainder of gingival margin is blunt instead of knife-edge. It was originally described by Stillman, as a result of occlusal trauma. It may be simple-cleavage in a single direction or compound-cleavage in more than one direction.

Q.10 Write the factors responsible for change in gingival color.

(A) Local factors:

(i) Acute gingivitis
- ANUG: Marginal bright red erythema
- Herpetic gingivostomatitis: Diffuse
- Chemical irritations : Patch like / diffuse

(ii) Chronic gingivitis: Reddish–blue, deep blue

(iii) Metallic pigmentation
- Bismuth pigmentation – Black line
- Arsenic pigmentation – Black
- Mercury pigmentation – Black line
- Lead pigmentation – Bluish red, deep blue (Burtonian line) Gray
- Silver pigmentation – Voilet marginal line

(B) Systemic factors:

(i) Endogenous factors:
- Addison's disease – Melanin pigment
- Peutz – Jeghers syndrome – Melanin pigment
- Albright's syndrome – Melanin pigment
- Jaundice – Bilirubin yellow
- Haemochromatosis – Iron – blue gray
- Diabetes
- Pregnancy
- Blood dyscrasias
- Hyperthyroidism
- Drugs -
  - Antimalarials – Quinacrine, Chloroquine- slate gray
  - Minocycline – Brown
  - Chlorpromazine
  - Zidovudine
  - Ketoconazole
  - Methyldopa
  - Busulphan

(ii) Exogenous factors:
- Tobacco / smoking - Gray
- Amalgam – Localized bluish black
- Coloring agents in food, lozenges and betel.

Q.11 Name various conditions in which there is change in the surface texture of gingiva.

A. Loss of stippling:
(i) Chronic gingivitis
(ii) Atrophic gingivitis
(iii) Chronic desquamative gingivitis

B. Leathery texture: Hyperkeratosis

C. Nodular surface: Drug induced gingival overgrowth.

Q.12 What is hypertrophy and hyperplasia?

Hypertrophy: It is an increase in size of cells causing the increase in the size of the tissues.

Hyperplasia: It is an increase in number of cells in a tissue, thus contributing to an overall increase in the size.

Q.13 Write various anticonvulsant drugs associated with gingival enlargement.

(i) Phenytoin
(ii) Phenobarbital
(iii) Carbamazepine
(iv) Sodium valproate
(v) Primidone
(vi) Felbamate.

Q.14 Which are the analogues of phenytoin that stimulates proliferation of fibroblast like cells?

- 1 – allyl - 5 - phenylhydantoinate
- 5 – methyl - 5 - phenylhydantoinate.

Q.15 Write various antihypertensive drugs associated with gingival enlargement.

(i) Nifedipine
(ii) Amlodipine
(iii) Nimodipine
(iv) Nicardine
(v) Nitrendipine
(vi) Diltiazem
(vii) Felodipine
(viii) Bepridil

Q.16 Write various gingival conditions/diseases that mainly involve interdental papilla and gingival margin.

(i) Gingival abscess
(ii) Necrotizing ulcerative gingivitis
(iii) Linear gingival erythema
(iv) Drug induced gingival enlargement.

Q.17 How clinically Idiopathic gingival fibromatosis is different from phenytoin-induced hyperplasia?

Idiopathic gingival fibromatosis involve gingival margin, interdental papillae and attached gingiva whereas in phenytoin-induced hyperplasia, only gingival margin and interdental papillae are involved.

Q.18 Why necrotizing ulcerative gingivitis (NUG) was called as trench mouth?

Because NUG was frequently found among soldiers in the frontline trenches during World War I.

Q.19 How necrotizing ulcerative gingivitis is clinically diagnosed?

NUG is clinically diagnosed on the basis of clinical findings:-

(i) Gingival pain – Constant radiating, gnawing pain, intensified by eating spicy / hot food.
(ii) Ulceration – Punched out, crater like depression at the crest of interdental papilla
(iii) Bleeding – Spontaneous gingival haemorrhage.

Q.20 What gingival changes are seen in mouth-breathers?

(i) Erythema
(ii) Edema
(iii) Enlargement
(iv) Diffuse smooth and shiny surface in the exposed gingival area. Affecting mainly maxillary anterior region.

Q.21 What gingival changes are seen in pregnancy?

(i) Erythema: bright red to bluish red
(ii) Edema
(iii) Smooth and shiny
(iv) Raspberry-like appearance
(v) Increased tendency to bleed
(vi) Pregnancy granuloma
(vii) Pregnancy tumors
Affecting mainly anterior region.

Q.22 What is the role of Prevotella intermedia in puberty and pregnancy gingivitis?

P. intermedia uses estrogen and progesterone as a substitute for Menadione vitamin K growth factor, which increases with increased level of gonadotrophic hormone in puberty and pregnancy.

10 Gingival Diseases in Childhood

Q.1 What is the difference between the periodontium of deciduous and permanent dentition?

		Deciduous dentition	*Permanent dentition*
Gingiva			
1	Color	Pale pink	Coral pink
2	Surface texture	Stippling usually absent	Stippling usually present
3	Consistency	Less fibrous	More fibrous and firm
4	Interdental papilla	Broad faciolingually and narrow mesiodistally	Narrow faciolingually and broad mesiodistally
5	Width of attached gingiva	Less	More
Periodontal ligament			
6	Width	More wider	Less wider
7	Direction of periodontal fibres	Principal fibres are parallel to long axis of teeth	They are arranged in different directions
Cementum			
8	Thickness	Thinner and less dense	Thicker and more dense
Alveolar bone			
9	Lamina dura	Prominent	Less prominent
10	Trabeculae	Fewer but thicker	More but thinner
11	Marrow spaces	Larger	Smaller
12	Crest of interdental septa	Flat	Angulated

Q.2 Name various gingival diseases found in children.

(i) Chronic marginal gingivitis
(ii) Localized gingival recession
(iii) Acute gingival infections
- Acute herpetic gingivostomatitis
- Candidiasis

(iv) Factitious gingivitis.

Q.3 Name various childhood diseases which present specific alterations in gingival tissues.

(i) Varicella (Chickenpox)
(ii) Rubella (Measles)
(iii) Scarlatina (Scarlet fever)
(iv) Diptheria.

Periodontal Pocket

Q.1 What is Periodontal pocket?

It is pathologically deepened gingival sulcus.

Q.2 Classify Pockets.

(I) Depending on the number of surfaces involved:
- A. Simple - Involving one tooth surface
- B. Compound - Two or more tooth surfaces. The base of the pockets is in direct communication with the gingival margin along each of the involved surface.
- C. Complex - Spiral type pocket that originates on one tooth surface and twists around the tooth to involve one or more additional surfaces. The only communication with gingival margin is at surface where the pocket originates.

(II) Depending on Morphology:
- A. Gingival / pseudo pocket
- B. Periodontal pocket
  - (a) Suprabony pocket
  - (b) Infrabony pocket
    - (i) According to number of walls
      - 3 walled defects
      - 2 walled defects
      - 1 walled defects
    - (ii) According to its depth and width
      - Type I shallow narrow
      - Type II shallow wide
      - Type III deep narrow
      - Type IV deep wide
- C. Combined pocket

(III) Depending on disease activity:
- A. Active pocket
- B. Inactive pocket

(IV) Depending on the nature of soft tissue wall:
- A. Edematous
- B. Fibrotic

(V) Depending on the lateral wall of the pocket:
   A. Suprabony: Consist of soft tissue alone
   B. Infrabony: Consist of soft tissue and bone.

Q.3 Differences between Suprabony and Infrabony pockets.

		Suprabony pockets	*Infrabony pockets*
1	Relationship of the soft tissue wall of the pocket to the alveolar bone	Base of the pocket coronal to the level alveolar bone	Base of the pocket is apical to the crest of the alveolar bone
2	Pattern of bone destruction	Horizontal	Vertical
3	Direction of transseptal fibers interproximally	Horizontal	Oblique
4	Direction of periodontal ligament, on facial and lingual surfaces	Normal horizontal-oblique course between the tooth and the bone	Follows the angular pattern of the adjacent bone. They extend from the cementum beneath the base of the pocket along the bone and over the crest to join with the outer periosteum

Q.4 Write theories related to pathogenesis of periodontal pocket.

1. Destruction of gingival fibers is a prerequisite for the initiation of pocket formation— *Fish*
2. The initial change in pocket formation occurs in cementum— *Gottlieb*
3. Stimulation of the epithelial attachment by inflammation rather than destruction of gingival fibers is the prerequisite for the initiation of periodontal pocket— *Aisenberg*
4. Pathologic destruction of the epithelial attachment due to infection or trauma is the initial histologic changes in pocket formation— *Skillen*
5. The periodontal pocket is initiated by invasion of bacteria at the base of the sulcus or the absorption of bacterial toxins through the epithelial lining of the sulcus— *Box*
6. Pocket formation is initiated as a defect in sulcus— *Becks*
7. Proliferation of the epithelium of the lateral wall, rather than epithelium at the base of the sulcus, is the initial change in the formation of periodontal pocket— *Wilkinson*
8. Two stage pocket formation— *James and Counsell*
   a. Proliferation of the subgingival epithelium (epithelial attachment).
   b. Loss of superficial layers of proliferated epithelium, which produces space or pocket.
9. Inflammation is the initial change in the formation of periodontal pocket— *J Nuckolls*
10. Pathologic epithelial proliferation occurs secondary to non-inflammatory degenerative changes in periodontal membranes.

12 Bone Loss and Patterns of Bone Destruction

Q.1 What is the distance between apical extent of calculus and alveolar crest?
1.97mm ± 33.16%

Q.2 What is the distance between attached plaque and alveolar bone?
0.5 - 2.7 mm

Q.3 What is the radius of action of bacterial plaque that can induce bone loss?
1.5 - 2.5 mm approx.

Q.4 What is the yearly rate of bone loss when periodontal disease is allowed to progress untreated?
Rate of bone loss depends upon the type of disease present. An average of 0.2 mm/year on facial surfaces and 0.3 mm/year on proximal surfaces.

Q.5 What was the Loe study on the loss of attachment?
Loe and coworkers identified 3 subgroups of patients in a study on Sri Lankan tea labourers with no oral hygiene and dental care, with periodontal disease based on interproximal loss of attachment and tooth mortality.

(a) Eight percent population had rapid progression of periodontal disease. Loss of attachment of 0.1 - 1.0 mm yearly.
(b) Eightyone percent had moderately progressive periodontal disease. Loss of attachment of 0.05 - 0.5 mm yearly.
(c) Eleven percent had minimal or no progression of periodontal disease. Loss of attachment of 0.05 - 0.09 mm yearly.

Q.6 What are the various factors determining bone morphology in periodontal disease?

(i) Normal variation in alveolar bone
(ii) Exostoses
(iii) Trauma from occlusion
(iv) Buttressing bone formation
(v) Food impaction
(vi) Aggressive periodontitis

Q.7 Write various anatomic features that influence bone destructive patterns in periodontal disease.

(i) Thickness, width and crestal angulation of the interdental septa
(ii) Thickness of facial and lingual alveolar plates
(iii) Presence of fenestration and dehiscence
(iv) Alignment of teeth
(v) Proximity with another tooth surfaces
(vi) Root and root trunk anatomy
(vii) Root position within alveolar bone.

Q.8 What are the various causes of bone destruction?

(i) Extension of gingival inflammation
(ii) Trauma from occlusion
(iii) Systemic disorders.

Q.9 What are the various systemic disorders which cause bone destruction?

(i) Hyperparathyroidism
(ii) Leukemia
(iii) Langerhan's cell histiocytosis.

Q.10 What are the various mechanisms of bone destruction?

According to Hausmann, the various mechanisms of bone destruction are:

(i) Direct action of plaque products on bone progenitor cells induces the differentiation of these cells into osteoclasts.
(ii) Plaque products act directly on bone, destroying it through a non-cellular mechanism.
(iii) Plaque products stimulate gingival cells, causing them to release mediators, which in turn induce bone progenitor cells to differentiate into osteoclasts.
(iv) Plaque products causes gingival cells to release agents that can act as cofactors in bone resorption.
(v) Plaque products causes gingival cells to release agents that destroy bone by direct chemical action, without oseoclasts.

Q.11 What is bone factor concept?

Bone factor concept was given by Irving Glickman in early 1950s. This concept is a clinical guide for determining the diagnosis and prognosis of periodontal disease based upon the response of alveolar bone to local injurious factors. The systemic regulatory influence upon the response of alveolar bone is termed as Bone factor in periodontal disease. The destructive effect of inflammation and trauma from occlusion varies with the status of the individual bone factor. It is less severe in a healthy individual in the presence of negative bone factor.

Q.12 What are the various bone destructive patterns in periodontal disease?

(i) Horizontal bone loss
(ii) Vertical / angular defects
(iii) Osseous craters
(iv) Bulbous craters
(v) Reversed architecture
(vi) Ledges
(vii) Furcation involvement.

Q.13 What is one walled, two walled and three walled osseous defects?

- One walled osseous defects : only one bony wall is present
- Two walled osseous defects : two bony walls are present
- Three walled osseous defects : three bony walls are present

Q.14 What is hemiseptum?

The one wall vertical defect is called as hemiseptum.

13 Periodontitis

Q.1 What is periodontitis?

It is defined as an inflammatory disease of the supporting tissue of the teeth caused by specific microorganism or groups of specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with pocket formation, recession or both.

Q.2 How is periodontitis clinically distinguished from gingivitis?

By the presence of –

(i) Clinical attachment loss

(ii) Alveolar bone loss.

Q.3 Explain progression of periodontal diseases.

(i) Continuous disease model - Where loss of attachment has commenced, this will proceed continuously and slowly until tooth loss eventually results. Linear correlation between age and loss of attachment, supports this concept of gradual destruction.

(ii) Random burst disease model - 1982 Goodson et al challenged the continuous disease model and proposed that destruction occurs during periods of exacerbation, interjected with intervals of remission. Breakdown occurs in recurrent acute episodes / burst of activity over a short time span, interspersed with periods of quiescence.

(iii) Stochastic disease model - 1989 Manji and Nagelkerke proposed Stochastic model for periodontal breakdown that essentially combines both of the above models. They suggested that, as well as an underlying slow continuous breakdown (the progression rate of which depends on host and sites), some sites of some individuals are also undergoing random bursts of activity as a result of a combination of biological events.

Q.4 Write main clinical features and characteristics of Chronic Periodontitis.

(i) Most prevalent in adults, but can occur in children and adolescents.

(ii) Amount of destruction is consistent with the presence of local factors.

(iii) Sublingual calculus is a frequent finding.
(iv) Slow to moderate rate of progression, but may have periods of rapid progression.
(v) Can be associated with local predisposing factors (e.g. tooth related or iatrogenic factors).
(vi) May be modified by and or associated with systemic diseases (e.g. diabetes mellitus).
(vii) Can be modified by factors other than systemic disease such as cigarette smoking and emotional stress.

Q.5 What are the primary and secondary features of Aggressive Periodontitis?

Primary features:
(i) Non-contributing medical history
(ii) Rapid attachment loss and bone destruction
(iii) Familial aggregation of cases.

Secondary features:
(i) Amount of microbial deposits inconsistent with the severity of periodontal tissue destruction.
(ii) Elevated proportions of A.a.
(iii) Phagocyte abnormality
(iv) Hyper-responsive macrophage phenotype, including elevated production of PGE_2 and IL - 1α in response to bacterial endotoxin.
(v) Progression of attachment loss and bone loss may be self arresting.

Q.6 Write specific features of Localized and Generalized Aggressive Periodontitis.

Localized aggressive periodontitis:
(i) Circumpubertal onset.
(ii) Localized first molar/incisor presentation with interproximal attachment loss on at least two permanent teeth, one of which is a first molar and involving no more than two teeth other than first molars and incisors.
(iii) Robust serum antibody response to infecting agents.

Generalized aggressive periodontitis:
(i) Usually affecting person under 30 years of age, but patients may be older.
(ii) Generalized interproximal attachment loss affecting at least 3 permanent teeth other than first molars and incisors.
(iii) Pronounced episodic nature of the destruction of attachment and alveolar bone.
(iv) Poor serum antibody response to infecting agents.

Q.7 Compare Chronic Periodontitis (CP), Localized Aggressive Periodontitis (LAP) and Generalized Aggressive Periodontitis (GAP).

	CP	*LAP*	*GAP*
1	Most prevalent in adults,can occur in children	Usually occur in adolescents	Usually affects people under 30 years of age, but patients may be older
2	Slow to moderate rate of progression	Rapid rate of progression	Rapid rate of progression (Pronounced episodic periods of progression)
3	Amount of microbial deposits consistent with severity of destruction	Amount of microbial deposits not consistent with severity of destruction	Amount of microbial deposits sometimes consistent with severity of destruction
4	Variable distribution of periodontal destruction; No discernible pattern	Periodontal destruction localized to permanent first molars and incisors	Periodontal destruction in addition to first molars and incisors
5	No marked familial aggregation	Familial aggregation	Marked familial aggregation
6	Frequent presence of subgingival calculus	Subgingival calculus usually absent	Subgingival calculus may or may not be present

Q.8 How is Recurrent periodontitis different from Refractory periodontitis?

		Recurrent periodontitis	*Refractory periodontitis*
1	Definition	Sites successfully treated but periodontitis returns. May refer to sites/patients	Sites do not respond to conventional therapy. Usually refers to patients but may refer to sites
2	Phases of therapy	May be due to inadequate therapy during maintenance or no maintenance	May be due to inadequate therapy during active treatment or other factors
3	Etiology	May be due to reinfection with microbes that were suppressed but not eliminated or reinfection with eliminated organisms or new bacteria	May be due to reinfection with tissue invasive microbes that can't be eliminated with conventional therapy or immunocompetence
4	Immunocompetence	Immunocompetent	May not be Immunocompetent
5	Antibiotic therapy	Not usually needed	Usually needed

AIDS and Periodontium

Q.1 Define AIDS.

The acquired immunodeficiency syndrome (AIDS) is a severe condition caused by infection with the human immunodeficiency virus (HIV – 1).

Q.2 Classify AIDS patients.

AIDS patients have been grouped as follows, according to the CDC surveillance case

Classification (1993)

I. Laboratory Categories:

Category 1: $\geq$ 500 CD4 lymphocytes / mm^3

Category 2: 200-499 CD4 lymphocytes / mm^3

Category 3: < 200 CD4 lymphocytes / mm^3

II. Clinical categories:

Category A: Asymptomatic (acute primary) HIV - 1

One or more of the following conditions.

- Asymptomatic HIV - 1 infection
- Persistent generalized lymphadenopathy (PGL)
- Acute (primary) HIV - 1 infection or history of acute HIV - 1 infection

Category B: Symptomatic (not A or C)

Examples of conditions in category B follow. They must be attributed to HIV – 1 infection, indicative of a defect in cell – mediated immunity, and not listed under category C.

- Bacillary angiomatosis
- Candidiasis, oropharyngeal (thrush)
- Candidiasis, vulvovaginal
- Cervical dysplasia (moderate or severe) / cervical carcinoma *in situ*
- Constitutional symptoms, such as fever (38.5° C) or diarrhoea lasting > 1 month
- Hairy leukoplakia, oral
- Herpes zoster (shingles), involving atleast two distinct episodes or more than 1 dermatome
- Idiopathic thrombocytopenic purpura

- Listerosis
- Pelvic inflammatory disease, particularly if complicated by tubo-ovarian abscess
- Peripheral neuropathy.

Category C: AIDS – Indicator conditions

Conditions that follow in category C are strongly associated with severe immunodeficiency, occur frequently in HIV – 1 infected individuals, and cause serious morbidity or mortality:

- Candidiasis of bronchi, trachea or lungs
- Candidiasis, esophageal
- Cervical cancer, invasive
- Coccidioidomycosis, disseminated or extrapulmonary
- Cryptococcosis, extrapulmonary
- Cryptosporidiosis, chronic intestinal (> 1 month's duration)
- Cytomegalovirus disease (other than liver, spleen or nodes)
- Encephalopathy, HIV – 1 related
- Herpes simplex: Chronic ulcer(s) (>1 month's duration); or bronchitis, pneumonitis or esophagitis
- Histoplasmosis, disseminated or extrapulmonary
- Isosporiasis, chronic intestinal (> 1 month's duration)
- Kaposi's sarcoma
- Lymphoma, Burkitt's (or equivalent term)
- Lymphoma, immunoblastic (or equivalent term)
- Lymphoma, primary, of brain
- *Mycobacterium avium* complex of *M. Kansasii*, disseminated or extrapulmonary
- *Mycobacterium tuberculosis*, any site (pulmonary or extrapulmonary)
- *Mycobacterium*, other species or unidentified species, disseminated or extrapulmonary
- *Pneumocystis carinii* pneumonia
- Pneumonia, recurrent
- Progressive multifocal leukoencephalopathy
- *Salmonella* septicemia, recurrent
- Toxoplasmosis of brain
- Wasting syndrome caused by HIV – 1.

Q.3 What are the various oral manifestations of AIDS?

A. Fungal infections:
   - Candidiasis
   - Histoplasmosis
   - Cryptococcus
   - Geotrichosis

B. Bacterial infections:
- ANUG
- Mycobacterium avium intracellulare
- Actinomycosis
- Cat-scratch disease
- Klebsiella pneumoniae
- *E. coli*
- Sinusitis
- Submandibular cellulitis

C. Viral infections:
- Herpes simplex
- Cytomegalovirus
- Ebstein-Barr virus
- Varicella-Zoster virus
- Papillomavirus

D. Neoplasms:
- Kaposi's sarcoma
- Non-Hodgkin lymphoma
- Squamous cell carcinoma

E. Neurological disturbances:
- Trigeminal neuropathy
- Facial palsy

F. Unknown causes:
- Recurrent aphthous ulceration
- Progressive necrotizing ulceration
- Toxic epidermolysis
- Delayed wound healing
- Idiopathic thrombocytopenia
- Salivary gland enlargement
- Xerostomia
- Oral mucosal hyperpigmentation.

Q.4 What are the various periodontal pathologies associated with HIV infected patient?

A. Linear gingival erythema (LGE)

B. Necrotizing ulcerative periodontal diseases
   (a) Necrotizing ulcerative gingivitis (NUG)
   (b) Necrotizing ulcerative periodontitis (NUP)
   (c) Necrotizing ulcerative stomatitis

C. Enhanced progression of chronic adult periodontitis.

Q.5 What are the peculiar features of linear gingival erythema (LGE)?

Linear gingival erythema is defined as a gingival manifestation of immunosuppressed patients which is characterized by a distinct linear erythema limited to the free gingival margin. The lack of response of linear gingival erythema to conventional periodontal therapy, including plaque control, scaling and root planing is a key diagnostic feature of linear gingival erythema. Another key feature of LGE is its association with Candida infection.

Q.6 What are the peculiar features of necrotizing ulcerative gingivitis?

(i) Ulceration of interdental papilla – punched out appearance of interproximal papilla and affected area typically appears to be covered with a fibrinous pseudomembrane.
(ii) Gingival bleeding spontaneously
(iii) Severe pain

Q.7 What are the differences between necrotizing ulcerative gingivitis (NUG) and acute herpetic gingivostomatitis (AHG)?

		NUG	*AHG*
1	Site of ulcers	Interdental papilla, marginal gingiva	Gingiva, no predilection for interdental papilla entire oral mucosa
2	Character of ulcers	a. Punched out, crater like depression covered by yellow/white/gray pseudo-membranous slough	a. Multiple vesicles that coalesce and form shallow fibrin-covered regular shaped ulcers.
		b. Bleed readily/spontaneo-usly. Painful on stimulation	b. No marked tendency to bleed non-tender
3	Fever	Doubtful/slight only	38° C (or more)
4	Symptoms	Painful gums/dead feeling teeth	Sore mouth
5	Duration of ulcers and discomfort	Short lived (1-3 days), with appropriate therapy	More than 1 week, even with therapy
6	Etiology	Interaction b/w host and bacteria, most probably fusospirochetes	Specific viral etiology
7	Age	Uncommon in children	More frequently in children
8	Contagious	Non-contagious	Contagious
9	Immunity	No demonstrated immunity	An acute episode results in some degree of immunity.

Q.8 What are the peculiar features of necrotizing ulcerative periodontitis?

(i) Lesions extend into alveolar bone
(ii) Gingival recession
(iii) Tooth mobility

(iv) Other features:
- Oral malodour
- Lymphadenopathy
- Fever
- Malaise.

Q.9 What is necrotizing ulcerative stomatitis?

Necrotizing ulcerative stomatitis is the extension of necrotizing ulcerative periodontitis lesions into adjacent maxillary and mandibular bone leading to osteonecrosis and sequestration of the bone.

Q.10 What are the goals of treatment in HIV patients?

(i) Reduce HIV related morbidity and mortality, improve the quality of life.
(ii) Restore and preserve immunologic function.
(iii) Suppress viral load maximally and durably.

Q.11 What are the various chemotherapeutic agents used in treatment of HIV?

Four classes of US – FDA approved antiretroviral agents:
(i) Nucleoside and nucleotide analog reverse transcriptase inhibitor
(ii) Non-nucleoside analog reverse transcriptase inhibitor
(iii) Protease inhibitors
(iv) Entry (Fusion) inhibitors.

Smoking and Periodontium

Q.1 Classify Smokers.

A. According to CDC
   (i) Current smokers - Those that had smoked ≥ 100 cigarettes over their lifetime and smoked at the time of interview.
   (ii) Former smoker - Those that had smoked ≥ 100 cigarettes over their lifetime but were not currently smoking.
   (iii) Non-smokers - Those that had not smoked ≥ 100 cigarettes in their lifetime.

B. According to number of cigarettes smoked / day
   (i) Heavy smokers- smoked ≥ 20 cigarettes / day.
   (ii) Light smokers- smoked ≤ 19 cigarettes / day.

Q.2 What are the various constituents of tobacco smoke?

A. Particulate phase
   (i) Nicotine and Cotinine – facilitates bacterial colonization of epithelial cells
   (ii) Tar (compound of many chemicals)
   (iii) Benzene
   (iv) Benzo(a)pyrene

B. Gas phase
   (i) Carbon monoxide decreases the oxygen capacity of Hb
   (ii) Ammonia
   (iii) Dimethyl nitrosamine
   (iv) Formaldehyde
   (v) Hydrogen cyanide inhibits enzyme system necessary for oxidative metabolism
   (vi) Acrolein.

Q.3 What is the effect of smoking on neutrophil?

(i) Alter neutrophil chemotaxis and phagocytosis
(ii) Increases the production of neutrophil derived degradative proteases: MMP - 1, MMP – 8 and IL - 8
(iii) Inhibits respiratory burst of neutrophils
(iv) Increases neutrophil collagenase.

Q.4 Write deleterious effects of smoking.

(i) Hypertension
(ii) Atherosclerosis
(iii) Cancer
(iv) Chronic lung disease
(v) Ischaemic heart disease
(vi) Hypercoagulability
(vii) Coronary artery disease, Stroke
(viii) Eosphageal reflux
(ix) Peripheral vascular disease
(x) Peptic ulcer disease
(xi) Spontaneous abortion
(xii) Prematurity
(xiii) Low birth weight
(xiv) Delayed wound healing
(xv) Risk factor for periodontal diseases.

Q.5 What is the relationship of smoking and vitamin C ?

Cigarette smoking is known to contain numerous oxidants causing tissue damage. OH^- radical can mediate tissue damage and accumulation of hydroperoxide, which can disrupt membrane functions. Vitamin C is known as scavenger of OH^- radicals, hypochlorous acid, strong oxidative agent that can activate both neutrophil derived and GCF collagenase. This oxidative activation can be prevented by vitamin C.

16 Halitosis

Q.1 Classify halitosis with corresponding treatment needs (TN).

1. Genuine halitosis
   - A. Physiologic halitosis: TN-1 Malodour arises through putrefactive processes within the oral cavity. Neither a specific disease nor a pathologic condition that could cause halitosis is found. Origin is mainly the dorsoposterior region of the tongue.
   - B. Pathologic halitosis:
     - (i) Oral TN-2 Halitosis caused by disease, pathologic condition or malfunction of oral tissues. Halitosis derived from tongue coating, modified by pathologic condition (e.g. periodontal disease, xero-stomia) is included in this subdivision.
     - (ii) Extraoral TN-3 Malodour originates from nasal, paranasal and / or laryngeal region, pulmonary tract or upper digestive tract, diabetes mellitus, hepatic cirrhosis, uremia, internal bleeding.
2. Pseudo-halitosis TN-4 Obvious malodour is not perceived by others although the patient stubbornly complains of its existence.
3. Halitophobia TN-5 After treatment for genuine halitosis or pseudo halitosis, the patient persists in believing that he / she has halitosis.

Treatment needs (TN) for breath malodour.

Category	Description
TN-1	Explanation of halitosis and instructions for 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 psychologist or psychiatrist.

Q.2 What are the causes of halitosis?

A. **Oral:**

- Tongue coat – fissured, hairy tongue
- Periodontal diseases
- Caries
- Acute primary herpetic gingivostomatitis
- Acute necrotising ulcerative gingivitis
- Pericoronitis

B. **Extra oral:**

(a) Upper respiratory tract infections -
- Bronchiectasis
- Lung abscess
- Chronic bronchitis
- Bronchial carcinoma

(b) Ear, nose and throat cause -
- Post nasal drip
- Sinusitis
- Tonsillitis
- Throat infections

(c) Systemic causes -
- Hepatic failure - mousy odour
- Azotemia / kidney failure - uremic odour
- Diabetic ketoacidosis - acetone odour
- Chronic glomerulonephritis

(d) Medications-
- Antidepressants
- Antihypertensives
- Antihistamines
- Disulfiram (used to treat alcoholism)
- Dimethyl sulphoxide (used in intersititial cystitis)
- Cysteamine (used in nephropathic cystinosis)

(e) Certain foods -
- Food containing lactose, e.g. dairy products like milk, cheese, yoghurt, ice-cream
- Food containing sulphur, e.g. onion, garlic

(f) Other predisposing factors -
- Smoking
- Dry mouth
- Stress
- Alcohol
- Hormonal changes.

Q.3 Why tongue is the primary source of oral malodour?

(i) Large surface area of the tongue exposed to the expired air.

(ii) Availability of substrates that can be degraded to malodorous molecules by the tongue flora.

(iii) Dorsal tongue mucosa shows a very irregular surface topography with innumerable depressions, which are ideal niche for bacterial adhesion and growth.

Q.4 Name various volatile organic compounds produced by saliva or tongue coatings.

(i) Sulphur compounds:
- Hydrogen sulphide
- Methyl mercaptan
- Dimethyl sulphide

(ii) Short chain fatty acids:
- Propionic
- Butyric
- Valeric

(iii) Polyamines:
- Cadaverine
- Putrescine

(iv) Alcohols: 1- propoxy-2-propanol

(v) Phenyl compounds:
- Indole
- Skatole
- Pyridine

(vi) Alkanines: 2-methyl-propane

(vii) Ketones

(viii) Nitrogen containing compounds :
- Urea
- Ammonia.

Q.5 How halitosis is examined clinically and in laboratory?

A. **Clinical examination:**

(a) Self examination

(b) Organoleptic measurement: It is a sensory test scored on the basis of the examiner's perception of the subject's oral malodour. A straw or a plastic tube (24 mm in diameter and 10 cm in length) is inserted into the patient's mouth. While the patient is exhaling slowly, the examiner judges the odour at the other end of the tube.

Organoleptic scoring scale:

0 – Absence of odour	– Odour can't be detected
1 – Questionable odour	– Odour is detectable, although the examiners could not recognize it as malodour
2 – Slight odour	– Odour is deemed to exceed the threshold of malodour recognition
3 – Moderate odour	– Malodour is definitely detected
4 – Strong odour	– Strong malodour which can be tolerated
5 – Severe malodour	– Malodour is detected which cannot be tolerated.

B. **Laboratory examination:**

(a) Sulphide monitor: Halimeter

(b) Gas chromatography

(c) Dark – Field / Phase contrast microscopy

(d) Saliva Incubation test

(e) Diamond probe

Q.6 What instructions are given to the patient and examiner before undergoing organoleptic test?

Instructions to patient:

(i) Patients are instructed to abstein from taking antibiotics three weeks before.

(ii) Stop eating foods that contain garlic, onion and spices 48 hours prior to assessment day.

(iii) Avoid eating, drinking, smoking, oral hygiene practices and breath freshners for 12 hours prior to the assessment.

Instructions to examiner:

(i) Should have normal sense of smell.

(ii) Refrain from drinking and using scented cosmetics before the assessment.

(iii) Not to wear gloves, odour of which may interfere with organ-oleptic assessment.
(iv) Assessment should be made at several appointments on different days.

Q.7 What are the advantages and disadvantages of organoleptic measurement?

Advantages:
- This procedure prevents the dilution of odour with room air.
- Does not require special equipment.

Disadvantage:
- Objectivity and reproducibility of organoleptic measurement are poor.

Q.8 What are the advantages and disadvantages of sulphide monitor?

Advantages:
(i) No need for skilled personnel
(ii) Portability
(iii) Non-invasive
(iv) Low likelihood of cross-infection
(v) Relatively inexpensive
(vi) Rapid turn around time of one to two minutes between measurements.

Disadvantages:
(i) Inability to distinguish between individual sulphides
(ii) Instrument show slight loss of sensitivity with time, necessitating periodic recalibration
(iii) Measurement cannot be made in the presence of high levels of ethanol or essential oils.

Q.9 What are the advantages and disadvantages of GAS chromatography?

Advantages:
(i) Separation and quantitative measurement of individual gases
(ii) Ability to measure extremely low concentration of gases

Disadvantages:
(i) Relatively high cost
(ii) Skilled personnel required
(iii) Cumbersome and lack of portability
(iv) More time is required for detection and measurement.

Q.10 What is Diamond Probe?

Diamond probe is recently developed instrument, which combines the features of a periodontal probe with the detection of volatile sulphur compounds in the periodontal pocket. It is a modified Michigan O style, disposable dental probe incorporating a silver sulfide sensor.

Q.11 What are the various treatment strategies to control halitosis?

A. Mechanical reduction of intraoral nutrients and microorganisms
   (a) Oral prophylaxis: Scaling, root planing and oral hygiene instructions.
   (b) Tongue cleaning: By either a brush or a tongue scraper.

B. Chemical reduction of oral microorganisms
   (a) Mouth washes: Chlorhexidine, Listerine, Cetylpyridium chloride and Zinc chloride
   (b) Antimicrobials / antibiotics: Penicillins, Metronidazole, Tetracyclines, Ciprofloxacin and Tinidazole.

C. Rendering malodorous gases non-volatile
   (a) Dentifrices: Baking soda dentifrices
   (b) Metal salt solutions: Zn^{++} ions

D. Masking the malodour: Bioadhesive tablets / Lozenges

E. Dietary recommendations: Avoid coffee and advised to drink plenty of liquids, fresh fibrous vegetable, rinse their mouth after eating or consuming milk products, fish and meat.

Q.12 What are the various Do's of tongue cleaning?

(i) Use small tongue scraper designed to reach as far back on tongue as possible.
(ii) Use light pressure that avoids abrading tongue surface.
(iii) Rinse and clean the instrument well after each use.

17 Dentin Hypersensitivity

Q.1 What is Dentin Hypersensitivity?

Dentin hypersensitivity is an exaggerated response to non-noxious stimuli. It is characterized by short, sharp pain arising from exposed dentin in response to stimuli typically thermal, evaporative, tactile, osmotic / chemical and which cannot be ascribed to any other form of dental defect / pathology.

Q.2 What is the etiology of dentin hypersensitivity?

A. Loss of enamel:
   (a) Attrition
   (b) Abrasion
   (c) Erosion

B. Loss of covering periodontal structures:
   (a) Gingival recession
   (b) Periodontal diseases
   (c) Chronic trauma from various habits
   (d) Horizontal tooth brushing.

Q.3 What are the various theories related to dentinal hypersensitivity?

(i) Transducer theory
(ii) Modulation theory
(iii) Gate control and vibration theory
(iv) Hydrodynamic theory – Brannstrom (1963) states that when the fluids within dentinal tubules are subjected to temperature changes or physical osmotic changes, the movement stimulates a nerve receptor sensitive to pressure, which leads to the transmission of the stimuli.

Q.4 How is dentinal hypersensitivity diagnosed?

(i) Visual examination of the teeth
(ii) Detailed dietary history
(iii) Occlusion assessment
(iv) Diagnostic tools:
   - Air / water syringe
   - Tactile method: Dental explorer, Yeaple probe

- Percussion testing
- Bite stress test
- Thermal test: Ice cube test.

Q.5 What is Verbal rating scale (VRS) and Visual analogue scale (VAS)?

Verbal rating scale (VRS): Given by Kanapka and Colucci (1986) and Gillman and Newman (1993).

This scale records the response of the patient after scratching and air-cold tests on a severity scale.

0 – No response
1 – Slight response but no pain
2 – Pain only when stimulus is applied
3 – Severe, sudden and lasting pain

Score – 0 and 1 – Classified as non-sensitive teeth
2 and 3 – Classified as hypersensitive teeth

Visual analogue scale (VAS): It is scale of 10 cm which is used to grade sensitivity, labelled at the extremes with no pain at the zero cm end of the scale and severe pain at the 10 cm end of the scale. Subjects are asked to place a mark on the 10 cm line at a location between no pain and severe pain ends. Measurements from the scale were made in mm giving a scoring range of 0-100.

Q.6 Name various desensitizing agents with trade names.

Trade name	***Agents***
Sensodyne	10% strontium chloride and sodium fluoride
Thermodent	10% strontium chloride
Protect	2% dibasic sodium citrate in a pleuronic gel
Promise	5% potassium nitrate, dicalcium phosphate and sodium Monofluoro-phosphate
Denquel	5% potassium nitrate
Isodan	Potassium nitrate, sodium fluoride, HEMA (hydroxy ethyl methacrylate)
Sensodyne F ^{R}	KCl and sodium monofluorophosphate
Colgate sensitive care	Potassium citrate and sodium monofluorophosphate
Macleans Sensitive	Strontium acetate and sodium monofluorophosphate

Q.7 What is the mechanism of action of potassium nitrate?

(i) Decrease fluid flow through the tubules by occluding them.

(ii) Decrease the level of activity of the dental sensory nerves, thus preventing the pain signals to be transmitted to the central nervous system.

Potassium ions of KNO_3 diffuses through the dentinal tubules and reaches the pulp sensory complex and forms a region of greatly increased concentration (K^+ ions) which depolarizes the pulpal sensory complex and reduces pain transmission.

Q.8 What is Hawthorne effect?

The term "Hawthorne effect" commonly refers to any "unexplained result in an experiment on human subjects, on the assumption that the result occurred simply because the subjects were in an experiment and thereby experienced something that otherwise would not have affected them." The effect is also recognized as a reaction of subjects to the realization they are in a study and are being observed. Hawthorne was a Western Electric Company plant where studies were conducted between 1924 and 1932 examining the influence of different work environment variables on productivity. "Hawthorne effect" has been used frequently to account for gains made by placebo control groups when none were expected.

Thus, subjects or patients who enroll in trials are always informed of the objectives and the nature of the research taking place. It is therefore quite possible that more time and effort are devoted to oral hygiene measures on the day of the visit to the trial unit. It is also possible that in such studies, participants appear to improve their toothcleaning habits and plaque scores decrease irrespective of the therapy provided.

Q.9 What is GLUMA?

It is combination of 5% glutaraldehyde and 35% HEMA.

Q.10 What are treatment approaches for dentinal hypersensitivity?

A. To occlude dentinal tubules; there by blocking hydrodynamic mechanism
   (a) In office surface applications of desensitizing agents
   (b) Home use applications of desensitizing agents

B. To block neural transmission at the pulp
   (a) Topically applied potassium salts
   (b) By endodontics
   (c) Tooth extraction.

Q.11 How is the dentinal hypersensitivity treated?

A. Patient counselling:
   (a) Oral hygiene practices
   (b) Dietary factors
   (c) Remove risk factors by educating about root caries

B. Interventional treatment:
   (a) At home treatment options: Dentifrices and Gels containing KNO_3, SnF_2
   (b) In office treatment options:
      (i) Non-invasive:
         • In office surface applications – Resin, HEMA, oxalates, fluorides
         • Class V restorations
         • LASERS
         • Iontophoresis
      (ii) Invasive:
         • Pulpectomy
         • Gingival graft surgery.

Q.12 What is fluoride iontophoresis?

It is a mean to drive fluoride ions more deeply into dentinal tubules. It involves the placement of a negative electrode to dentin and a positive electrode to the patient's face or arm. Saliva becomes the medium in which ions commence their selective motion. Negative ions flows through the positively charged teeth and positive ions to the negatively charged bristles.

Clinical Diagnosis and Prognosis

Q.1 What are the various tools for periodontal assessment?

	Tools	*Assessment: Natural Dentition*	*Assessment: Dental Implants*
1	Visual inspection	Gingival color, contour, tone Calculus detection	Same
2	Compressed air	Gingival tone Calculus detection	Same
3	Calibrated probe	Using metal/plastic probe: Gingival tone Clinical attachment level Bleeding points Exudate Mucogingival examination Measuring of oral deviations	Using plastic probe: Gingival tone Clinical attachment level Bleeding points Exudate
4	Furcation probe	Furcation involvement	Not applicable
5	Instrument handle	Mobility	Same, using plastic handles
6	Explorer	Calculus detection Detection of plaque retentive factors and subgingival calculus	Not applicable
7	Radiographic	Bone height and density	Same

Q.2 What is Attrition, Erosion, and Abrasion?

Attrition - It is a occlusal wear resulting from functional contacts with opposing teeth.

Erosion - It is a wear to the non-occluding tooth surfaces, which is sharply defined wedge-shaped depression in the crevical area of the facial tooth surface.

Abrasion - It refers to the loss of tooth substance induced by mechanical wear other than that of mastication.

Q.3 Write clinical presentation of Attrition, Erosion, Abrasion and of their combinations.

1. Attrition -
   (i) Flat occlusal or incisal surfaces.
   (ii) Accurate interdigitation of upper and lower teeth.
   (iii) Masseteric hypertrophy.

2. Erosion -
   (i) Buccal and lingual surfaces of upper incisors appear smooth and shiny with a generated loss of anatomy.
   (ii) On the palatal surface of the upper incisors the exposed dentin is smooth often with a halo of enamel surrounding the lesion.
3. Abrasion -
   (i) May manifest at the cusp tip / incisal edge that has been rounded, blunted or worn flat.
   (ii) It often exposes the dentin, causing a "scooped out" appearance that is softer and more porous than enamel.
4. Erosion and Attrition together -
   Some cupping or undermining of occlusal surfaces, the dentin being less mineralized than enamel appears to wear preferentially resulting in occulsal cupping.

Q.4 What are the causes for erosion?

(i) Vomiting associated with eating disorders like anorexia nervosa, bulimia nervosa and rumination
(ii) Reflux or chronic regurgitation associated with gastrointestinal problems
(iii) Regular and high intake of acidic medication (chewable acetylsalicylic acid tablets)
(iv) Regular intake of chewable vitamin C tablets
(v) High consumption of acidic drinks and foods
(vi) Professional wine tasting
(vii) Field of occupation — acid battery worker
(viii) Pregnancy.

Q.5 How can you differentiate whether the erosion lesion is active or inactive?

(i) Smooth, clean surfaces and presence of dentin hypersensitivity suggest the process is active whereas stained teeth, as then sufficient time for the stain to be taken up onto the tooth surface, suggest inactivity.
(ii) Restoration is resistant to acid and so it remains unchanged, but the tooth is gradually dissolved.
(iii) Comparison of dated study casts to the clinical condition of the teeth over the time period.

Q.6 What are the dietary advices to prevent the dental erosion?

(i) Reduce the frequency and amount of consumption of acidic drinks and food, especially at bedtime.
(ii) If soft drinks are consumed, should be chilled and consumed in one sitting at mealtime.

(iii) Avoid sipping the acidic drink or swishing it around the mouth before swallowing.
(iv) Consume neutralizing food such as cheese after the intake of an acidic drink or food.
(v) Encourage the consumption of water and nutritious beverages such as milk.

Q.7 What are the causes of abrasion?

(i) Hard-bristle toothbrush.
(ii) Coarse abrasive tooth powder.
(iii) Horizontal tooth brushing technique.
(iv) Action of clasps.
(v) Abrasion of incisal edges due to habits such as opening bobby pins, nails held by carpenters, pins by dressmakers.
(vi) Pipe held between teeth.

Q.8 What is abfraction?

It is the flexure of a tooth under heavy lateral load, which may lead to displacement/fracture of enamel rods at the CEJ. The lost enamel exposes more dentin, in which dentin tubules may be crushed by the same stresses and are more readily demineralized.

Q.9 What is frictional ablation?

It is a process caused by juxtaposition of natural and artificial dental surfaces and hyper functional oral soft tissues. It is caused by the action of soft tissues and saliva against the dentition due to vestibular pressures of suction, swallowing, tongue motion and the intervening forced flow of saliva.

Q.10 What is food impaction?

It is the forceful wedging of food into the periodontium by occlusal forces.

Q.11 Write classification and etiology of food impaction.

Hirschfeld in 1930 classified vertical food impaction relative to etiologic factors: -

Class I — Occlusal wear
Class II — Loss of proximal support
Class III — Extrusion of a tooth beyond the occlusal plane
Class IV — Congenital morphologic abnormalities
Class V — Improperly constructed restorations.

Q.12 What is the sequelae of food impaction?

(i) Feeling of pressure and the urge to dig the material from between the teeth.
(ii) Vague pain which radiates deep in the jaws.

(iii) Gingival inflammation with bleeding and a foul taste in the involved area.
(iv) Gingival recession.
(v) Periodontal abscess formation.
(vi) Varying degree of inflammatory involvement of the periodontal ligament with an associated elevation of the tooth in its socket, prematurity in functional contact and sensitivity to percussion.
(vii) Destruction of alveolar bone.
(viii) Caries of the tooth.

Q.13 What are the plunger cusps?

Plunger cusps are the cusps that tend to forcibly wedge food into interproximal embrasures of opposing teeth.

Q.14 Which is the most common plunger cusp?

Distolingual cusps of maxillary molars.

Q.15 How are plunger cusp formed?

(i) Plunger cusp effect may occur with tooth wear.
(ii) It may be the result of a shift in tooth positions following the failure to replace missing tooth.

Q.16 In which conditions the tooth preparations are needed to be extended into gingival sulcus?

(i) Esthetics in maxillary anterior region
(ii) Problems with retention
(iii) Extensive carious lesions
(iv) Replacement of defective and extensive restoration.

Q.17 Which are the plaque retentive areas in oral cavity?

A. Natural areas / factors:
   (a) Supragingival:
      - Supragingival calculus
      - A carious lesions
      - Exposed cementum
   (b) Subgingival:
      - Subgingival calculus
      - Cavitated caries lesions
      - Furcation involvement
      - Root grooves
      - Rough, unplaned cementum
      - Deep, narrow infrabony pockets
      - CEJ and enamel projection

B. Iatrogenic factors:
   - Overhanging restoration margins
   - Orthodontic band

- Overcontoured and inadequate crown margins
- Portions of removable prosthesis that impinge on gingiva.

Q.18 How overhanging margin contributes to periodontal diseases?

(i) By providing ideal niches for the accumulation of plaque.
(ii) By changing the ecological balance of the gingival sulcus from gram positive facultative species to gram negative anaerobic species.

Q.19 How overcontoured crowns and restorations contributes to periodontal diseases?

(i) By providing ideal locations for accumulation of plaque.
(ii) By preventing self-cleaning mechanisms of adjacent cheek, lips and tongue.

Q.20 Which are the characteristics of restorations that leave effect on periodontium?

(i) Margin of restoration
(ii) Contour and overhang
(iii) Material
(iv) Occlusion
(v) Design of removable partial prosthesis
(vi) Restorative procedure

Q.21 What is the effect of excessive overbite on periodontium?

It causes:
(i) Impingement of the teeth on gingiva
(ii) Food impaction
(iii) Gingival inflammation
(iv) Gingival enlargement
(v) Pocket formation.

Q.22 What is the effect of open bite on periodontium?

Reduced mechanical cleansing by the passage of food may lead to:
(i) Accumulation of debris
(ii) Calculus formation
(iii) Extrusion of teeth.

Q.23 What is the effect of cross bite on periodontium?

(i) Trauma from occlusion
(ii) Food impaction
(iii) Spreading of mandibular teeth.

Q.24 In which gingival or periodontal diseases lymph node enlargement is seen?

(i) Necrotizing ulcerative gingivitis
(ii) Primary herpetic gingivostomatitis
(iii) Acute periodontal abscesses.

Q.25 What are various mucogingival problems?

(i) Inadequate width of attached gingiva
(ii) Abnormal frenum attachment
(iii) Gingival recession
(iv) Decreased vestibular depth
(v) Pockets extending upto mucogingival junction
(vi) Gingival excess – pseudopocket, inconsistent gingival margin, excessive gingival display, gingival enlargement and abnormal color of gingiva.

Q.26 What are the problems associated with inadequate attached gingiva?

Inadequate attached gingiva zone would:

(i) Facilitate subgingival plaque formation because of improper closure of the pocket resulting from movability of the marginal tissue.
(ii) Favor attachment loss and soft tissue recession due to the less resistance of the tissue.
(iii) Accumulation of food particles during mastication.
(iv) Impede proper oral hygiene measures.

Q.27 How is the width of attached gingiva measured?

(i) Anatomically: Stretch the lip/cheek to demarcate the mucogingival line while pocket is being probed. Measure the total width of gingiva (gingival margin to mucogingival line) and subtract the sulcus/pocket depth from it to determine width of attached gingiva.
(ii) Functionally:
  (a) Tension test – Stretch the lip or cheek outward and forward to demarcate the mucogingival line and to see for any movement of free gingival margin. Measure the total width of gingiva (gingival margin to mucogingival line) and subtract the sulcus/pocket depth from it to determine width of attached gingiva.
  (b) Roll test – Push the adjacent mucosa coronally with a dull instrument to mark mucogingival line. Measure the total width of gingiva (gingival margin to mucogingival line) and subtract the sulcus/pocket depth from it to determine width of attached gingiva.
(iii) Histochemically: Staining test – Paint the mucosa with Schiller's potassium iodide solution, which stain keratin, i.e. marginal, attached gingiva and interdental papilla. Measure the total width of the stained gingiva and subtract the sulcus/pocket depth from it to determine width of attached gingiva.

Q.28 How is the thickness of gingiva measured?

Earlier the thickness was measured using traumatic techniques like probes and injection needle. But now it can be measured atraumatically using the newer ultrasonic device called 'KRUPP SDM'. This uses a pulse echo principle. With the aid of a pulse generator and a measurement frequency of 5 MHz, a piezo crystal is allowed to oscillate. Ultrasonic pulses are transmitted at an interval through the sound permeable gingiva. When it reaches the bone or tooth surface its starts being reflected due to difference in acoustic impedence. A transducer probe of 4 mm diameter is moistened with saliva and applied to the measurement site with slight pressure to produce acoustic coupling. By timing the received echo with respect to transmission of pulse, the thickness of mucosa is determined within seconds and is digitally displayed with a resolution of 0.1 mm.

Q.29 When frenum is judged abnormal?

(i) When the frenum is unusually broad.
(ii) There is no apparent attached gingiva in the midline.
(iii) Interdental papilla moves by stretching the frenum.

Q.30 How abnormal frenum jeopardizes gingival health?

(i) Interfere with proper placement of a toothbrush.
(ii) Open gingival crevice by muscle pull.

Q.31 What is tension test and its significance?

Stretch the lip or cheek outward and forward to demarcate the mucogingival line and to see for any movement of free gingival margin.
Significance:
(i) Detects any abnormal frenum attachment.
(ii) Tell whether attached gingiva is adequate or inadequate.
(iii) To identify mucogingival junction.

Q.32 What is tongue thrusting?

Tongue thrusting: It is the persistent, forceful wedging of the tongue against the teeth, especially in anterior region. It is a habit in which patient instead of placing the dorsum of the tongue against the palate with the tip behind the maxillary teeth during swallowing, tongue is thrust forward against the mandibular anterior teeth which tilt and also spread laterally.

Q.33 What is the effect of tongue thrusting on dentition?

(i) Tongue thrusting causes excessive lateral pressure, which may be traumatic to periodontium.
(ii) Causes spreading and tilting of the anterior teeth.
(iii) Open bite, anteriorly and posteriorly.
(iv) Pathologic migration.

Q.34 What are parafunctional habits? Classify.

Parafunctional means altered / abnormal function.

Classified in 3 ways according to the cause:

(i) Tooth to tooth function, e.g. bruxism
(ii) Tooth to soft tissue, e.g. digit-sucking
(iii) Tooth to foreign object, e.g. chewing of pens and pencils.

Q.35 Classify the habits which affect the initiation and progression of periodontal disease.

Classified by Sorrin and Cheek:

A. Neurosis:
   (i) Lip biting
   (ii) Fingernail biting
   (iii) Tongue thrusting
   (iv) Pencil / pen biting

B. Occupational habits:
   (i) Holding of nails in the mouth by cobblers, upholsterers, carpenter and thread biting.
   (ii) Pressure of a reed during the playing of musical instruments.

C. Miscellaneous habits:
   (i) Pipe / cigarette smoking
   (ii) Tobacco chewing
   (iii) Incorrect methods of toothbrushing
   (iv) Mouth breathing
   (v) Thumb sucking.

Q.36 What is bruxism?

Bruxism means constant or intermittent occlusal contact of the teeth, aside from mastication, swallowing / speech. It is the term for abnormal grinding of the teeth.

Q.37 What are the effects of bruxism?

(i) Causes excessive tooth wear characterized by facets on tooth surfaces.
(ii) Exaggerated facets in normal functional areas.
(iii) Widening of the occlusal surfaces.
(iv) Reduction in vertical dimension, in severe cases.

Q.38 What is the difference between trauma from occlusion and traumatic occlusion?

When occlusal forces exceeds the adaptive capacity of the tissues, tissue injury results, thus resultant injury is termed trauma from occlusion. Trauma from occlusion refers to the tissue injury and not the occlusal force.

An occlusion that produces such injury is called a traumatic occlusion.

Q.39 What is the effect of failure to replace first molars?

(i) Decrease in vertical dimension because second and third molars tilt
(ii) Anterior overbite is increased, thus traumatize the gingiva as mandibular incisors strike against the gingiva of maxillary incisors
(iii) Separation of anterior teeth leading to diastema formation
(iv) Maxillary incisors moved labially and laterally
(v) Anterior teeth extrude
(vi) Premolars move distally.

Q.40 What is the etiology of mobility?

A. Local factors:
(i) Bone loss or loss of tooth support
(ii) Trauma from occlusion, either in absence or associated with inflammation
(iii) Hypofunction
(iv) Periapical pathology
(v) After periodontal surgery
(vi) Parafunctional habits like bruxism or clenching
(vii) Pathology of jaws like tumors, cysts, osteomyelitis etc.
(viii) Traumatic injuries to dentoalveolar unit.

B. Systemic factors:
(i) Menstrual cycle
(ii) Oral contraceptives
(iii) Pregnancy
(iv) Systemic diseases: Papillon Lefevre syndrome, Down's syndrome, Neutropenia, Chediak Higashi syndrome, Hypophosphatasia, Hyperparathyroidism, Acute leukemia, Pagets disease.

Q.41 What is fremitus test and its significance?

Dampen index finger and place along the buccal and labial surfaces of maxillary teeth. The patient is then asked to tap the teeth together in the maximum intercuspation and to do lateral, protrusive movements.

- Class I : Mild vibration detected , recorded as "+"
- Class II : Easily palpable vibration, recorded as "++"
- Class III : Movements visible with naked eye, recorded as "+++".

Significance:

It is a test used to diagnose a case of trauma from occlusion, by measuring the vibratory pattern of teeth, when teeth are placed in contacting positions.

Q.42 What is the rationale behind fremitus test?

Periodontal fremitus occurs in either of the alveolar bones when an individual sustains trauma from occlusion. It is a result of teeth

exhibiting at least slight mobility rubbing against the adjacent walls of their sockets, the volume of which has been expanded ever so slight by inflammatory responses, bone resorption or both. As a test to determine the severity of periodontal disease a patient is told to close his or her mouth into maximum intercuspation and is asked to grind his or her teeth. Finger placed in the labial vestibule against the alveolar bone can detect fremitus.

Q.43 How is fremitus different from mobility?

Fremitus is tooth displacement which is created by patient's own occlusal force. Therefore, the amount of force varies greatly from patient to patient, whereas in mobility the force with which it is measured tends to be the same for each examiner. Fremitus is a guide to the ability of the patient to displace and traumatize the teeth.

Q.44 Write clinical and radiographic findings of trauma from occlusion.

Clinical findings:

(i) Progressively increasing tooth mobility
(ii) Fremitus test positive
(iii) Pathologic migration
(iv) Open contacts related to food impaction
(v) Sensitivity of teeth to pressure or percussion
(vi) Neuromuscular disturbances in the muscles of mastication
(vii) Temporomandibular joint symptoms

Radiographic findings:

(i) Widened periodontal ligament space
(ii) Angular bone loss
(iii) Thickened lamina dura
(iv) Root resorption
(v) Furcation involvement.

Q.45 What is gingivitis toxica?

According to Pindborg, it is a specific type of gingivitis in which there is destruction of gingiva and the underlying bone due to the chewing of tobacco.

Q.46 Classify abscesses.

According to Meng (1999) abscesses are classified into:

(a) Gingival abscesses
(b) Periodontal abscesses
(c) Pericoronal abscesses.

Q.47 What is Gingival abscess and its etiology?

Gingival abscess is a localized painful, rapidly expanding lesion usually of sudden onset. It is generally limited to the marginal gingiva or interdental papilla.

Etiology:

(i) Irritation from foreign substances, toothbrush bristle
(ii) Apple core
(iii) Lobster shell forcefully embedded into the gingiva.

Q.48 Classify periodontal abscesses.

Depending on the cause of acute infectious process, two types of periodontal abscess occur:

A. Periodontitis-related abscesses:
   (a) Exacerbation of chronic lesion
   (b) Post-therapy periodontal abscesses
      (i) Post-scaling periodontal abscesses
      (ii) Post-surgery periodontal abscesses
      (iii) Post-antibiotic periodontal abscesses

B. Non-periodontitis-related abscesses:
   (a) Impaction of foreign body in the gingival sulcus or periodontal pocket.
   (b) Root morphology alterations or iatrogenic endodontic perforations.

Q.49 What are the differences between Periodontal abscesses and Periapical abscesses?

		Periodontal abscesses	*Periapical abscesses*
1	History	• Periodontal disease • Periodontal treatment	• Caries, fracture, toothwear • Restorative and endodontic treatment
2	Clinical findings	• Vital pulp responses	• Questionable/non responsive pulp tests
		• Periodontal probing release pus	• Narrow probing defect (May be isolated lesion)
		• Periodontal disease evident	• Advanced caries, advanced toothwear, large restoration, discolored tooth
		• Swelling is generalized and located around the involved tooth and gingival margin. Seldom with a fistulous tract	• Swelling is localized often with fistulous opening in the apical area
		• Pain is usually dull, constant and less severe than in a periapical abscess. Pain is localized and patient usually can locate the offending tooth	• Pain is usually severe, throbbing and patient may not be able to locate the offending tooth
3	Radiographic	• Alveolar crest bone loss, angular bone defects, furcation involvements findings	• Apical radiolucency • Endodontic or post perforations
4	Response to treatment	• Responds dramatically to release of pus, subgingival debridement	• Responds poorly, or not at all to periodontal treatment

Q.50 What is level of attachment?

Level of attachment: It is the distance between the base of the pocket and the fixed reference point such as cementoenamel junction (CEJ) on the crown, margin of a permanent restoration; for animal research – a notch made in the tooth; in human research studies – template / splint may be made for each patient. There may be gain or loss of attachment.

Q.51 What is the significance and limitations of clinical attachment loss?

Significance:

The clinical attachment loss reveals the approximate extent of root surface that is devoid of periodontal ligament.

Limitation:

(i) It is used as an indicator of the amount of periodontal support at a specific location on the tooth, this measurement clearly does not provide an accurate assessment of support in terms of 3 dimensions / root surface area.

(ii) Apical extent of periodontal probe penetration and depth measurement is dependent on degree of inflammation, probing force, probe tip thickness, angulation and position of probing and root anatomy, particularly in furcation areas.

Q.52 What is bone sounding?

Anesthetize the tissue locally and the probe is inserted horizontally and walked along the tissue - tooth interface, so that the operator can feel the bony topography which gives three – dimensional information regarding bone contour. It is also called as transgingival probing.

It helps to determine:

(i) The height and contour of facial and lingual bone.

(ii) The architecture of the interdental bone.

(iii) The extent and configuration of intrabony component of the pocket and furcation defects.

Q.53 When is the periodontal lesion said to be inactive or active?

Inactive lesion:

A. Clinically:
   (a) Little / no bleeding
   (b) Minimal amounts of gingival fluid

B. Microbiologically: Dark field microscopy. Bacterial flora – consists of greater number of coccoid cells.

C. Histologically: Pocket epithelium intact

Active lesion:

A. Clinically:
   (a) Readily bleed on probing
   (b) Large amount of gingival fluid and exudate.

B. Microbiologically: Dark field microscopy. Bacterial flora – consists of greater number of spirochetes and motile bacteria.

C. Histologically:
   (a) Pocket epithelium is thin and ulcerated
   (b) Infiltrate composed mainly of plasma cells and PMNs.

Q.54 What is the minimum number of intraoral periapical radiographs that should be taken for radiographic survey of complete periodontium?

14.

Q.55 What is prognosis?

Prognosis is a predilection of the probable course, duration and outcome of a disease based on a general knowledge of the pathogenesis of the disease and the presence of risk factors for the disease.

Q.56 What are the various types of prognosis?

(i) Excellent prognosis
(ii) Good prognosis
(iii) Fair prognosis
(iv) Poor prognosis
(v) Questionable prognosis
(vi) Hopeless prognosis.

Q.57 What are the various overall and local factors affecting the prognosis?

Overall Factors:

(i) Patient's age
(ii) Disease severity
(iii) Plaque control
(iv) Patient compliance
(v) Environmental factors
   - Smoking
   - Systemic disease
   - Genetic factors
   - Stress

Local factors:

(i) Plaque / calculus
(ii) Subgingival restorations
(iii) Anatomic factors :
   - Short, tapered roots
   - CEPs
   - Enamel pearls
   - Root concavities
   - Developmental grooves
   - Root proximity
   - Furcation involvement
   - Tooth mobility.

Q.58 How does the patient's age affect the overall prognosis?

The prognosis is not good for younger patients because of the shorter time frame in which the periodontal destruction has occurred.

Q.59 In which teeth palatogingival grooves are found?

(i) Maxillary lateral incisors (5.6%)
(ii) Maxillary central incisors (3.4%).

Q.60 In which teeth mainly root concavities are found?

(i) Maxillary first premolars
(ii) Mesiobuccal root of maxillary first molars
(iii) Both roots of mandibular first molars
(iv) Mandibular incisors.

Q.61 Write the factors which are common for both prognosis and diagnosis of the disease.

(i) Patient's age
(ii) Severity of disease
(iii) Genetic susceptibility
(iv) Presence of systemic disease.

19 Diagnostic Aids

Q.1 What is a Biomarker?

Biomarker is a substance that is measured objectively and evaluated as an indicator of normal biologic processes, pathogenic processes, or pharmacological responses to a therapeutic intervention.

Q.2 What are the various diagnostic tools to measure periodontal disease at the clinical, tissue, cellular and molecular level?

	Levels	*Findings*	*Diagnostic tools*
1	Clinical level	Attachment loss, bone loss	Periodontal probing, radiographs
2	Tissue level	Down growth of junctional epithelium, bone and connective tissue loss	Histomorphometry Immunohistochemistry
3	Cellular level	Inflammatory cell activation Neutrophil activation Osteoclast activation	ELISA Immunohistochemistry
4	Molecular level	Activation of receptors for endotoxins CD-14; Toll-like receptors	PCR DNA – DNA Hybridization, Laser-capture microdissection

Q.3 What are the various diagnostic aids for detecting bacteria?

(i) Dark ground microscopy
(ii) Staining method
(iii) Phase contrast microscopy
(iv) Culture techniques
(v) Immunodiagnostic method
(vi) DNA probes
(vii) Enzymatic method of bacterial identification
(viii) Restriction endonuclease analysis
(ix) Polymerase chain reaction (PCR).

Q.4 What are the uses of diagnostic test?

Diagnostic procedures may be used to:

(i) Identify people at risk of developing disease – At risk
(ii) Detect early stage disease in clinically asymptomatic individuals – Screening

(iii) Classify disease categories – Classification
(iv) Predict likely responders to specific treatments – Treatment Planning
(v) Monitor treatment efficacy and detect disease recurrence - Monitoring

Q.5 What are the sources of sample for diagnostic purposes?

(i) Blood
(ii) Saliva
(iii) Subgingival plaque
(iv) Gingival crevicular fluid
(v) Gingival crevicular cells
(vi) Urine.

Q.6 What are the methods to collect subgingival plaque samples?

(i) Curettes
(ii) Paper points.

Q.7 What are the main candidates in the search for biomarkers?

(i) Bacteria
(ii) Inflammatory and immune products
(iii) Enzymes released from dead cells
(iv) Connective tissue degradation products
(v) Products of bone resorption.

Q.8 Which are the inflammatory mediators and products of GCF?

(i) Cytokines:
- Tumor necrosis factor alpha (TNF-α)
- Interleukin 1 α (IL-1 α)
- IL-1β
- IL-6
- IL-8

(ii) Prostaglandin:
- PGE_2

Q.9 Which are host-derived enzymes of GCF?

(i) Asparate aminotransferase
(ii) Elastase
(iii) β-glucuronidase
(iv) Alkaline phosphatase
(v) Arylsulphatase
(vi) Neutral proteases
(vii) Cathepsins
(viii) Lactate dehydrogenase
(ix) Matrix metalloproteinases
(x) Myeloperoxidase.

Q.10 Which are the enzymes released by dead cells?

(i) Aspartate aminotransferase (AST)
(ii) Lactate dehydrogenase (LDH)

Q.11 Which are the connective tissue breakdown products?

(i) Collagen
  (a) Hydroxyproline
  (b) Collagen cross links
  (c) N-peptide
(ii) Proteoglycans
  (a) Glycosaminoglycans (GAGs)
  (b) Heparan sulphate
  (c) Chondroitin 6 - sulphate
  (d) Chondroitin 4 - sulphate
(iii) Fibronectin.

Q.12 Name the various bone formation markers.

(i) Type I procollagen propeptide proliferation -
  (a) C-terminal propeptide fragment (PICP)
  (b) N-terminal propeptide fragment (PINP)
(ii) Alkaline phosphatase -
  (a) Total alkaline phosphatase (Al-p)
  (b) Bone alkaline phosphatase (BAl-p)
(iii) Osteocalcin, Bone Gla protein mineralization (BGP) -
  (a) C-terminal fragment
  (b) Mid portion
  (c) Intact.

Q.13 Name the various bone resorption markers.

(i) Pyridinium cross-link -
  (a) Urine pyridinoline (PYP), deoxypyridinoline (DPD), HPLC method
  (b) Urine free deoxypyridinoline (fDPD).
(ii) Pyridinium cross-link collagen peptide fragment -
  (a) Serum C-terminal telopeptide (ICTP)
  (b) Urine C-terminal telopeptide (CTx)
  (c) Urine N-terminal telopeptide (NTx)
(iii) Tartrate-resistant acid phosphatase (TRAP)
(iv) Galactosyl hydroxylysine (GHYL)
(v) Hydroxyproline
(vi) N-terminal osteocalcin fragment
(vii) Glycosaminoglycans (GAGs).

Q.14 What are the aims of microbial analysis in periodontics?

(i) To discriminate between different microbial types of periodontal infections.

(ii) To select subjects likely to benefit from adjunct systemic antimicrobial system.

(iii) To assist in selecting the most appropriate antibiotic treatment in accordance with the composition of subgingival microflora.

(iv) To contribute in minimizing the overuse of potent antimicrobials.

Q.15 Why is there inconsistency in microbiological diagnostic test results?

(i) Technical difficulties:
- Sample taking
- Dispersion of plaque sample
- Difficulties in cultivation of plaque microorganisms
- Characterization and identification of isolates

(ii) Conceptual problems:
- Complexity of the microbiota
- Mixed infections
- Opportunistic infections

(iii) Problems associated with the nature of periodontal diseases:
- Disease activity
- Inability to differentiate between diseases in different subjects
- The possibility of multiple diseases within a subject

(iv) Data analysis.

Q.16 Which microorganisms can be detected through DNA probe?

A. actinomycetemcomitans, P. gingivalis, B. intermedius, C. rectus, E. corrodens, T. denticola and F. nucleatum.

Q.17 What is Omnigene?

It is DNA probe system for a number of subgingival bacteria. A paper point sample of subgingival plaque is placed in the container provided and mailed off to the company for assay.

Q.18 Which microorganisms can be detected through BANA test?

Treponema denticola, P. gingivalis, Tannerella forsythia and Capnocytophaga.

Q.19 What is Perioscan?

This is a chair side diagnostic test kit system, which utilizes the BANA test for bacterial trypsin - like proteases.

Q.20 What is Evalusite?

It is a chairside kit consisting of enzyme linked immunosorbent assays (ELISA) using antibodies to detect antigens. It is used to detect *A. actinomycetemcomitans, P. gingivalis* and *P. intermedius.*

Q.21 What are the advantages and disadvantages of culturing medium?

Advantages:

(i) Can obtain relative and absolute count of the cultured species.

(ii) Able to assess for antibiotic susceptibility of microbes.

Disadvantages:

(i) Putative pathogens such as Treponemas and Tannerella forsythia are fastidious and difficult to culture

(ii) Strict sampling and transport conditions are essential

(iii) Time consuming

(iv) Expensive

(v) Sophisticated equipments and experienced personnel are required

(vi) Can only grow live bacteria.

Q.22 What are the advantages and disadvantages of dark field microscopy?

Advantages:

(i) Spirochetes, are difficult to stain

(ii) Assesement can be done during the progression and treatment of the disease.

Disadvantages:

(i) Unable to identify non-motile species

(ii) Unable to differentiate among the various species of Treponema

(iii) Inability to speciate microorganisms

(iv) Inability to determine their relative susceptibility to antimicrobial agent.

Q.23 What are the drawbacks of BANA test?

(i) Lack of quantitative data.

(ii) Inability to determine which of the three bacteria are responsible for the enzyme production.

(iii) BANA system does not include inhibitors of host proteinases, which could cleave this substrate and could also contaminate the bacterial sample tested.

(iv) Cannot identify the presence of other pathogens that do not produce trypsin like enzymes.

Q.24 What are the advantages and disadvantages of Immunoassays test?

Advantage:

(i) Identify dead target cells, thus not requiring stringent sampling and transport methodology.

Disadvantages:

(i) Local sampling cannot be done, so site-specific disease parameters cannot be assessed.

(ii) Immunoassays cannot be used to determine bacterial virulence.

(iii) Cannot be used to determine antibiotic susceptibility.

Q.25 What are Reactive Oxygen Species (ROS)?

ROS is a collective term, which includes –

(i) Oxygen derived free radicals: Super oxide (O_2^-), Hydroxyl (OH^-) and Nitric oxide (NO)

(ii) Non-radical derivatives of oxygen: Hydrogen peroxide (H_2O_2) and Hypochlorous acid (HOCL).

Q.26 What are various Reactive Oxygen Species (ROS)?

(i) Superoxide
(ii) Hydroxyl
(iii) Perhydroxyl
(iv) Alkoxyl
(v) Aryloxyl
(vi) Arylperoxyl
(vii) Peroxyl
(viii) Acyloxyl
(ix) Hydrogen peroxide
(x) Hypochlorous acid
(xi) Ozone
(xii) Singlet oxygen.

Q.27 What are the sources of ROS?

A. Exogenous sources – Heat, trauma, ultrasound, UV light, ozone, smoking, exhaust fumes, radiation, infection, excessive exercise and therapeutic drugs.

B. Endogenous sources –

(a) Byproducts of metabolic pathways – electron leakage from mitochondrial electron transport system forming superoxide.

(b) Functional generation of host defense cells (phagocytes) and cells of the connective tissues (osteoclasts and fibroblasts).

Q.28 Classify Antioxidants.

A. According to mode of action:

(a) Preventive: Superoxide dismutase enzymes (1, 2, 3), Catalase, Glutathioneperoxidase, DNA repair enzyme, Polymerase, Albumin, Lactoferrin, Transferrin, Haptoglobin, Carotenoids, Uric acid.

(b) Scavenging (Chain breaking): Vitamin C, Carotenoids, Uric acid, Albumin, Bilirubin, Polyphenols, Reduced glutathione.

B. According to location:

(a) Intracellular: Superoxide dismutase enzymes 1 and 2, DNA repair enzyme

(b) Extracellular: Superoxide dismutase enzyme 3, Lactoferrin, Transferrin, Haptoglobin, Carotenoids, Uric acid.

c. Membrane associated: Alpha – Tocopherol

C. According to solubility -
   a. Water soluble: Haptoglobin, Albumin, Uric acid, Ceruloplasmin
   b. Lipid soluble: Alpha – Tocopherol, Carotenoids, Bilirubin.

D. According to structures they protect -
   a. DNA protective antioxidants: Superoxide dismutase enzymes 1 and 2, DNA repair enzyme, Reduced glutathione, Cysteine.
   b. Protein protective antioxidants: Sequestration of transition metals by preventive antioxidants.
   c. Lipid protective antioxidants: Alpha-tocopherol, Carotenoids, Bilirubin, Reduced glutathione.

E. According to their origin –
   a. Exogenous: Carotenoids, Ascorbic acid, Folic acid, Cysteine, Alpha-tocopherol.
   b. Endogenous: Catalase, Superoxide dismutase, Transferrin, Ceruloplasmin.
   c. Synthetic: N-acetyl cysteine, Tetracyclines, Penicillamine.

Q.29 What is RANKL?

Receptor activator of nuclear factor kappa β Ligand is a cytokine essential for osteoclastogenesis, which is expressed by osteoblast. Osteoclast precursor express RANK (a receptor of RANKL) and recognizes RANKL expressed by osteoblasts through cell to cell interaction and differentiate into osteoclasts in the presence of macrophage colony-stimulating factor.

Q.30 What is osteoprotegerin?

It is a soluble decoy receptor for RANKL, produced mainly by osteoblasts. It blocks osteoclastogenesis by inhibiting RANKL – RANK interaction.

Q.31 What is matrix metalloproteinases (MMPs)?

Matrix metalloproteinases (MMPs) are a family of homologous Zn(++) endopeptidases that collectively cleave most, if not all of the constituents of the extracellular matrix.

Q.32 What are the various types of MMPs?

There are about 28 MMPs:

Protease	*MMP Number*	*Matrix substrate*
Collagenase – 1	MMP – 1	Collagen
Collagenase – 2	MMP – 8	Collagen
Collagenase – 3	MMP – 13	Collagen
Collagenase – 4	MMP – 18	Collagen
Gelatinase – A	MMP – 2	Gelatin, Elastin

Gelatinase – B	MMP – 9	Gelatin, Elastin
Stromelysin – 1	MMP – 3	Laminin, fibronectin, non-triple helical region of collagen types II and III
Stromelysin – 2	MMP – 10	—
Stromelysin – 3	MMP – 11	Fibronectin
Matrilysin	MMP – 7 (Smallest)	Aggrecan, Laminin
Enamelysin	MMP – 20	Amelogenin

Mechanical Plaque Control

Q.1 Write the historical background of toothbrush.

1600 - Bristle toothbrush appear in China
1728 - Pierre Fauchard in his book *'The Surgeon Dentist'* advocated wet sponges and specially prepared herb roots
1780 - William Addis of England made the first toothbrush
1840 - England, France and Germany started producing bristle toothbrush
1857 - HN Wadsworth patented the first American toothbrush
1900 - Celluloid handles were used
1919 - AAP defined specifications
1938 - Nylon was first applied to toothbrush construction
1939 - Synthetic were substituted for natural materials.

Q.2 What are the ADA specifications for the toothbrush?

Brushing Surface:

Length - 1-1.25 inches
Width - 5/16-3/8 inches
Rows - 2-4 rows of bristles
Tufts - 5-12 per row

Q.3 What are the various dimensions of toothbrush?

(i) Handle:
- Adult - 6 inches
- Junior - 1/6th smaller than adult size
- Child - 1/3rd smaller than small size

(ii) Head Size:
- Length - 1-1¼ inch
- Width - 5/16-3/8 inch

(iii) Bristle length/height - 7/16 inches

(iv) Filament diameter:
- Soft - 0.2 mm/.007 inches
- Medium - 0.3 mm/.012 inches
- Hard - 0.4 mm/.014 inches

Filament stiffness α diameter^2/length^2

Q.4 How toothbrushes are cleaned?

(i) Hold the brush head under strong stream of warm water to remove dentifrices and bacteria present between filaments.
(ii) Tap the brush handle on edge of sink to remove excess water.
(iii) Use another toothbrush to clean one toothbrush to remove resistant debris.
(iv) Keep brush in open air with head in an upright position, apart from contact with other brush.

Q.5 After how much time toothbrush should be changed?

Brush should be replaced before filaments fray, atleast every 2-3 months. But patients who are debilitated, have a known infection or are about to undergo surgery should be advised to disinfect their brushes or use disposable brushes.

Q.6 What are the indications of powered toothbrush?

(i) Those who wear orthodontic appliances
(ii) Children and adolescents
(iii) Those undergoing complex restorative and prosthodontic treatment
(iv) Those with dental implants
(v) Patients with physical or mental disabilities
(vi) Hospitalized patients, elder ones who need to have their teeth cleaned by caregivers
(vii) Poorly compliant periodontal maintenance patient.

Q.7 What is a novel toothbrush design?

The head is located horizontal to the tooth surface, multiple tufts of bristles are angled in the different directions of the approximal tooth surfaces. This design aims of improving plaque removal in approximal areas and based on the fact that the majority of the subjects use a simple horizontal brushing action.

Q.8 What are the various toothbrushing methods?

(i) Roll:
  (a) Roll method
  (b) Modified Stillman
(ii) Vibratory:
  (a) Stillman
  (b) Charter
  (c) Bass
(iii) Sulcular: Bass
(iv) Simultaneous Sulcular: Collis
(v) Circular: Fones
(vi) Vertical: Leonard
(vii) Horizontal: Scrub
(viii) Physiologic: Smith

Q.9 Write indications of various toothbrushing methods.

(i)	Modified Stillman method:	In areas with progressing gingival recession and root exposure to minimize abrasive tissue destruction.
(ii)	Charters method:	(a) Aid in plaque removal from proximal tooth surfaces when interproximal tissue is missing, for example, following periodontal surgery. (b) Cleaning in orthodontic appliances patient. (c) Remove bacterial plaque from abutment teeth and under the gingival border of a fixed partial denture (bridge) or from the undersurface of sanitary bridge.
(iii)	Bass method:	(a) For open interproximal areas, cervical areas beneath the height of contour of the enamel and exposed root surfaces. (b) Recommended for any patient with or without periodontal involvement.
(iv)	Fones method:	School children / young children because of simplicity.
(v)	Scrub:	Very young child to get feeling of brushing his teeth.
(vi)	Roll:	Meant for general cleaning in conjunction with the use of a vibratory technique.

Q.10 What are the advantages of Bass toothbrushing method?

(i) It cleans the gingival sulcus.
(ii) It also cleans the interproximal and cervical portion of teeth.

Q.11 What is the other name of Bass toothbrushing method?

Intrasulcular method.

Q.12 What is the difference between Bass and Modified Bass toothbrushing method?

In modified Bass method, bristles are swept towards the occlusal surface after completing the vibratory motion in the gingival sulcus.

Q.13 What is the sequence of toothbrushing?

(i) Maxillary teeth first, then mandibular to avoid the deposition of loosened debris from maxillary teeth on brushed mandibular teeth.
(ii) Start brushing from a molar region of one arch around to the opposite side, then back around the lingual / facial. Repeat in the opposing arch.

(iii) Each brush placement must overlap the previous one for thorough coverage.
(iv) Encourage the patient to begin brushing the area that are most frequently missed or most difficult for brush placement.
(v) Sequence be varied at least once each day.

Q.14 What are deleterious effects of overzealous horizontal brushing?

(i) Gingival recession
(ii) Bacteremia
(iii) Abrasion, wedge-shaped defects in the cervical area of root surfaces
(iv) Painful ulceration of the gingiva.

Q.15 What are the various factors, which should be taken into consideration while recommending an interdental cleaning methods?

(i) Type and size of the interproximal embrassure
(ii) Contour and consistency of gingival tissues
(iii) Tooth position and alignment
(iv) Ability and motivation of patient
(v) Presence of orthodontic appliance or fixed prostheses
(vi) Presence of furcations lesions.

Q.16 What is approximal and interproximal areas?

(i) Approximal areas are the visible spaces between teeth that are not under the contact area.
(ii) Interproximal areas refer to the area under and related to the contact point.

Q.17 Name various interdental cleaning aids.

(i) Interdental brushes
(ii) Dental floss
(iii) Interdental tips, Wooden tips, Rubber tips, and Plastic tips
(iv) Dental tape.

Q.18 Classify Interproximal embrassures and accordingly Interdental cleanser.

Embrassures	**Interdental cleanser**
Type 1 embrassures - No gingival recession	Dental floss
Type 2 embrassures - Moderate papillary recession	Interdental brush
Type 3 embrassures - Complete loss of papillae	Unitufted brush

Q.19 What are the indications for recommending Dental floss and tape, Toothpick, Interproximal brush, Single tufted brush and Tongue scraper?

(i) Dental floss and tape – They are recommended in patients where interdental papillae completely fill the embrassure space.
(ii) Toothpick - They are recommended in patients with open interdental spaces as secondary prevention for periodontal disease.
(iii) Interproximal brush - They are recommended in exposed root surfaces having concavities or grooves, through and through furcation.
(iv) Single tufted brush - Recommended in furcation areas, distal surfaces of the most posterior molars.
(v) Tongue scraper - Indicated in high caries risk, periodontal risk patients and patients suffering from halitosis

Q.20 What are the advantages of interdental brushes over dental floss?

(i) Interdental brushes clean concave root surface and furcations more efficiently than dental floss.
(ii) Interdental brushes are much easier to use than dental floss.

Q.21 How is dental floss used?

(i) 12-18 inches of dental floss is wrapped around fingers or ends may be tied together in a loop.
(ii) The floss is stretched tightly between thumb and forefinger or between both forefingers and is passed through each contact area with a firm back and forth motion.
(iii) Wrap the floss around the proximal surface of one tooth, once it is placed apical to contact area between the teeth. The floss is then moved firmly along the line angle of tooth upto the contact area and gently down into sulcus, with repeated up and down strokes several times.

Q.22 How is dental tape used?

Dental tape is used with fluoride dentifrice which is recommended for cleaning the approximal surfaces of molars and premolars in children and adults with a rubbing motion, holding the tape by the hand / in a special holder.

Q.23 What are fluoridated wooden toothpicks?

Wood can store sodium fluoride crystals both on the surface and in porosites. NaF crystal dissolves readily in contact with liquids such as water / saliva. The toothpicks should be moistened in the saliva for a few seconds just before use to accelerate the release of fluoride.

Q.24 Where is toothpick contraindicated?

Toothpick is contraindicated in children and young adults because interdental space is filled by a normal papilla.

Q.25 Name various adjunctive aids used for cleansing oral cavity.

(i) Irrigators
(ii) Tongue scraper
(iii) Dentifrices.

Chemotherapeutic Agents

Q.1 Name various drugs used in periodontal therapy.

(i) Antibiotics
(ii) Analgesics
(iii) Sedatives
(iv) Muscle Relaxants
(v) Postoperative periodontal dressings
(vi) Desensitizing agents
(vii) Mouthwashes
(viii) Dentifrices
(ix) Disclosing solutions
(x) Corticosteroids
(xi) Hemostatics and vasoconstrictors
(xii) Anesthetics.

Q.2 Name various chemotherapeutic agents used as premedication in periodontal surgery.

(i) Anxiolytics
(ii) Antibiotics
(iii) Antiseptics
(iv) NSAIDS.

Q.3 What is disclosing agent?

Disclosing solution contains a dye or other coloring substance, which imparts its color to calculus, plaque and films on the surface of teeth, tongue and gingiva.

Q.4 What are the uses of disclosing agents?

(i) Excellent oral hygiene aids because they can provide the patient with additional motivational tool to improve the efficiency of plaque control procedures.
(ii) To conserve operating time by making inconspicuous deposits more evident.

O.5 What are the factors to be considered in the selection of a disclosing solution?

(i) Intensity of color
(ii) Taste

(iii) Irritation to mucous membrane
(iv) Diffusibility - neither too thin nor too thick
(v) Astringent and antiseptic.

Q.6 Name various disclosing agents.

(i) Skinner iodine solution
(ii) Iodine disclosing solution
(iii) Diluted tincture of iodine
(iv) Berwick's solution
(v) Buckley's solution
(vi) Talbot iodoglycerol
(vii) Metaphen
(viii) Basic fuschin
(ix) Bismarck Brown
(x) Easlick's solution
(xi) Bender's solution
(xii) Mercurochrome solution
(xiii) Erythrosin (FDC red No. 3)
(xiv) DC yellow no. 8 fluorescein
(xv) Two tone dye (FDC red no. 3 and FDC green no. 3)

Q.7 Which disclosing agents differentiate between old or thick plaque accumulations and recent or thin plaque accumulations?

Two - tone dye test that uses FDC red no. 3 and FDC green no. 3. The solution stains thick accumulation of plaque as blue and thin deposits are stained red / pink.

Q.8 What are the constituents of Skinner Iodine solution?

(i) Iodine crystals - 3.3 gm
(ii) Potassium iodide - 1.0 gm
(iii) Zinc iodide - 1.0 gm
(iv) Distilled water - 16 ml
(v) Glycerin - 16 ml

Q.9 What are the constituents of Bismarck Brown solution?

(i) Bismarck brown (powder) - 3 g
(ii) Ethyl alcohol - 10 ml
(iii) Glycerin - 120 ml
(iv) Anise oil - 1 drop

Q.10 Write limitations of disclosing agent.

(i) Do not selectively disclose bacteria plaque, but rather stain all soft debris and pellicle.
(ii) Exposed cementum in particular can stain vividly although it is free of bacterial plaque.

(iii) Disclosing solution may stain silicate cement or resin restoration.
(iv) Disclosing solutions containing alcohol should not be kept for more than 2-3 months since the alcohol will evaporate and render the solution too highly concentrated.

Q.11 What are the constituents of Dentifrices?

			Powder	*Paste/Gel*
1.	Abrasives	Calcium Carbonate Calcium Phosphate Hydrated alumina Hydrated silica	90-98%	20-25%
2.	Binder	Carrageenan	0	3%
3.	Detergent	Sodium lauryl sulphate	1-6%	1-2%
4.	Humectants	Sorbitol Glycerin	0	20-35%
5.	Colorants	Food colorants	1-2%	1-2%
6.	Flavoring	Oil of peppermint Spearmint Wintergreen Cinnamon	1-2%	1-2%
7.	Fluorides	NaF SnF_2	0	0-1%
8.	Tartar control agents	Disodium pyrophosphate Tetrasodium pyrophosphate Tetrapotassuim pyrophosphate	0	0-1%
9.	Water		0	15-25%
10.	Desensitizing agents	Potassium nitrate Strontium chloride	0	0-5%

Q.12 Name various topical hemostatic agent.

	Agent	*Main constituent*
(i)	Avitene	Collagen
(ii)	Collacote	Microfibrillar
(iii)	Collacope	Collagen
(iv)	Collaplug	Collagen
(v)	Thrombinar	Thrombin
(vi)	Thrombogen	Thrombin
(vii)	Thrombostat	Thrombin
(viii)	Gelfoam	Gelatin
(ix)	Beriplast	Fibrin
(x)	Surgicel	Cellulose
(xi)	Cyclo kapron	Tranexamic acid

Q.13 Classify periodontal dressings.

(i) Zinc oxide eugenol
(ii) Zinc oxide non-eugenol
  (a) Coepak
  (b) Periocare
  (c) Periopac
  (d) Perioputty
  (e) Vocopac
(iii) Others
  (a) Photocuring periodontal dressing (Barricaid)
  (b) Collagen dressings
  (c) Methacrylic gel
  (d) Cyanoacrylate.

Q.14 What are various Generations of antimicrobials?

(i) First generation agents: Poor substantivity and thus used 4-6 times daily. Reduces plaque score by 20-50%, e.g Sanguinarine, Quaternary Ammonium compounds, Antibiotics.
(ii) Second generation agents: Reduces plaque score by 70-90%. Used twice daily, e.g Chlorhexidine, Triclosan.
(iii) Third generation agents: Effective against specific periodontal pathogens, e.g Delmopinol.

Q.15 What are the indications for use of antibiotics in periodontal therapy?

(i) Therapeutic therapy: Aims to treat established clinical infection.
(ii) Prophylactic therapy: Involves administration of antimicrobial agents to the individuals susceptible to a clinical disease. Prevention of infective endocarditis is a prime example of prophylactic antibiotic therapy.
(iii) Pre-emptive therapy: Involves antimicrobial therapy to individuals prior to the onset of clinical disease based on clinical, epidemiological or laboratory indications of disease risk. Pre-emptive therapy may include A.a from younger siblings of adolescents having localized aggressive periodontitis / from children of parents with localized aggressive periodontitis.

Q.16 Compare Local and Systemic antimicrobial therapy.

		Local	*Systemic*
1	Effective range	Narrow	Wide
2	Drug Concentration	High dose at the site	Low
3	Compliance	Less	More
4	Systemic side effects	Less	More
5	Cost	Expensive	Inexpensive
6	Drug dose	Less	More
7	Therapeutic potential	Act better locally on biofilm associated microorganisms	Reach widely distributed microorganisms better
8	Super Infections	More chances	Less chances

Q.17 What is the difference between antiseptic and disinfectant?

Antiseptic is applied topically or subgingivally on living surfaces where as disinfectant is applied to inanimate surfaces to destroy microorganisms.

Q.18 What are the various approaches of chemical supragingival plaque control?

(i) Antiadhesive – Prevents bacterial attachment, e.g. chlorhexidine, delmopinol, amine alcohol

(ii) Antimicrobial – Stop / slow bacterial proliferation, e.g. chlorhexidine, antibiotics

(iii) Established Plaque removal / chemical tooth brush, e.g amine alcohol, enzyme

(iv) Antipathogenic – Alter the pathogenicity of plaque.

Q.19 Write various chemical antiplaque agents.

(i)	Antibiotics	–	Penicillin, vancomycin, kanamycin
(ii)	Bisbiguanide antiseptics	–	Chlorhexidine, alexidine, octenidine
(iii)	Phenols and essential oils	–	Thymol, triclosan, hexylresorcinol
(iv)	Natural products	–	Sanguinarine
(v)	Quaternary ammonium compounds	–	Cetylpyridinium chloride
(vi)	Oxygenating agents	–	Hydrogen peroxide, Sodium peroxy borate, Sodium peroxycarbonate
(vii)	Detergents	–	Sodium lauryl sulfate
(viii)	Enzymes	–	Protease, lipase, nuclease, dextranase, mutanase-glucose oxidase, amyloglucosidase
(ix)	Amine alcohol	–	Octapinol, delmopinol
(x)	Metal salts	–	Tin, zinc, copper
(xi)	Fluorides	–	Sodium fluoride, stannous fluoride, amine fluoride.

Q.20 Classify chemical antiplaque agents.

(i)	Cationic plaque control agents	–	Bisbiguanide, quaternary ammonium compounds, heavy metals, pyrimidines, herbal extracts
(ii)	Anionic plaque control agents	–	Sodium lauryl sulfate
(iii)	Non-ionic plaque control agents	–	Phenol, thymol, listerine, triclosan

(iv) Other plaque control agents – Delmopinol, enzymes
(v) Combination of plaque control agents – Heavy metal ions and detergents.

Q.21 What are the advantages of chemical plaque control over mechanical plaque control?

The advantages of chemical approach is that zone of diffusion achieved with chemical agent is greater than the limited radius of effect of a mechanical agent.

Q.22 Which are the antiplaque agents accepted by FDA for treatment of gingivitis?

(i) Chlorhexidine - Prescription drug
(ii) Listerine - Over the counter / non-prescription drug
In September 1987, Listerine antiseptic mouthwash was the first non-prescription product to be awarded the ADA council on Dental Therapeutic seal of acceptance as an aid in controlling supragingival dental plaque.

Q.23 What is the composition of Listerine mouthwash?

It is phenol related essential oils:

(i) Thymol – 0.064%
(ii) Eucalyptol – 0.092%
(iii) Methanol – 0.042%
(iv) Methyl salicylate – 0.060% in hydrochloride solution.
(v) Benzoic acid – 0.15 %

Q.24 What are the advantages and disadvantages of Listerine?

Advantages:

(i) Listerine benefits patients who do not tolerate the taste or staining of chlorhexidine.
(ii) It is less expensive.
(iii) It is easier to obtain than chlorhexidine, as it is sold over the counter.

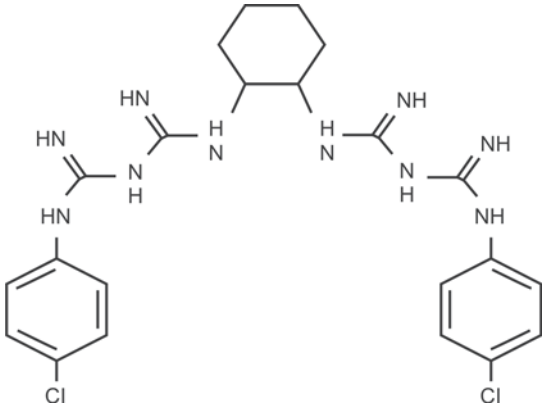
Disadvantages:

(i) It is high in alcohol concentration (ranging from 21.6-26.9%), which may exacerbate xerostomia.
(ii) It is generally contraindicated in patients under the treatment of alcoholism who take antabuse (disulfiram).

Q.25 Who developed Chlorhexidine (CHX)?

Chlorhexidine was developed by Imperial Chemical Industries, England in 1940s and marketed in 1954 as an antiseptic for skin wound.

Q.26 Describe the structure of Chlorhexidine molecule.



It is symmetrical molecule consisting of:

(i) Two 4 chlorophenyl rings
(ii) Two biguanide groups
(iii) A central hexamethylene bridge connecting chlorophenyl and biguanide groups.

Q.27 What is the mechanism of action of Chlorhexidine?

I. **Antibacterial activity:**
Cationic chlorhexidine is rapidly attracted to the negatively charged bacterial cell surface, with specific and strong adsorption to phosphate containing compounds.

II. **Antiplaque activity:**
(a) It blocks the acidic groups on the salivary glycoproteins thus inhibit pellicle formation.
(b) It directly binds to the bacterial surface in sublethal amounts, thus prevents the adsorption of bacteria onto tooth surface.
(c) It inhibits acid production in established plaque.

Q.28 What is the peculiar feature of Chlorhexidine?

Substantivity – The quality of prolonged contact time between a substance and substrate is known as substantivity. It is influenced by the concentration of the medication, its pH and temperature and length of contact of the solution with oral surfaces.

Q.29 What are the clinical uses of chlorhexidine?

(i) Presurgical preparation of periodontal patients
(ii) Post oral surgery including periodontal surgery / root planing
(iii) In patients with jaw fixation
(iv) Medically compromised patients predisposed to oral infections
(v) Mentally and physically handicapped patients
(vi) High caries risk patients

(vii) Recurrent oral ulceration
(viii) Removable and fixed orthodontic appliance wearers
(ix) In the denture stomatitis patient
(x) Preoperative rinsing during ultrasonic scaling and polishing with speed instruments.

Q.30 What are the adverse effects of Chlorhexidine?

(i) Staining- Brown discoloration of teeth, restoration and dorsum of tongue
(ii) Taste alteration
(iii) Oral mucosal erosion
(iv) Increased calculus formation
(v) Unilateral or bilateral parotid swelling.

Q.31 Write mechanism of Chlorhexidine staining.

(i) Degradation of CHX molecule to release parachloraniline
(ii) Precipitation of anionic dietary chromogens
(iii) Protein denaturation with metal sulfide formation
(iv) Catalysis of Maillard reactions.

Q.32 What instructions are given to patient after prescribing CHX mouthwash?

(i) The patient is asked to brush with dentifrice atleast after half an hour because of the binding of cationic CHX to anionic components of the dentifrice. There is reduction in the activity by decreasing the number of active cationic sites (Addy 1989).
(ii) Advice the patients using CHX mouthwash to avoid the intake of tea, coffee and red wine for the duration of use.

Q.33 What is PerioChip?

It is a degradable, baby's thumbnail size of 4 × 5 × 0.35 mm orange color chip composed of hydrolyzed gelatin matrix, cross-linked with glutaraldehyde and also contain glycerin and water into which 2.5 mg Chlorhexidine gluconate is incorporated.

Q.34 What is Chlo- Site?

It is new, simple and efficient safe way to treat periodontal pockets which is active for minimum of 15 days. It is a gel with 1.5% Chlorhexidine. It is a combination of chlorhexidine gluconate – slow release (0.5%) and chlorhexidine dihydrochloric- rapid release (1.0%). This combination makes bactericidal activity 40-60 times greater than MIC for bacteria.

Q.35 What is the advantage of Triclosan over CHX?

Triclosan is not significantly impaired by the presence of SLS (sodium lauryl sulphate) as CHX do. Because most commercial toothpastes and mouthrinses contain SLS.

Q.36 Compare the substantivity of chlorhexidine, sodium lauryl sulfate, triclosan, cetylpridinium chloride, hexetidine and povidine iodine.

(i) Chlorhexidine — around 12 hours
(ii) Sodium lauryl sulfate — around 5-7 hours
(iii) Triclosan — around 5 hours
(iv) Cetylpyridinium chloride — around 3-5 hours
(v) Hexetidine — around 1-3 hours
(vi) Povidine Iodine — around 1 hour.

Q.37 What is Sanguinarine?

It is a benzophenathridine alkaloids derived from alcoholic extraction of powered rhizosomes of blood root plant *Sanguinaria Canadensis*, which grows in USA and Canada. After precipitation and putrefaction of alcohol extract, an orange powder containing 30-35% of Sanguinarine is obtained. The trade name of Sanguinarine is *VIADENT*.

Q.38 What is Viokase?

It is a dehydrated pancreas preparations, which contain trypsin, chymotrypsin, carboxypeptidase, amylase, lipase and nucleases.

Q.39 Name the combination of drugs that has both therapeutic as well as prophylactic benefit.

Metronidazole and ciprofloxacin combination—

Therapeutic benefit as metronidazole targets obligate anaerobes and ciprofloxacin acts against facultative anaerobes.

Prophylactic benefit as ciprofloxacin has minimal effect on Streptococcus species, which is responsible for periodontal health.

Q.40 What are the advantages of Ciprofloxacin over other antibiotics for combating Aggressive periodontitis?

(i) It has minimal effect on Streptococcus species, which is associated with periodontal health.
(ii) All strains of *A. actinomycetemcomitans* are susceptible to ciprofloxacin.

Q.41 Write peculiar feature of Azithromycin.

Azithromycin penetrates fibroblasts and phagocytes in concentrations 100-200 times greater than extracellular compartment, thus transported and released directly into site of inflammation through phagocytes. Therapeutic dose—Initial loading dose of 500 mg followed by 250 mg / day for 5 days.

Q.42 Which are the drugs that concentrate in GCF and thus are effective against Periodontal pathogens?

		Local	*Systemic*
(i)	Doxycycline	-	2-8 µg / ml
(ii)	Tetracycline	Actisite 1300µg / ml	5-12 µg / ml
(iii)	Metronidazole	-	Approx 14 µg / ml
(iv)	Chlorhexidine	Periochip 100µg / ml	-
(v)	Clindamycin	-	1-2 µg / ml

Q.43 What are the major side effects associated with Clindamycin, Tetracycline, Metronidazole and Amoxicillin?

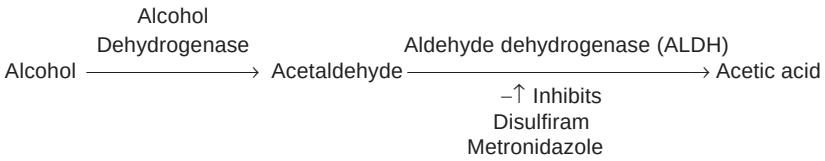
Clindamycin - Pseudomembranous collitis (due to *Clostridium difficile)* and hepatitis

Metronidazole - Antabuse effect when alcohol is ingested, metallic taste and GIT intolerance

Tetracycline - GIT intolerance and Candidiasis

Amoxicillin - Hypersenstivity.

Q.44 What is disulfiram like reaction or antabuse effect of alcohol?



Disulfiram or metronidazole irreversibly inhibits ALDH enzyme, which leads to accumulation of toxic levels of acetaldehyde in liver and systemic circulation causing vomiting, visual disturbances, postural fainting and circulatory collapse. So, alcohol and products containing alcohol should be avoided during metronidazole therapy and for at least 1 day after the drug is discontinued.

Q.45 What are the advantages of Doxycycline over other Tetracyclines?

(i) Better compliance, has to be given once daily

(ii) Calcium, antacids, and milk do not alter absorption.

Q.46 What are the advantages of Minocycline over other Tetracyclines?

(i) Better compliance, has to be given twice daily due to high lipid solubility

(ii) Less phototoxicity

(iii) Less renal toxicity.

Q.47 What is Host modulation? Write various host modulatory agents.

Host modulatory therapy is a treatment concept that aims to reduce tissue destruction and stabilize or even regenerate the periodontium by modifying or down regulating destructive aspects of the host response and upregulating protective or regenerative responses.

Host modulatory agents**:**

A. Inhibition of matrixmetalloproteinases (MMPs):
   Through chemically modified tetracyclines (CMTs)

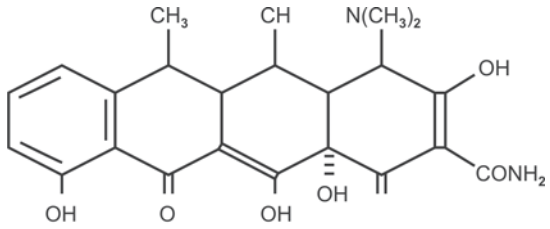
B. Inhibition of arachidonic acid metabolites
   (a) Through NSAIDS: COX – 1 inhibitors: Indomethacin, Naproxen, Flurbiprofen
   (b) COX – 2 inhibitors: Coxibs - Rofecoxib
   (c) COX and LOX inhibitors: Triclosan, Topical Ketoprofen
   (d) LOX inhibitors: Lipoxins

C. Modulation of bone metabolism:
   (a) Calcium supplementation
   (b) Hormone replacement therapy (HRT)
   (c) Bisphosphonates (Alendronate)

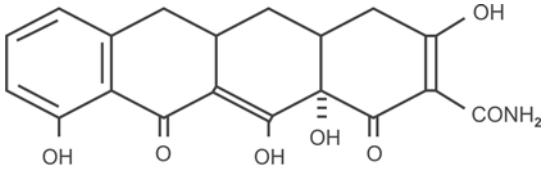
D. Regulation of immune and inflammatory responses:
   (a) Generation of protective antibiotics through vaccines
   (b) Nitric oxide inhibition
   (c) Suppressing proinflammatory cytokines (IL-1 and TNF-receptor antagonists)
   (d) Infusion / supplementary anti-inflammatory cytokines (IL-4 and IL-10).

Q.48 What are chemically modified Tetracyclines (CMT)?

Chemically modified Tetracyclines are those which lack dimethylamino group on the 4th carbon atom.



Doxycycline

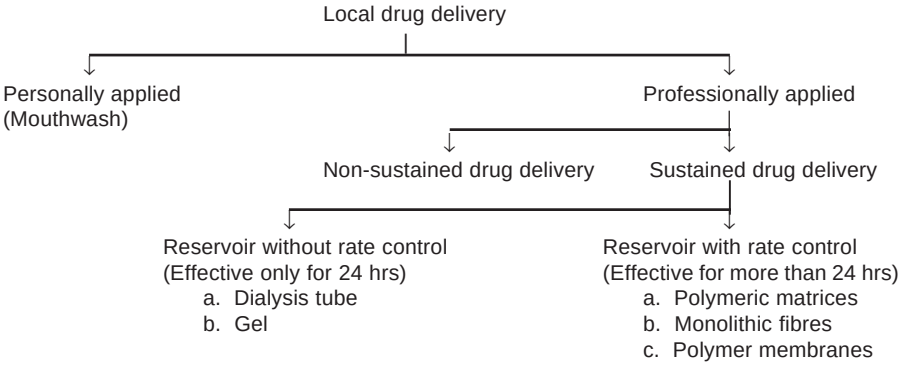


4-Dedimethylaminosancycline
CMT3

Q.49 What is the mechanism of action of chemically modified tetracyclines?

(i) Inhibits or chelates the calcium atoms that matrix metalloproteinase (MMPs) requires for their action
(ii) Inhibit already active MMPs
(iii) Down-regulate MMPs expression
(iv) Scavenges reactive oxygen species
(v) Modulates the osteoclast formation

Q.50 Classify Local Drug Delivery (LDD) System.



Q.51 Name various locally delivered antimicrobials for periodontal therapy.

	Trade name	*Antimicrobial agent*
(i)	Actisite	Tetracycline
(ii)	Atridox	Doxycycline
(iii)	Arestin	Minocycline
(iv)	Dentamycin / Periocline	Minocycline
(v)	Elyzol	Metronidazole
(vi)	Periochip	Chlorhexidine
(vii)	Atrigel	5% Sanguinarine

Q.52 Which are the drugs contraindicated in kidney failure and liver diseases?

	Kidney failure	*Liver diseases*
(i)	Aminoglycosides	(i) Tetracycline
(ii)	Tetracycline	
(iii)	Phenactin	

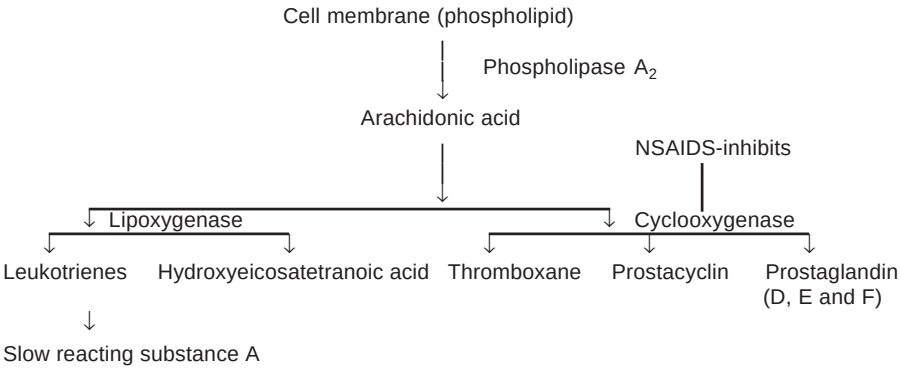
Q.53 Name drugs contraindicated in hypothyroidism.

(i) Sedatives
(ii) Narcotics.

Q.54 Name drugs contraindicated in pulmonary diseases.

(i) Narcotics
(ii) Sedatives
(iii) GA.

Q.55 What is mechanism of action of NSAIDS?



Q.56 What is Keyes technique? What are its advantages and disadvantages?

It consists of packing a mixture of NaCl, $NaHCO_3$ and H_2O_2 (3%) subgingivally and then irrigating the pockets with an antiseptic solution such as iodine solution, e.g. 0.5% Povidine solution.

Advantages:

(i) Freely available, inexpensive materials are used
(ii) Salt and H_2O_2 are powerful antiseptics
(iii) H_2O_2 is effective against anaerobic organisms
(iv) Frothing action of H_2O_2 aids in dislodging debris

Disadvantages:

(i) Minute burning spots can occur in some individuals which can be prevented by decreasing the concentration.
(ii) Use of this technique can give false impression that healing has occurred due to superficial resolution of hyperemia and edema.

Q.57 Which polymer inhibits the formation of plaque and stains on the surface of acrylic dentures?

Cetyl dimethicone copolymer

22 Periodontal Instruments and Instrumentation

A. PERIODONTAL INSTRUMENTS

Q.1 Classify periodontal instruments.

A. Non-surgical instruments:

According to the purpose they serve –

(a) Periodontal probes – locate, measure and mark pocket

(b) Explorers – locate calculus deposits and caries

(c) Scaling, Root planing and Curettage instruments

(i) Hand instruments
- Sickle scalers
- Curette
- Hoe
- Chisel
- File scalers

(ii) Ultrasonic and sonic

(iii) Rotating instruments

(iv) Reciprocating instruments

(v) LASERS

(d) Periodontal endoscope – to visualize deposits subgingivally in pocket and furcation.

(e) Cleaning and polishing instruments – to clean and polish tooth surface.

(i) Rubber cups

(ii) Brushes

(iii) Dental tape.

B. Surgical instruments:

According to the purpose they serve –

(a) Excisional and Incisional instruments

(i) Gingivectomy knives – used for excision and incision during gingivectomy. Kirkland 15-16, Crane – Kaplan 3 -4. Interdental – Orbans 1-2, Waerhaug, Merrifield 1, 2, 3 and 4.

(ii) Surgical blades – used for incisional purpose
Surgical blades No. 11,12,15 and 15C
(iii) Electrosurgical instruments - used during electrosection, electrocoagulation, electrofulguration, electrodessication

(b) Surgical curettes and sickle scaler – used for removing granulation tissue, subgingival deposits and interdental tissues during surgery
(i) Kramer curettes and Kirkland surgical instruments
(ii) Ball scaler

(c) Periosteal elevators - used to reflect periodontal flap
(i) Goldman fox 14
(ii) Glickman 24G
(iii) Prichard
(iv) Molt

(d) Surgical chisel and hoes - used for removing and reshaping bone.
(i) Wiedelstadt
(ii) Todd – Gilmore
(iii) Ochsenbein 1-2
(iv) Rhodes 36-37
(v) Fedi

(e) Surgical files - used for smoothening rough bony edges and removing bone
(i) Sugarman
(ii) Schluger

(f) Scissors and Nippers - used for removing tissue tags during gingivectomy and trimming the margins of flap
(i) Goldman fox 16
(ii) LaGrange
(iii) Dean Scissors

(g) Needle holders- used to suture the flap.
(i) Crile wood
(ii) Castroviejo
(iii) Thomas Walker
(iv) Osler Hegar
(v) Mathieu
(vi) Mayohegar
(vii) Kilner
(viii) Halsey
(ix) Rayden vascular.

Q.2 What are the various types of dental mirror surfaces?

(i) Plane / Flat surface mirror: Reflecting surface on the back of the mirror lens which produces double image.

(ii) Concave surface mirror: Produces magnified image that may be distorted.

(iii) Front surface mirror: Reflecting surface on the front of the lens that eliminates double image, producing a clear image.

Q.3 Which is the mirror of choice for dental procedures?

Front surface mirror

Q.4 What are the uses of dental mirrors?

(i) Indirect vision

(ii) Indirect illumination

(iii) Retraction

(iv) Transillumination.

Q.5 Explain various types of explorers.

(i) 17 – Orbans Explorer: Single ended instrument is ideal for calculus detection interproximally and in deep periodontal pocket. Long, thin shank and less than 2 mm fine tip allow easy access to deep, narrow pockets. Tip is right angle to shank.

(ii) 23 – Shepherd's hook Explorer : Single ended / paired with 17. It has thicker shank and working end than other explorers which makes it more rigid. Rigidity enhances the role in caries detection, but limits its role in calculus detection.

(iii) Pigtail / Cowhorn / 3CH Explorer no. 21 and 22 : Double ended instrument that is easily adapted throughout the mouth. Working end is curved and shank is thin for calculus detection. Because shank is curved and relatively short, instrument is best used in children / adults with minimal periodontal pocket depth less than 1 mm. It can also be used for detecting calculus in areas of furcation involvement (similar instrument design to Naber's probe), detecting proximal and cervical caries. Ineffective in evaluating occlusal caries because the instrument's design limits the force needed to determine occlusal caries.

(iv) 3A Explorer : It has a long, fine, arc like tip. It adapts well in deep pocket and furcation areas. Its fine tip allows for good tactile sensitivity, especially for calculus detection.

(v) Old Dominion University (ODU) 11 / 12 Gracey Type Explorer or EXD 11-12: Shank design of ODU is similar to that of Gracey 11 / 12 curette. It was developed by faculty of Old Dominion University, thus name ODU 11 / 12. It is a double ended, paired instrument for calculus detection.

Q.6 What is a periodontal probe?

It is a tapered rod like instrument caliberated in mm with a blunt, rounded tip.

Q.7 What are the uses of periodontal probe?

(i) To locate pocket depth
(ii) To mark pocket depth
(iii) To measure pocket depth
(iv) To determine pocket course and topography
(v) To measure width of attached gingiva
(vi) To measure clinical attachment level
(vii) To measure gingival recession
(viii) To check the bleeding on probing
(ix) To evaluate success and completeness of treatment
(x) To evaluate bone support in the furcation areas
(xi) To determine amount of bone loss that has occurred.

Q.8 What are the characteristic features of periodontal probe?

(i) Calibration: It must be accurately marked.
(ii) Thickness: A thinner probe slips through a narrow pocket more readily.
(iii) Readability: Aided by markings and color coding.

Q.9 Which mark should be taken as final reading, when the gingival margin appears at a level between two probe marks?

Higher mark should be taken as final reading.

Q.10 Which periodontal probes are not calibrated?

Merritt and Gilmore probes.

Q.11 What is the advantage of color band on some periodontal probes?

The colored band on the periodontal probe is designed to make periodontal examination readings more objective and faster.

Q.12 What are the factors affecting the accuracy of periodontal probing?

(i) Size of the probe
(ii) Angulation of the probe
(iii) Contour of the tooth and root surface
(iv) Probing force used
(v) Inflammatory state of the tissue.

Q.13 What are the various generations of periodontal probe?

(i) First generation; Conventional probes; Manual probes: The usual clinical instrument with a thin tapering line marked to be read in mm.
(ii) Second generation; Constant force probes; Pressure sensitive probes: As above, but with a spring or electronic cut-out when the appropriate force is reached. With force of 30 g, the probe tip remains on CEJ. Force of 50 g are necessary to diagnose osseous defects, e.g. Vine valley, Vivacare TPS.
(iii) Third generation; Automated probes: When probe is in place with specified force, a device is activated that reads the measurement

accurately. Automated and computerized probe, e.g Florida, Foster miller, Toronto automated.

(iv) Fourth generation; Three-dimensional probes: Currently under development, these are aimed at recording sequential probe positions along gingival sulcus.

(v) Fifth generation; Non-invasive three-dimensional probes: These will add ultrasound or another device to a fourth generation probe.

Q.14 Explain various types of probes.

(i) Marquis probe: Calibrations are in 3 mm sections.

(ii) UNC–15 probe: 15 mm long probe with mm markings at each mm and color at 5th, 10th and 15th mm.

(iii) University of Michigan 'O' probe with Williams marking at 1, 2, 3, 5, 7, 8, 9 and 10 mm.

(iv) Michigan 'O' probe with marking at 3, 6 and 8 mm

(v) WHO probe: 0.5 mm ball at the tip and mm markings at 3.5, 8.5 and 11.5 and color coding from 3.5-5.5.

(vi) Goldman fox probe: Flat, rectangular probe with markings at 1, 2, 3, 5, 7, 8, 9 and 10 mm.

(vii) Nabers probe: Curved probe used for furcation areas.

(viii) Moffitt / Maryland probe: WHO design with Williams marking.

(ix) NIDR probe: It is a color coded and is graduated in 2 mm increments at 2, 4, 6, 8, 10 and 12 mm with alternating increments colored in yellow.

(x) Florida probe: The criteria given by NIDR were met by Florida research group. Gibbs et al in the year 1988 developed the Florida Probe system. This incorporates constant probing force, precise electrical measurement, computer storage of data. The parts are Probe Hand piece, Digital readout, Switch, Computer Interface and Computer. Two models have been developed which differ in their fixed reference point –

(a) Stent model: The probe has 1 mm metal collar that rests on a prepared ledge on a prefabricated vacuoform stent.

(b) Disk model: Has a 11 mm disk which rests on the occlusal surface or incisal edge of the tooth.

Shortcomings are: Lack tactile sensitivity, forces the operator to predetermine the point of insertion, underestimation of deep probing depths, measurement taken with this system are less variable than with conventional probing.

(xi) Toronto automated probe: Developed by researchers at the University of Toronto. Like the Florida probe, it uses the occlusal – incisal surface to measure clinical attachment levels. The sulcus is probed with 0.5 mm nickel-titanium wire that is extended under

air pressure. It controls angular discrepancies by means of a mercury tilt sensor that limits angulation within ± 30º, but it requires reproducible positioning of the patient's head and can't easily measure third and second molars.

(xii) Interprobe: Developed by Goodson and Kondon in 1988. This has an optical encoder transduction element.

(xiii) Briek probe: Developed by Briek et al. in 1987. Works by constant air pressure and uses the occlusal surface as its reference point.

(xiv) The Jeffcoat probe (or Foster Muller Probe): Developed by Jeffcoat in 1986. It is capable of coupling pocket depth measurement with detection of the CEJ. The probe extends a thin metal fiber along the tooth surface into the sulcus and detects a slight acceleration rise when encountering the CEJ and then undergoes final extension, under constant force, on reaching the base of the pocket.

Q.15 What is Expros?

Expros are double end instrument with an explorer on one end and probe on other. e.g 17/Williams, 23/0 Michigan, 23/Williams.

Q.16 What are Novatech probes?

These are the probes with unique right angle design for improved adaptability in posterior.

Q.17 What is balanced instrument?

Balanced instrument: The working ends are centered on a line running through the long axis of the handle.

Q.18 What are the various design features of handle?

(i) Weight: Hollow handle increase tactile transfer and minimize fatigue.

(ii) Diameter: Small handle decreases control and increases muscle fatigue. Large handle maximize control and reduces muscle cramps but restrict movement in areas where access is limited (e.g. posterior areas).

(iii) Serration: Knurled handles maximize control and decreases hand fatigue. Smooth handles decrease control and increase muscle fatigue.

Q.19 What are the design features of Shank?

(i) Functional shank length: The functional shank length may be short, long or intermediate. The functional shank length extends from the working end to the shank bend closest to the instrument handle. Long functional shanks are needed to reach the tooth surfaces of posterior teeth or the root surfaces of teeth within periodontal pockets. Short functional shanks are found on instruments used to remove supramarginal calculus deposits or to reach the surfaces of anterior teeth.

(ii) Shank curvature: An instrument shank is curved if it has bends that deviate from the long axis of the shank. Instruments with curved shanks can usually be used on both posterior and anterior teeth and are referred to as universal instruments. Instruments with straight shanks are limited to use on anterior sextants and thus are referred to as anterior instruments.

(iii) Terminal shank: The shank nearest to the working end of the instrument.

(iv) Shank Flexibility: Instrument shank may be flexible, moderately flexible, or rigid in design. Shank flexibility is related to instrument use.

Shank Types	*Uses*	*Examples*
Rigid	• Removal of heavy calculus deposits. • Rigid shank limits tactile conduction so that calculus detection is difficult	• Sickle scalers • Periodontal files
Moderately flexible	• Removal of moderate or light calculus • Moderately flexible shank provides good level of tactile transfer, allowing detection and removal of moderate sub-marginal deposits	• Universal curettes
Flexible	• Detection of submarginal calculus • Removal of fine calculus • Flexible shank provides the best tactile information to the operator's finger pads through the shank and handle	• Gracey curettes • Explorers

Q.20 How are the working ends of Double ended instrument identified?

(i) If the design name and number are stamped along the length of the handle, each working end is identified by the number closest to it.

(ii) If the design name and number are stamped across the instrument handle, the first number (on the left) identifies the working end at the top and the second number identifies the working end at the bottom of the handle.

Q.21 Mention characteristic design feature of Explorer, Periodontal probe, Sickle Scaler, Curette, Periodontal file, Hoe and Chisel.

Explorer:

(i) Fine, wire-like working end

(ii) Sharp point

(iii) Circular in cross-section

Periodontal probe:
(i) Rod-shaped working ends
(ii) Smooth, rounded tip
(iii) Rounded or rectangular in cross-section
(iv) Some designs have millimeter markings

Sickle Scaler:
(i) Two cutting edges
(ii) Cutting edges meet in pointed tip
(iii) Pointed back
(iv) Triangular in cross-section

Curette:
(i) Two cutting edges
(ii) Spoon-shaped working end
(iii) Cutting edges meet in a rounded toe
(iv) Rounded back
(v) Semicircular in cross-section

Periodontal file:
(i) Many cutting edges
(ii) Cutting edges at 90°-105° angle to the shank
(iii) Have strong, rigid shank

Hoe:
(i) One straight cutting edge
(ii) Working end at 99°-100° angle to the shank
(iii) Have strong, rigid shank.

Chisel:
(i) One straight cutting edge
(ii) Heavy, straight shank.

Q.22 What are the differences between Area specific and Universal curettes?

		Area specific curettes	*Universal curettes*
1	Area of use	These are designed for specific areas and surfaces.	Designed for all areas and surfaces.
2	Cutting edge used	One cutting edge is used, i.e outer edge	Both cutting edges are used
3	Curvature	Curved in two planes, blade curves up and to the side	Curved in one plane
4	Blade angle	Offset blade: Face of blade beveled at 60° to shank	Not offset: Face of blade beveled at 90° to shank
5	Examples	• Gracey series • Kramer-Nevin series • Turgeon series • After five series • Mini five series • Curvette series	• Columbia 2R/2L • Columbia 4R/4L • Columbia13-14 • Barnhart1/2 • Barnhart 5/6 • Langer 1/2 • Langer 3/4 • Langer 5/6 • Langer 17/18

Q.23 Explain Langer curettes.

The design characteristics of Langer curettes differ from Universal curettes in following manner:

(i) More than one Langer curette is needed to instrument the entire dentition.

(ii) These curettes combine the shank design of standard Gracey 5-6, 11-12 and 13-14 curettes with Universal blade honed at 90^0, thus called as the marriage of Gracey and Universal curette design.

Instrument	*Area of use*
Langer 1/2	Mandibular posterior teeth
Langer 3/4	Maxillary posterior teeth
Langer 5/6	Mandibular and maxillary anterior teeth
Langer 17/18	Mandibular and maxillary second and third molars.

Q.24 Give examples of Area-specific curettes.

(i) Gracey curettes
(ii) Kramer - Nevins series
(iii) Turgeon series
(iv) Hu - Friedy After five series
(v) Hu - Friedy Mini five series
(vi) Hu - Friedy Curvette series
(vii) Furcation curettes.

Q.25 Explain Area-specific Gracey curettes.

Instrument	*Area of use*
Gracey 1-2 and 3-4	Anterior teeth
Gracey 5-6	Anterior teeth and premolars
Gracey 7-8 and 9-10	Posterior teeth; facial and lingual surfaces
Gracey 11-12	Posterior teeth; mesial surfaces
Gracey 13-14	Posterior teeth; distal surfaces
Gracey 15-16	Posterior teeth; mesial surfaces
Gracey 17-18	Posterior teeth; distal surfaces.

Q.26 What are the differences between Rigid and Finishing type of Gracey curettes?

	Rigid Gracey curettes	*Finishing Gracey curettes*
1	Larger shank and blade	Smaller
2	Stronger shank and blade	Less stronger
3	Less flexible shank and blade	More flexible
4	Used to remove moderate to heavy calculus without employing separate set of heavy scalers.	Require separate set of heavy scalers
5	Less tactile sensitivity	Enhanced tactile sensitivity

Q.27 Explain Curvette area specific curettes.

The design characteristics of Curvette curettes differ from those of standard Gracey curettes in following manner:

(i) 50% shorter working end
(ii) Increased curvature of the working end
(iii) Straighter shank on anterior instruments
(iv) Extended lower shank on posterior instrument

Instrument	***Area of use***
Curvette Sub-zero	Anterior teeth and premolars (facial and lingual surfaces)
Curvette 1/2	Anterior teeth and premolars (inter-proximal surfaces)
Curvette 11/12	Mesial surface of molars
Curvette 13/14	Distal surface of molars.

B. INSTRUMENTATION

Q.1 What are the general principles of Instrumentation?

A. Accessibilty
   (a) Position of the patient
   (b) Position of the operator
B. Visibility, Illumination and Retraction
C. Condition of the instruments
   (a) Sharpness
   (b) Sterilization
D. Maintaining a clean field
E. Instrument stabilization
   (a) Instrument grasp
   (b) Finger rest
F. Instrument activation
   (a) Adaptation
   (b) Angulation
G. Instruments for scaling and root planing
   (a) Scalers
   (b) Universal curettes
   (c) Gracey curettes.

Q.2 Write the basic positioning of operator on the stool and patient on the chair during dental treatment.

A. Operator on the stool:

(i) Head is relatively erect and is in the least strained position vertically and horizontally.

(ii) Eyes are directed downward in a manner that prevents neck and eye strain.
(iii) Distance from the patient's mouth to the eyes of the clinician should be 14-16 inches.
(iv) Shoulders are relaxed.
(v) Forearm and wrist are kept in a straight line, wrist is neither flexed nor extended.
(vi) Body weight is completely supported by the chair.
(vii) Back is straight and erect.
(viii) Thighs parallel with the floor.
(ix) Feet are flat on the floor.

B. Patient on the chair:

(i) Upright: Initial position from which chair adjustments are made.
(ii) Semi-upright: Respiratory and Cardiovascular patient should be in the semi-upright position during treatment.
(iii) Supine: Flat position with head and feet on the same level.
(iv) Trendelenburg: Modified supine position when the head is lower than the heart. The brain is lower than heart and feet are slightly elevated

Q.3 What should be the distance from the patient's mouth to the eyes of the clinician during dental treatment?

14-16 inches

Q.4 What should be the assistant level in relation to the clinician?

Assistant is seated with eye level 4-6 inches above the clinician's eye level and facing towards the head of dental chair.

Q.5 What is Critical, Semicritical and Non-critical objects ?

(i) Critical objects: Are those which penetrate soft tissue or bone, e.g blades, needles, curettes, explorers, and probes. They are sterilized or disposable.
(ii) Semicritical objects: Are those which touch intact mucosa, membranes, oral fluids and does not penetrate them, e.g mirror, ultrasonic handpiece, radiographic biteblock. They are sterilized or high level disinfected.
(iii) Non-critical objects: are those which do not touch mucous membrane, e.g light handles, safety eyewear, X-ray machine parts. They are disinfected.

Q.6 Why are the instruments sharpened?

(i) To produce a functionally sharp edge.
(ii) To preserve the shape, contour of the instrument.
(iii) To maintain the useful life of the instrument.

Q.7 What are the advantages of sharp instruments?

(i) Improved tactile sensations
(ii) Increased efficiency of deposit removal

(iii) Less pressure required for deposit removal
(iv) Improved instrument control
(v) Minimized patient discomfort
(vi) Decreased burnished calculus.

Q.8 What are the various undesirable edges formed on the instrument during sharpening?

(i) Wire edge: These are undesirable unsupported metal fragments extending beyond the cutting edge from the lateral side or face of the blade. If a coarse stone is used and instrument is oversharpened, there may be tendency to produce what has been called as wire edge of saw tooth like projections of metal.
(ii) Round edge: Undesirable edges that began as sharp edges but are dulled through use / overuse of the instrument.
(iii) Beveled edges: These are cutting edges created beneath the original cutting edge by improper stone to instrument placement.

Q.9 What are the various Instrument Grasps?

(i) Standard pen grasp
(ii) Modified pen grasp
(iii) Palm and thumb grasp.

Q.10 Which is the most effective and stable grasp for periodontal instruments?

Modified pen grasp

Q.11 Explain modified pen grasp.

Thumb, index finger and middle finger are used to hold the instrument. The pad of the middle finger rests on the shank. The index finger is bent at the second joint from the finger tip and is positioned well above the middle finger on the same side of the handle. The pad of the thumb is placed midway between the middle and index fingers on the opposite side of the handle.

Q.12 What is the major difference between standard pen grasp and modified pen grasp?

In standard pen grasp, the side of the pad of middle finger rests on the shank while in modified pen grasps, the pad of middle finger rests on the shank.

Q.13 Where are palm and thumb grasp used?

(i) For stabilizing instruments during sharpening.
(ii) For manipulating air and water syringes.
(iii) For manipulating porte polisher

Q.14 What are the various types of finger rests?

A. Intraoral finger rests:
(a) Conventional: Finger rests on the immediately adjacent tooth.

(b) Cross arch: Finger rests is placed on tooth surface on the other side of same arch.
(c) Opposite arch: Finger rests is placed on tooth surface on the opposite arch.
(d) Finger on finger: Finger rests is placed on the thumb or index finger of the non-operating hand.

B. Extraoral fulcrums:
(a) Palm-up: The back surfaces of the middle and fourth finger is placed on the skin overlying the lateral aspect of the mandible on the right side of the face.
(b) Palm-down: The front surfaces of middle and fourth fingers is placed on the skin overlying the lateral aspect of the mandible on the left side of the face.

Q.15 What is adaptation and angulation?

(i) Adaptation: It refers to the manner in which the working end of a periodontal instrument is placed against the surface of a tooth. The objective of which is to make working end of instrument conform to the contour of the tooth surface.
(ii) Angulation: It refers to the angle between the face of a bladed instrument and the tooth surface. It is also called as tooth-blade relationship.

Q.16 What is instrumentation zone?

Scaling and root planing strokes is confined to the portion of the tooth where calculus or altered cementum is found, this zone is called as instrumentation zone.

Q.17 What are the various instrumentation strokes and their indication?

A. Exploratory stroke:
(a) Vertical direction
(b) Horizontal direction
(c) Oblique direction
- These are light feeling strokes.
- Used to evaluate dimensions of the pocket, to detect calculus and tooth surface irregularities

B. Scaling stroke:
(a) Vertical direction
(b) Horizontal direction
(c) Oblique direction
- These are short, powerful pull strokes.
- Used to remove supragingival and subgingival calculus.

C. Root planing stroke:
(a) Vertical direction
(b) Horizontal direction

(c) Oblique direction
- These are long, moderate to light pull strokes.
- Used for final smoothing and planing of the root surfaces with the help of curettes, hoes, files and ultrasonic instruments.

Q.18 What is the optimal angulation for subgingival insertion of instrument, scaling and root planing and gingival curettage?

Subgingival insertion of instrument – 0°
Scaling and root planing – 45°-90°
Gingival curettage – more than 90°.

Q.19 What is oral prophylaxis?

It is one of the essential element of preventive program. A thorough prophylaxis includes:

(i) Removal of all supragingival and subgingival calculus
(ii) Root planing
(iii) Removal of all extrinsic stains and bacterial plaque
(iv) Correction of overhanging restoration and improperly contoured restoration
(v) Polishing of all tooth and restorative surfaces
(vi) Application of caries preventive agents
(vii) Home care instructions.

Q.20 What is scaling and root planing?

Scaling – is the process by which plaque and calculus is removed from both supragingival and subgingival tooth surfaces.
Root planing – is the process by which residual calculus and portions of cementum are removed from the roots to produce smooth, hard and clean surface.

Q.21 What is the objective of scaling and root planing?

The primary objective of scaling and root planing is:-

(i) To restore gingival health by completely removing plaque, calculus and endotoxin from the tooth surface that provoke gingival inflammation.
(ii) Suppression / elimination of the pathogenic periodontal microflora.
(iii) Conversion of inflamed, bleeding / suppurative pathogenic pockets to healthy gingival tissue.
(iv) Shrinkage of the deepened pathologic pocket to a shallow, healthy gingival sulcus.
(v) Providing a root surface compatible with reestablishment of a healthy connective tissue and epithelial attachment.

Q.22 What is the difference between Scaling and Root planing?

	Scaling	*Root planing*
1	Remove sources of irritation to the periodontium: • Calculus deposit that harbor dental plaque • Calculus deposit that act as a mechanical irritant	Remove sources of irritation to the periodontium: • Residual calculus embedded in the cementum • Endotoxins and other bacterial toxins contained in the outermost layers of cementum • Hypermineralized cementum • Necrotic grainy cementum that makes plaque control more difficult
2	Promote a shift from high levels of disease-related organisms to a greater percentage of health-related organisms in sulci and shallow pockets	Promote a shift from high levels of disease-related organisms to a greater percentage of health-related organisms in pockets
3	Establish an environment that is conducive to the health of the gingival tissues	Allow for re-evaluation of tissues to determine if surgical procedures are necessary
4	Create an environment that will facilitate the patient's home care efforts	Create a hard, glass-like root surface that will facilitate the patient's home care effort

Q.23 What are the contraindications of polishing?

(i) Patients who have communicable disease that could be spread by aerosols.
(ii) Patients who are susceptible for bacteremia.
(iii) Tooth surfaces with extrinsic stain where stain is incorporated into plaque or calculus.
(iv) Areas of thin or deficient enamel; cementum or dentin surfaces; areas of hypersensitivity.
(v) Caries susceptible teeth; areas of white spot demineralization; thin or deficient enamel.
(vi) Gold restorations can be easily scratched by abrasive agents.
(vii) A restricted sodium diet, including patient with controlled hypertension.
(viii) Exposed cementum or dentin.
(ix) Inflammed gingival tissues.
(x) Composite restorations.

Sonic and Ultrasonic Instrumentation

Q.1 What are the advantages and disadvantages of ultrasonic instrumentation ?

Advantages:

(i) Rapidly removes heavy calculus and stains
(ii) Less clinician fatigue
(iii) Increased patient discomfort and acceptance
(iv) Minimal soft tissue trauma
(v) Fragments of calculus are flushed out with the water
(vi) Requires less time than hand scaling

Disadvantages:

(i) Decreased tactile sensitivity
(ii) Reduced visibility due to water spray
(iii) If tip is not used correctly there is a chance of causing surface irregularties
(iv) Messy to operate because of water spray
(v) Cannot accomplish definitive calculus removal
(vi) Excessive heat build up can lead to pulp damage and sensitivity
(vii) Requires high speed evacuation
(viii) Produces contaminated aerosol.

Q.2 What are the indications and contraindications of using ultrasonic instruments?

Indications:

(i) Heavy tenacious calculus and stain
(ii) Overhanging margins of amalgam restorations
(iii) Orthodontic cement removal

Contraindications:

(i) Patients with contagious diseases
(ii) Patients with a pacemaker especially to magnetostrictive
(iii) Composite resin restorations
(iv) Porcelain inlays or crown
(v) Patients with deep, pus producing pockets
(vi) Desquamative gingivitis

(vii) Local osteomyelitis
(viii) Local metastatic neoplasms.

Q.3 Compare Ultrasonic and Hand instrumentation.

	Ultrasonic instrument	*Hand instrument*
1	Mechanism of action: Vibration, acoustic streaming and cavitation	Mechanical removal of deposit
2	Used on heavy tenacious deposits and stains	Used on all amounts of deposits
3	Instrument tip is dull and bulky	Sharp and thin
4	Less tactile sensitivity	Good tactile sensitivity
5	Digital motion activation is used with light pressure	Hand motion activation is used with firm pressure
6	Inaccessible to some areas because of tip design	Greater accessibility
7	Less time required	More time required
8	Less clinician fatigue	More clinician fatigue
9	Water spray cause patient discomfort	No water spray, no discomfort
10	Possibility of damage to tooth from heat build up	No heat build-up
11	Aerosols are produced	No aerosols are produced
12	Contraindicated in patients with pacemaker and having contagious diseases	No such contraindications
13	Sharpening not needed frequently	Frequently required
14	Smaller tip size (0.3 - 0.55 mm)	Larger tip size (0.76 - 1 mm)

Q.4 What is white finger?
Large amplitudes produced by pneumatic drills will cause white finger. It is because of the disruption in blood flow to the fingers caused by the vibration that is passed from the drill to the hand.

Q.5 What is the difference between splatter and aerosol?
A particle of true aerosol is less than 50 μm in diameter while splatter particles are greater than 50 μm.

Q.6 What are the methods to reduce the hazards of aerosols produced by ultrasonic instrumentation?
(i) Pre-procedural antiseptic mouthrinse
(ii) Use of high volume evacuators
(iii) High risk infective patients should be treated with hand instruments.

Q.7 For how much minimum time period the aerosol which are produced by ultrasonic scaling remains in the air.
30 minutes.

Q.8 What is Acoustic streaming and Cavitation?

Acoustic streaming: The pressure produced by the continuous stream of fluid flowing into the confined space of the periodontal pocket is known as acoustic streaming or turbulence. Bacteria and gram negative motile rods, in particular, are sensitive to acoustic energy.

Cavitation: The vibratory motion of the tip and the continuous stream of water cause tremendous pressure, creating powerful bursts of collapsing bubbles. This is referred to as cavitation. It is a combination of the vibrating instrument's tip against the deposit, high frequency sound waves, and exploding bubbles that allow for calculus removal.

Q.9 What is the principle of Magneto and Piezo ultrasonic instruments?

Magneto: The instrument is comprised of an electronic generator, a handpiece assembly containing a coil to energize the insert, and a variety of interchangeable inserts. The generator produces an alternating low voltage electric current in the handpiece. This current produces a magnetic field in the handpiece that causes the insert to expand and contract along its length and inturn, causes the insert tip to vibrate.

Piezo: This system is comprised of an electronic generator, a handpiece assembly containing piezo (ceramic) crystals to energize a scaling tip, and a variety of interchangeable screw-on tips. The generator produces an alternating, high voltage in the handpiece. This voltage produces an electric field in the handpiece that causes the piezo crystals to expand and contract along their diameter and inturn, causes the scaling tip to vibrate.

24 Periodontal Surgery

A. GENERAL PRINCIPLES OF PERIODONTAL SURGERY

Q.1 What are the objectives of surgical phase of periodontal surgery?

(i) Improvement of the prognosis of teeth and their replacements
(ii) Improvement of esthetics

Q.2 Classify periodontal surgery.

A. Pocket reduction surgery:
   (i) Resective - Gingivectomy, Apically displaced flap and Undisplaced flap with or without osseous resection.
   (ii) Regenerative - Flaps with grafts and membranes
B. Correction of Anatomic / Morphologic defects:
   (i) Plastic surgery techniques to widen attached gingiva-
      • Epithelial grafts
      • Connective tissue grafts
   (ii) Esthetic surgery -
      • Root coverage
      • Re-creation of gingival papillae
   (iii) Preprosthetic surgery techniques -
      • Crown lengthening
      • Ridge augmentation
      • Vestibular deepening
   (iv) Placement of dental implants -
      • With GBR
      • Sinus grafts.

Q.3 What are the critical zones in pocket surgery?

- Zone 1- Soft tissue wall
- Zone 2- Tooth surface
- Zone 3- Bone
- Zone 4- Attached gingiva.

Q.4 What are the indications for periodontal surgery ?

(i) Persistent inflammation in areas with moderate to deep pockets.
(ii) Areas with irregular bony contours and deep craters.

(iii) When removal of root irritant is not possible due to deep pockets especially in molars and premolars.
(iv) In case of furcation involvement.
(v) Infrabony pockets on the distal areas of last molars, complicated by mucogingival problems.

Q.5 What are the possible outcomes of periodontal therapy?

(i) New attachment
(ii) Long junctional epithelium
(iii) Root resorption / ankylosis
(iv) Recurrence of pocket.

Q.6 Name various chemotherapeutic agents used for premedications.

(i) Anxiolytics
(ii) Antibiotics
(iii) Antiseptics
(iv) NSAIDS.

Q.7 What are the causes of excessive bleeding during surgery?

(i) Laceration of large blood vessels
(ii) Incomplete removal of granulation tissue
(iii) Hypertensive patient
(iv) Patient with bleeding disorder
(v) Patient on anticoagulant therapy

Q.8 Name various topical hemostatic agents.

	Agent	***Main constituent***
(i)	Avitene	Collagen
(ii)	Collacote	Microfibrillar
(iii)	Collacope	Collagen
(iv)	Collaplug	Collagen
(v)	Thrombinar	Thrombin
(vi)	Thrombogen	Thrombin
(vii)	Thrombostat	Thrombin
(viii)	Gelfoam	Gelatin
(ix)	Beriplast	Fibrin
(x)	Surgicel	Cellulose
(xi)	Cyclo kapron	Tranexamic acid.

Q.9 Who introduced periodontal dressings?

Dr. AW Ward in 1923, introduced periodontal dressings.

Q.10 What purpose do the periodontal dressings serve?

(i) Protection of wound area from irritants
(ii) Enhancement of patient comfort
(iii) Maintenance of a debris free area

(iv) Repositions soft tissues, protect sutures
(v) Protection of newly exposed root surface from temperature changes.

Q.11 Classify periodontal dressing.

(i) Zinc oxide eugenol
(ii) Zinc oxide non-eugenol:
  (a) Coepak
  (b) Periocare
  (c) Periopac
  (d) Perioputty
  (e) Vocopac
(iii) Others:
  (a) Photocuring periodontal dressing: Barricaid
  (b) Collagen dressings
  (c) Methacrylic gel
  (d) Cyanoacrylate.

Q.12 How is periodontal dressing retained in the particular areas of oral cavity?

(i) In case of edentulous areas—With the help of splints, hawley appliance and stents.
(ii) In case of isolated teeth—Tie dental floss or gauze loosely around the teeth and over which pack is applied.
(iii) In case of dentulous areas—Mechanically by interlocking in interdental spaces and joining the lingual and facial portions of the pack.

Q.13 What are the parts of surgical needle?

(i) Point – It is the working end of needle.
(ii) Body – It refers to the grasping area which forms the majority of length of needle. It starts where the point of needle ends and ends at the contour change, that marks the beginning of swage of the needle.
(iii) Eye / Swage – It is the segment at which needle and suture materials are joined.

Q.14 What are the types of needles?

A. On the basis of shape–
  (i) Straight
  (ii) Curved:
    (a) 1 / 4
    (b) 3 / 8
    (c) 1 / 2
    (d) 5 / 8

B. On the basis of eye -
   (i) Eyed: It is designed to reuse. In this, suture material is tied to the needle.
   (ii) Eyeless / Swaged: The suture material is inserted into hollow end during manufacturing and metal is compressed around it. Needle is not reusable.

C. On the basis of function -
   (i) Tapered: Used for closing mesenchymal layers such as muscle / fascia that are soft and easily penetrable
   (ii) Cutting: Used for keratinized mucosa and skin.
      (a) Conventional cutting
      (b) Reverse cutting.

Q.15 What are the various techniques of wound closure?

(i) Sutures
(ii) Skin clips / staples
(iii) Skin tapes
(iv) Wound adhesive-
   (a) Autologous fibrin glue
   (b) Fibrin fibronectin sealing system (Tissucol)
   (c) Cyanoacrylate
   (d) Mussel adhesive protein

Q.16 What are the various intraoral anchoring structures for use in securing movable tissues?

(i) Teeth: Easiest and most secure of all intraoral anchors
(ii) Bound down tissue: Gingiva affixed to bone via periosteum, is the second most reliable anchor
(iii) Periosteum
(iv) Loose connective tissue: Least secure anchoring point in the mouth, e.g. Connective tissue in the vestibule and fatty tissue in the retromolar area.

Q.17 What are the objectives of suturing?

(i) To stabilize the tissue
(ii) To secure the tissue in the desired location
(iii) To maintain hemostasis
(iv) To permit healing by primary intention
(v) As a tool to retract flap for photography or to retrieve free gingiva / connective tissue autografts.

Q.18 Classify suture materials.

A. Based on the number of filaments:
   (a) Monofilament, e.g steel, nylon
   (b) Multifilament, e.g silk, cotton

B. Based on suture diameter by US Pharmacopeia in descending order from 5, 4, 3, 2, 1, 0, 1-0, till 11-0 size. 5 being the largest diameter and 11-0 the smallest.

C. Based on resorbability of suture material:
   (a) Absorbable
   (b) Non-absorbable

D. Based on the source:
   (a) Natural–
      (I) Absorbable:
         (i) Plain gut
         (ii) Chromic gut
         (iii) Fast absorbing gut
         (iv) Plain collagen
         (v) Chromic collagen
      (II) Non-absorbable:
         (i) Silk
         (ii) Cotton
         (iii) Linen
   (b) Synthetic –
      (I) Absorbable
         (i) Polyglactin
         (ii) Polyglyconate
         (iii) Polyglycolic
         (iv) Polydioxanone
      (II) Non-absorbable
         (i) Nylon
         (ii) Polyester
         (iii) Decron
         (iv) Polypropylene
         (v) Nurolone.

Q.19 What is the method of prescription of suture?

It should contain the name of the suture, its size, length and atraumatic, the type of needle, size of the needle and number of foils required, e.g. Prolene 2-0, 70 cms with atraumatic reverse cutting needle (3/8 circle 45 mm) – 1 foil.

Q.20 Write principles of suturing.

(i) Needle holder should grasp the needle approximately ¾ th of the distance from point.
(ii) Needle should enter the tissue perpendicular to the surface.
(iii) Needle should be passed through the tissue following curvature of the needle.

(iv) Suture should be placed at an equal distance (2-3 mm) from incision on both sides and at an equal depth.
(v) Needle should be passed from free to fixed side.
(vi) Needle should be passed from thinner to the thicker side.
(vii) If one tissue plane is deeper than the other, needle should be passed from deeper to superficial side.
(viii) The distance that the needle is passed into tissue should be greater than the distance from the tissue edge.
(ix) The tissue should not be closed under tension, it will either tear or necrose.
(x) Suture should be tied so that tissue is merely approximated, not blanched.
(xi) Suture knot should not be placed over the incision line.
(xii) Suture should be placed approx. 3-4 mm apart.

Q.21 What are the various types of sutures?

A. Interrupted sutures:
   (a) Circumferential: Direct / loop
   (b) Figure of eight
   (c) Mattress – Vertical and horizontal
   (d) Interstitial papillary placement
B. Continuous suture:
   (a) Independent sling suture
   (b) Mattress – Vertical and Horizontal
   (c) Continuous locking
C. Simple sling suture
D. Periosteal sutures.

Q.22 What is Mattress sutures?

A mattress means the suture passes through the flap twice. The material does not pass under the incision line, thus minimizing wicking.

(a) Vertical mattress
   (i) Everting
   (ii) Inverting
(b) Horizontal mattress.

Q.23 What are the indications of each suturing technique?

(i) Simple sling suture: It is used primarily in apically positioned flap and in repositioning the flap.
(ii) Everting vertical mattress: It is useful in papilla preservation technique in anterior arch.
(iii) Periosteal sutures: Used in apically positioned partial thickness flap.

(iv) Direct / loop sutures:
  (a) Performed when bone grafts are used.
  (b) When closed apposition of scalloped incision is required.
(v) Figure of eight: When flaps are not in close apposition because of apical flap position or nonscalloped incisions.
(vi) Horizontal mattress suture: Interproximal areas of diastema with short and wide papillae.
(vii) Vertical mattress suture: In areas with long and narrow papillae.
(viii) Anchor suture: Used in closing of the flap in the mesial / distal wedge procedures.
(ix) Closed anchor suture: Used to close a flap located in the edentulous area mesial / distal to a tooth.
(x) Circumferential suturing: Indicated for suturing grafts.

Q.24 What are the components of sutured knots?

(i) Loop- Loop created by the knot.
(ii) Knot- Knot, which is composed of a number of tight throws, each throw represents a weave of the two strands.
(iii) Ears- Ears are the cut ends of the suture.

Q.25 What are the various types of knots?

(i) Square knot: Two single tie in opposite direction.
(ii) Granny knot: Two or three tie in same direction.
(iii) Surgeon's knot 2-1: 1st tie is double and 2nd tie is single in opposite direction.
(iv) Surgeon's knot 2-2: 1st tie is double and 2nd tie is also double in opposite direction.

Q.26 What are the principles of suture removal?

(i) Areas should be swabbed with hydrogen peroxide for removal of encrusted necrotic debris, blood and serum from suture.
(ii) A sharp suture scissor should be used to cut the loops of suture, close to the epithelial surface as possible. In this way a minimal amount of portion of sutures that was exposed to the outside environment and has become laden with debris and bacteria will be dragged through the tissue.
(iii) A cotton pliers is then used to remove the sutures. The location of knots should be noted so that they can be removed first, which will prevent unnecessary entrapment of the flap.

Q.27 What is healing?

Healing is a phase of the inflammatory response that leads to a new physiological and anatomical relationship among the disrupted body elements.

Q.28 What are the healing rates of various periodontal tissues?

	Tissue type	Healing rate (approx)
(i)	Junctional epithelium	5 days
(ii)	Sulcular epithelium	7 – 10 days
(iii)	Gingival surface epithelium	10 – 14 days
(iv)	Connective tissue	21 – 28 days
(v)	Alveolar bone	4 – 6 weeks

Q.29 What are the instructions given to the patient after periodontal surgery?

Instructions to the patient after surgery:

Do's:

(i) Take 2 tablets of Acetaminophen every 6 hrs on first day.
(ii) Chew on non operated side.
(iii) Take semisolid food.
(iv) Apply ice, intermittently on the face over the operated side on the first day.
(v) Use chlorhexidine mouthwash.
(vi) If the bleeding does not stop, take piece of gauge and form it into U-shape and hold it in thumb and index finger, apply it to both sides of the pack, and hold it there under pressure for 20 min.
(vii) Swelling is usual in extensive surgical procedure. It subsides in 3 or 4 days. Apply moist heat if it persists.
(viii) If any other problem arise do call the doctor.

Do not's:

(i) Avoid hot food.
(ii) Do not smoke or take alcohol.
(iii) Avoid citrus, highly spicy food.
(iv) Do not brush over the pack.
(v) Avoid exertion.
(vi) Do not try to stop bleeding by rinsing.

B. GINGIVECTOMY

Q.1 What is gingivectomy?

It is excision of the soft tissue wall of the pocket.

Q.2 What are the objectives of gingivectomy?

(i) Pocket elimination by gingival resection
(ii) Development of physiologic tissue form for disease prevention.

Q.3 What is gingivoplasty and its objective?

It is recontouring of gingiva that has lost its physiologic form.
Objective: Is to create the physiologic gingival form.

Q.4 How is gingivoplasty different from gingivectomy?

The purpose of gingivoplasty is different from gingivectomy, as gingivoplasty is just reshaping of gingiva to create physiologic gingival contours, with the sole purpose of recontouring the gingiva in the absence of pockets, while the objective of gingivectomy is to eliminate pocket.

Q.5 What are the indications and contraindications of gingivectomy?

Indications:

(i) Elimination of suprabony pockets
(ii) Elimination of gingival enlargement
(iii) Elimination of suprabony periodontal abscesses
(iv) To expose additional clinical crown to gain added retention for restorative purposes and to provide access to subgingival caries
(v) The presence of furcation involvement (without associated bone defects) where there is a wide zone of attached gingiva
(vi) Pericoronal flap

Contraindications:

(i) The need for bone surgery or examination of the bone shape and morphology.
(ii) Situations in which the bottom of the pocket is apical to the mucogingival junction.
(iii) Esthetic considerations, particularly in anterior maxilla.

Q.6 What are the indications of gingivoplasty?

(i) Need for correction of the grossly thickened gingival margin.
(ii) Gingival clefts and craters caused by necrotizing ulcerative gingivitis that interfere with normal food excursion, collect plaque and food debris.
(iii) Sharply varying levels of gingival margin in adjacent areas.
(iv) Saucer shaped deformities buccolingual in the interproximal regions.

Q.7 What are the steps in the gingivoplasty procedure?

(i) Tapering the gingival margin.
(ii) Creating an scalloped marginal outline.
(iii) Thinning the attached gingiva.
(iv) Creating vertical interdental grooves and shaping the interdental papillae to provide sluiceways for the passage of food.

Q.8 What are the steps in the gingivectomy procedure?

(i) Mark bleeding points with the help of pocket marker, pinpoint perforations that individuate pocket depth.
(ii) Incision: Discontinuous / continuous incision is given apical to the bottom of the bleeding point indicated by pinpoint markings.

(iii) Beveled incision at an angle of 45° apical to the base of the pocket with the help of Kirkland, Orban's knives, or blade no.12 or 15 with BP handle or angulated Blake's handle.
(iv) Tissue removal: Pocket wall is removed easily with the help of curette or scaler.
(v) Scaling and root planing is done with the help of scaler and curette.
(vi) Periodontal dressing is applied over the treated site.

Q.9 Name instruments used in gingivectomy.

(i) Pocket markers: Specially formed college pliers for pocket marking. Goldman – fox, Crane Kaplan
(ii) Broad-bladed, round scalpels: Goldman – fox no. 7, Kirkland
(iii) Narrow bladed, tapered (Spear shaped), Interproximal: Orban's knife, Goldman – fox no. 8, 9 and 10
(iv) Surgical handle with detachable and replacable – surgical blades: Bard Parker no.15, angulated handle – Blake's handle
(v) Curettes
(vi) Tissue nipper.

Q.10 What are the disadvantages of gingivectomy by chemosurgery?

(i) The depth of chemical action cannot be controlled.
(ii) Gingival remodeling cannot be accomplished effectively.
(iii) Epithelization and reformation of the junctional epithelium, re-establishment of the alveolar crest fiber system are slower in chemically treated gingival wounds than in those produced by scalpel.

Q.11 What are the advantages and disadvantages of LASER gingivectomy?

Advantages:

(i) LASER offers an almost completely dry, bloodless surgery.
(ii) Because of dry field, surgical time may be reduced.
(iii) There is an instant sterilization of the area, decreasing the chances of bacteremia.
(iv) This is a non contact surgery, thus no mechanical trauma to the surgical site.
(v) There is prompt healing with minimal postoperative swelling and scarring.
(vi) Postoperative pain appears to be greatly reduced.

Disadvantages:

(i) There is loss of tactile feedback in using the instrument.
(ii) It is imperative that all operating room personnel wear safety glasses for protection of their eyes.
(iii) There is the necessity for hospitalization.
(iv) High cost of the equipment.

Q.12 What are the advantages and disadvantages of gingivectomy by electrosurgery?

Advantages:

(i) It provides clear operating area with little / no bleeding.
(ii) Lack of pressure to incise the tissue, thus allowing a more precise incision than is obtained by a scalpel.
(iii) Minor tissue loss after healing.
(iv) Self-sterilization of the tip of the active electrode.
(v) Scar-free healing by primary intention, when used properly.
(vi) Greater ease for the patient as well as for the operator.

Disadvantages:

(i) It cause an unpleasant odor.
(ii) If the electrosurgery point touches the bone, irreparable damage can occur.
(iii) When electrode touches the root, areas of cementum burns are produced.

C. PERIODONTAL FLAP

Q.1 Describe various incisions used in periodontal surgery.

A. Horizontal incisions:
   (a) Internal bevel incision – First / basic incision. It starts from a designated area on the gingiva and is directed to an area at or near the crest of the alveolar bone. Given with the help of 11 or 15 no. surgical blade.
   (b) Crevicular / sulcular incision – Second incision. It is made from the base of the pocket to the crest of the alveolar bone. Given with the help of 12 no. surgical blade.
   (c) Interdental incision – Third incision. Given by Orban's knife.

B. Vertical incision / oblique releasing incision:
   (a) It must extend beyond the mucogingival line.
   (b) It should be made at the line angles of a tooth either to include the papilla in the flap or to avoid it completely.
   (c) Vertical incision in lingual and palatal areas is avoided.
   (d) Vertical incision should be designed so as to avoid short flap mesiodistally with long apically directed horizontal incision because this could jeopardize the blood supply to the flap.
   (e) Given with the help of 11 or 15 no. surgical blade.

C. Thinning incision:
   (a) Extends from gingiva towards the base of the flap in palatal flap and distal wedge procedures
   (b) Given with the help of 11 or 15 surgical blade.

D. Cut-back incision:
   (a) Made at apical aspect of releasing incision and directed towards base of the flap in laterally positioned flap.
   (b) Given with the help of 11 or 15 surgical blade.

E. Periosteal releasing incision:
   (a) Made at the base of flap severing the underlying periosteum.
   (b) Given with the help of 15 or 15C surgical blade.

Q.2 Which incision is called as Reverse bevel incision?

Internal bevel incision because its bevel is in reverse direction from that of the gingivectomy incision.

Q.3 What is periodontal flap?

It is the portion of gingiva and or alveolar mucosa surgically separated from the underlying tissues to provide visibility and access to the bone and root surface.

Q.4 What are the objectives of periodontal flap procedures?

(i) Provides access for root surface detoxification.
(ii) Reducing probing depths including those that extend to or beyond the mucogingival junction.
(iii) Preserves / create an adequate zone of attached gingiva.
(iv) Permit access to underlying bone for treatment of osseous defects.
(v) Facilitate regenerative procedures.

Q.5 Classify periodontal flaps.

A. According to flap reflection or tissue content:
   (a) Full thickness flap-consist of the complete mucoperiosteum and is raised by a periosteal elevator.
   (b) Split-thickness flap-gingiva is dissected from the underlying periosteum which is left on the bone.

B. According to flap placement after surgery:
   (a) Nondisplaced flap
   (b) Displaced flap:
      (i) Apical displaced flap
      (ii) Coronal displaced flap
      (iii) Lateral displaced flap

C. According to management of papilla:
   a. Conventional flap
   b. Papilla preservation flap.

Q.6 What is an envelope flap?

The flap without vertical incision is called envelope flap.

Q.7 What is Papilla preservation flap?

It is the procedure which incorporates the entire papilla in one of the flaps by means of crevicular interdental incisions to severe the

connective tissue attachment and a horizontal incision at the base of the papilla, leaving it connected to one of the flaps.

Q.8 What are the indications and contraindications of Papilla preservation flap technique?

Indications:

(i) Diastema region
(ii) Bone grafting areas

Contraindication:

(i) Narrow embrassures

Q.9 What are the advantages and disadvantages of Papilla preservation flap technique?

Advantages:

(i) Esthetically pleasing
(ii) Primary coverage of implant
(iii) Prevents postoperative tissue craters

Disadvantages:

(i) Technically difficult
(ii) Time consuming.

Q.10 Compare full thickness and partial thickness flaps.

		Full thickness flap	Partial thickness flap
1	Healing	Primary intention	Secondary intention
2	Degree of difficulty	Moderate	High
3	Osseous surgery	Yes	No
4	Periosteal retention	No	Yes
5	Widen zone of keratinized gingiva	No	Yes
6	Bleeding and tissue trauma	Limited	Greater
7	Variability of suture	Low	High

Q.11 Why the surgical approach for palatal flap is different from other flaps?

The surgical approach for palatal flap is different from other flaps because of the nature of palatal tissue which is attached and keratinized with no elastic properties.

Q.12 Difference between palatal incision, palatal flap and other flaps and incisions.

Palatal incision:

(i) If the purpose of surgery is debridement then internal bevel incision is given such that flap is adapted at root-bone junction, when sutured.

(ii) If osseous resection is to be done, then incision is planned to compensate for lowered level of bone when the flap is closed.

(iii) The apical portion of the scalloping should be narrower than the line angle area because of the taper of palatal root apically.

Palatal flap:

(i) Cannot be displaced apically.

(ii) Split – thickness flap could not be accomplished.

Q.13 What are the indications and contraindications of palatal flap?

Indications:

(i) Areas that require osseous surgery

(ii) Pocket elimination

(iii) Reduction of enlarged and bulbous tissue

Contraindication:

(i) When a broad, shallow palate does not permit a partial thickness flap to be raised without possible damage to the palate.

Q.14 What are the advantages and disadvantages of palatal approach procedures?

Advantages:

(i) Esthetics

(ii) Easier access for osseous surgery

(iii) Wider palatal embrassure space

(iv) A naturally cleansing area

(v) Less resorption because of thicker bone

Disadvantage:

(i) Close root proximity**.**

Q.15 Difference between Original and Modified Widman flap surgery.

		Original Widman flap	*Modified Widman flap*
1	Given by	Leonard Widman, 1918	Ramjford and Nissle,1974
2	Purpose	For pocket elimination	Provide access for adequate instrumentation
3	Collar of tissue attached to the teeth torn	With Curettes	With Orban's knife
4	Releasing incision	Given	Not given
5	Flap reflection	High flap reflection	Minimal flap reflection
6	Bone contouring	Done	No bone contouring
7	After suturing	Flaps do not cover interproximal bone, remains exposed	Flaps cover interproximal bone

Q.16 What are the advantages of Modified Widman flap over Original Widman flap procedure?

(i) Close adaptation of soft tissue to root surface.
(ii) Minimum trauma to alveolar bone.
(iii) Provide access to adequate instrumentation of the root surface

Q.17 What is the main purpose of Modified Widman flap, Undisplaced and Apically displaced flap?

A. Modified Widman flap:
   (i) To facilitate instrumentation on root surfaces by exposing them.
   (ii) To remove the pocket lining.
B. Undisplaced flap:
   (i) To reduce or eliminate pocket by removing pocket wall.
   (ii) To improve accessibility for instrumentation.
C. Apically displaced flap:
   (i) To eliminate pocket by apically positioning the soft tissue wall of the pocket.
   (ii) To preserve / increase the width of attached gingiva.
   (iii) To improve accessibility.

Q.18 What are the indications, advantages and disadvantages of coronally displaced flap?

Indication:
(i) Cover denuded root surface with adequate width of keratinized gingiva.

Advantages:
(i) No shortening of vestibule.
(ii) Can treat multiple areas of recession.
(iii) Flaps are not under tension and requires no sutures.

Disadvantages:
(i) Fails if adjacent papillae are not wide enough because flap derives its blood supply from adjacent papillae.
(ii) Cannot be used in mandibular teeth with narrow interdental papillae.

Q.19 What are the advantages, disadvantages and contraindications of laterally positioned pedicle flap?

Advantages:
(i) Single surgical site
(ii) Esthetic, close color blend
(iii) Good vascularity

Disadvantages:
(i) Dehiscence or fenestration at the donor site
(ii) Possibility of recession at donor site
(iii) Limited to one or two teeth with recession

Contraindications:

(i) Shallow vestibule
(ii) Lack of keratinized attached gingiva
(iii) Excessive root prominence
(iv) Presence of deep interproximal pockets
(v) Deep or extensive root abrasion / erosion
(vi) Significant loss of interproximal bone height

Q.20 What are the indications, advantages and disadvantages of Double Papilla flap procedures?

Indications:

(i) When the interproximal papillae adjacent to the mucogingival problems are sufficiently wide.
(ii) When the attached gingiva on an approximating tooth is insufficient to allow for a laterally positioned flap.
(iii) When periodontal pocket is not present.

Advantages:

(i) Risk of resorption of alveolar bone is minimized because interdental bone is more resistant to loss than is radicular bone.
(ii) Clinical predictability of the procedure is good.
(iii) Papilla usually supplies a greater width of attached gingiva than radicular surface of tooth.

Disadvantages:

(i) Two flaps are sutured over root surface.
(ii) Manipulation of freed papilla during suturing is difficult.

Q.21 What are the advantages of distal wedge procedure?

(i) Maintenance of attached tissue
(ii) Accessibility for treatment of both distal furcation and underlying osseous irregularities.

Q.22 What are the various distal wedge procedure designs?

(i) Triangular
(ii) Square, parallel or H design
(iii) Linear or pedicle.

Q.23 Compare flap procedure with gingivectomy.

		Flap Procedure	*Gingivectomy*
1	Reattachment	Possible	No
2	Healing	Primary intention	Secondary intention
3	Bleeding postoperatively	Low	High
4	Preservation of keratinized gingiva	Yes	No
5	Visibility and ability to treat osseous irregularities and defects	Good	Inadequate
6	Time required	Slow	Fast
7	Degree of difficulty	High	Low

Q.24 Which flap procedure is considered as Internal bevel gingivectomy?
Undisplaced flap.

Q.25 Which flap procedures surgically remove the pocket wall?
(i) Undisplaced flap
(ii) Gingivectomy.

Q.26 What are the differences in incision and flap for regenerative surgery from other surgical procedures?
(i) Papilla preservation or conventional flap or sulcular incision is given in regenerative surgical procedure.
(ii) Incision for regenerative surgery is given such as to preserve the maximum amount of gingival tissue.
(iii) Do not thin the flap in regenerative surgery as for other surgical procedures.

D. FURCATION

Q.1 What is furcation, furcation involvement, furcation entrance and furcation fornix?
(i) Furcation is the area located between individual root cones.
(ii) Furcation involvement is the extension of pocket formation into interradicular area of bone of multi-rooted tooth.
(iii) Furcation entrance is the transitional area between the undivided and divided part of the root.
(iv) Furcation fornix is the roof of the furcation.

Q.2 What is degree of separation and co-efficient of separation?
(i) Degree of separation is the angle of separation between two roots (cones).
(ii) Co-efficient of separation is the length of root cones in relation to the length of root complex.

Q.3 Classify furcation lesion/involvement.
A. According to Glickman (1953):
Grade I: It is the incipient stage of furcation involvement, but radiographically changes are not usually found.
Grade II: The furcation lesion is a cul-de-sac with a definite horizontal component. Radiographs may or may not depict the furcation involvement.
Grade III: The bone is not attached to the dome of the furcation. Class III furcations display the defect as a radiolucent area in the crotch of the tooth.
Grade IV: The interdental bone is destroyed and soft tissue has receded apically so that the furcation opening is clinically visible.

B. According to Hamp et al (1975):

Degree I: Horizontal loss of periodontal support not exceeding 1/3rd of the width of the tooth.

Degree II: Horizontal loss of periodontal support exceeding 1/3rd of the width of the tooth.

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

C. According to Tarnow and Fletcher (1984): Based on vertical component. Depending on the distance from the base of the defect to the roof of the furcation:

Subgroup A: Vertical destruction of bone upto 1/3rd of the inter-radicular height (1-3 mm).

Subgroup B: Vertical destruction of bone upto 2/3rd of the inter – radicular height (4-6 mm).

Subgroup C: Vertical destruction beyond the apical-third (7 mm or more).

Q.4 Write etiological factors for furcation lesion.

(i) Extension of inflammatory periodontal disease
(ii) Cervical enamel projections (CEPs)
(iii) Trauma from occlusion
(iv) Pulpal periodontal disease
(v) Iatrogenic cofactors – Pin and endodontic perforations Overhanging restorations.

Q.5 What are the various anatomic factors which influence the treatment of furcation?

(i) Root trunk length:
- Less attachment has to be lost before furcation is involved if root trunk is short.
- Short root trunk facilitate surgical procedure and are more accessible to maintenance therapy than long root trunk.

(ii) Root length:
- It is directly related to the quantity of attachment supporting the tooth.

(iii) Root form:
- Curvature and fluting increases the potential for root perforation during endodontics and vertical root fracture.

(iv) Interradicular dimensions:
- Narrow, inter-radicular zone complicate the surgical procedure.
- Divergent roots have more treatment options and easily hemisected, readily treated.

- In divergent rooted tooth adequate instrumentation can be done during scaling, root planing and surgery.

(v) Anatomy of furcation:
- Bifurcational ridges, concavity in the dome and accessory canals complicates scaling, root planing, surgical procedures and maintenance.

(vi) CEPs:
- Affect plaque removal
- Complicate scaling and root planing
- Act as local factor in the development of gingivitis and periodontitis.

Q.6 What is the distance of furcation entrance from CEJ in case of maxillary molars, maxillary premolars and mandibular premolars?

(i) Maxillary molars –
- Mesial furcation entrance is located about 3 mm from CEJ
- Distal furcation entrance is located about 5 mm from CEJ
- Buccal furcation entrance is located about 4 mm from CEJ

(ii) Maxillary premolars – 8 mm

(iii) Mandibular premolars –
- Buccal – 3 mm
- Lingual – 4 mm.

Q.7 What is the width of furcation entrance of maxillary premolars, maxillary molars and mandibular molars?

(i) Maxillary premolars – Approx. 0.7 mm

(ii) Maxillary molars –
- Buccal 0.5 mm (Narrower than mesial and distal)
- Mesial 0.75 mm
- Distal 0.5 mm to 0.75 mm

(iii) Mandibular molars –
- Buccal Less than 0.75 mm
- Lingual More than 0.75 mm

Q.8 Why the dimensions of furcation entrance should be taken into consideration during the selection of instruments?

According to Bower, 81% of furcations of maxillary and mandibular permanent first molar has orifices that are 1 mm or less wide, while in 58% molars, furcation entrance is narrower (0.75 mm or less) than the width of the blade of standard curettes (greater than 0.75 mm).

Q.9 Classify Cervical Enamel Projections (CEPs).

Masters and Hoskins in 1964 classified CEPs into 3 grades:
- Grade I – The enamel projection extends from CEJ towards the furcation entrance.

- Grade II – Enamel projection approaches the entrance of the furcation without entering the furcation with no horizontal component.
- Grade III – Enamel projection extends horizontally into the furcation.

Q.10 In which teeth the prevalence of CEPs is highest/maximum?

Mandibular and maxillary second molars.

Q.11 How furcation defects can be diagnosed?

Furcation can be diagnosed:

(i) Clinically: By using Naber's probe and transgingival probing / bone sounding.

(ii) Radiographically: Radiographs also help to detect the furcation defects.

Q.12 From which aspect mesial furcation of maxillary molar is easily probed?

Mesial furcation of maxillary molar is easily probed from the palatal aspect because mesial furcation opens about 2 / 3rd of the way towards the palate, rather than midway buccolingually.

Q.13 What are the objectives of furcation therapy?

(i) To facilitate maintenance of existing furcation defect – Scaling and root planing.

(ii) To increase access to the furcation – Gingivectomy, apically positioned flap, odontoplasty, ostectomy / osteoplasty and tunnel preparation.

(iii) To prevent further attachment loss or eliminate the furcation– Root amputation, tooth resection and hemisection.

(iv) To obliterate the furcation defect – Filling furcation defects with biocompatible material such as polymeric reinforced zinc oxide eugenol (IRM) and GIC.

(v) To regenerate the lost attachment – through GTR procedures and bone grafting.

Q.14 What are the factors to be considered during treatment of furcation involved molars?

A. Tooth-related factors:

(i) Degree of furcation involved
(ii) Amount of remaining periodontal support
(iii) Probing depth
(iv) Tooth mobility
(v) Root trunk length
(vi) Root length
(vii) Root form
(viii) Interradicular dimensions

(ix) Anatomy of furcation
(x) Cervical enamel projections
(xi) Tooth position and occlusal antagonisms
(xii) Endodontic conditions and root canal anatomy

B. Patient-related factors:
(i) Strategic value of the tooth in relation to the overall plan
(ii) Patient's age and health condition
(iii) Oral hygiene capacity.

Q.15 What are the treatment modalities for Class I, II, III and IV furcation defects?

Class I furcation defects:
- Oral hygiene
- Scaling and root planing
- Odontoplasty
- Recontouring

Class II furcation defects:
- Open flap debridement
- Guided tissue regeneration
- Bone grafting

Class III and Class IV furcation defects:
- Tunnel preparation
- Hemisection
- Root resection
- Root amputation

Advanced Class IV furcation defects:
- Extraction.

Q.16 What is Furcationplasty?

In 1975, Hamp, Nyman and Lindhe described furcationplasty as raising a mucoperiosteal flap to provide access to the furcation area, and combining scaling and root planing , osteoplasty and odontoplasty to remove local irritants and to open the furcation to allow the patient access to clean and maintain the area. It is done in Grade I and early Grade II furcation lesions.

Q.17 What is the difference between Tooth resection, Root amputation, Sectioning and Hemisection?

- Tooth resection involves removal of one or more roots of tooth as well as corresponding portion of the crown.
- Root amputation is the removal of one or more roots from a multi-rooted tooth leaving the majority of crown intact.
- Sectioning is the surgical sectioning of a tooth into segments consisting of the root and overlying crown.
- Hemisection is the splitting of a two rooted tooth into two separate portions.

Q.18 What are the indications and contraindications of hemisection?

Indications:

(i) Strategic teeth with Grade III furcation involvement
(ii) Teeth with divergent well supported roots

Contraindications:

(i) When remaining periodontal support is inadequate
(ii) Tooth that cannot be treated endodontically
(iii) Adequate restorations of the remaining tooth including splinting cannot be performed.

Q.19 What is Non-vital and Vital root resection?

(i) Non vital root resection – The endodontic therapy is done prior to root resection.
(ii) Vital root resection - The root resection is accomplished first and then endodontic therapy.

Q.20 What are the factors to be considered while selecting the case of tunnel preparation?

(i) Mandibular molars
(ii) Low caries index
(iii) Root trunk should be short with high furcation entrance and long roots
(iv) Wide furcal entrance
(v) The floor of the pulp chamber should not be close to the roof of the furcation.

Q.21 What are the drawbacks of tunnel preparation?

(i) Dental caries
(ii) Subsequent pulpal pathology
(iii) Reverse architecture
(iv) Retained plaque in furcation cavities leading to progressive periodontal breakdown.

Q.22 What are the indications of extraction of furcation involved tooth?

(i) Individuals who do not maintain oral hygiene
(ii) Patients with high level of caries activity
(iii) Financial consideration
(iv) If an otherwise heroic effort to save a tooth with a questionable prognosis would be better handled by an implant.

Q.23 What are the causes of failures in surgical furcation therapy?

(i) Inadequate plaque control and maintenance
(ii) Poor root resection
(iii) Improper restoration
(iv) Endodontic failures
(v) Cracked roots
(vi) Root caries
(vii) Patients who respond poorly, despite best treatment.

E. PERIODONTAL PLASTIC SURGERY

Q.1 What is Periodontal plastic surgery?

It is defined as the surgical procedures performed to correct / eliminate anatomic, development / traumatic deformities of the gingiva / alveolar mucosa. It includes the following:

(i) Periodontal prosthetic corrections
(ii) Crown lengthening
(iii) Ridge augmentation
(iv) Esthetic surgical corrections
(v) Coverage of the denuded root surface
(vi) Reconstruction of papillae
(vii) Esthetic surgical correction around implants
(viii) Surgical exposure of unerupted teeth for orthodontics.

Q.2 What are the objectives of periodontal plastic surgery?

To deal with the problems associated with:

(i) Attached gingiva
(ii) Shallow vestibule
(iii) Aberrant frenum

Q.3 What are various mucogingival problems?

(i) Inadequate width of attached gingiva
(ii) Abnormal frenum attachment
(iii) Gingival recession
(iv) Decreased vestibular depth.

Q.4 Write criteria for selection of techniques for solving mucogingival problems.

(i) Surgical site free of plaque, calculus and inflammation.
(ii) Adequate blood supply to the donor tissue.
(iii) Anatomy of the recipient and donor site.
(iv) Stability of the grafted tissue to the recipient site.
(v) Minimal trauma to the surgical site.
(vi) Extension of pockets.

Q.5 What are the various techniques for increasing attached gingiva?

A. Gingival augmentation apical to recession:
   (a) Free epithelial autograft
   (b) Free connective tissue autograft
   (c) Apically positioned graft
   (d) Fenestration
   (e) Vestibular extension

B. Gingival augmentation coronal to recession / root coverage:
   (a) Free epithelial autograft
   (b) Free connective tissue autograft

c. Pedicle autografts:
  (I) Rotational
    (i) Lateral Pedicle
    (ii) Double Papilla
  (II) Advanced
    (i) Coronally displaced
    (ii) Semilunar
(d) Subepithelial connective tissue
(e) Subpedicle connective tissue
(f) Pouch and tunnel technique
(g) Envelope technique
(h) Guided tissue regeneration technique

Q.6 Write etiological factors responsible for gingival recession.

(I) Anatomical / Developmental factors:
  A. Dehiscence –
    (i) Abnormal direction of tooth eruption
    (ii) Malposition of teeth
    (iii) Buccolingual thickness of root is more than crestal bone thickness
    (iv) Morphotypes having narrow long teeth
    (v) Orthodontic tooth movement
  B. Fenestration
  C. Lack of attached gingiva
  D. Abnormal path of tooth eruption
  E. Individual tooth shape
  F. Tooth eruption compensation
  G. Abnormal tooth position in the arch
(II) Physiological factors:
  A. Senile atrophy / aging process
  B. Genetic predisposition
  C. Orthodontic movement of teeth -
    (i) Controlled
    (ii) Erratic
(III) Pathological factors:
  A. Gingivitis / Periodontitis
  B. Chronic trauma:
    (i) Impaction of foreign bodies against gingiva
    (ii) Factitious injuries
    (iii) Fingernail scratching of the gums
    (iv) Over rigorous and incorrect tooth brushing
    (v) Occlusal injury

C. Frenal pull
D. Tobacco chewing
E. Acute traumatic injuries
F. Psychological factors – stress and emotions.

Q.7 Write etiopathogenesis of gingival recession.

It is based on inflammation and subsequent destruction of connective tissue of free gingiva. The oral epithelium migrates to the borders of destroyed connective tissue. The thickening of gingival and sulcular basal lamina reduces the quantity of connective tissue between them. Thus blood supply is reduced, negatively influencing the repair of initial lesion. As the lesion progresses, connective tissue disappears and oral epithelium fuse with junctional or sulcular epithelium. In recession caused by plaque and calculus, initial ulcer appears in the junctional epithelium of sulcus and destruction of connective tissue occurs from inside out. In toothbrush trauma lesions, destruction occurs from outside in.

Q.8 Classify recession defects.

I. According to Miller:
   - Class I: Marginal tissue recession not extending to the mucogingival junction. No loss of interdental bone/soft tissue.
   - Class II: Marginal tissue recession extends to or beyond the mucogingival junction. No loss of interdental bone/soft tissue.
   - Class III: Marginal tissue recession extends to or beyond the mucogingival junction. Loss of interdental bone/soft tissue or there is malpositioning of the tooth.
   - Class IV: Marginal tissue recession extends beyond the mucogingival junction. Loss of interdental bone and soft tissue loss interdentally and/or severe tooth malposition.

II. According to Sullivan and Atkins:
   - Shallow-narrow
   - Shallow-wide
   - Deep-narrow
   - Deep-wide

Q.9 What are the indications for root coverage procedure?

(i) To reduce root sensitivity
(ii) To improve esthetic
(iii) To manage defects resulting from root caries removal or cervical abrasions.

Q.10 Classify papillary height.

According to Nordland and Tarnow (1998):

Normal: The interdental papilla occupies the entire embrassure space apical to the interdental contact point / area.

Class I: The tip of the interdental papilla is located between the interdental contact point and the level of the CEJ on the proximal surface of the tooth.

Class II: The tip of the interdental papilla is located at or apical to the level of the CEJ on the proximal surface of the tooth but coronal to the level of the CEJ mid-buccally.

Class III: The tip of the interdental papilla is located at or apical to the level of CEJ mid-buccally.

Q.11 What are the causes of loss of interdental papilla?

(i) Tooth extraction
(ii) Excessive surgical periodontal treatment
(iii) Localized progressive gingival and periodontal lesion.

Q.12 What are the effects of loss of interdental papilla?

(i) Lead to cosmetic deformities
(ii) Phonetic problems
(iii) Lateral food impaction

Q.13 What are the various methods to create interdental papilla?

A. Non-surgical papilla creation:
   (a) If interdental papilla is absent because of diastema – orthodontic closure is the treatment
   (b) Orthodontic forced eruption
   (c) Repeated scaling, root planing and curettage procedure.
B. Surgical papilla creation:
   (a) Pedicle graft technique
   (b) Semilunar coronally repositioned papilla

Q.14 Classify soft tissue procedures used for Root coverage.

Cohen classified soft tissue grafting procedures as:

A. Free soft tissue autografts:
   a. Epithelial graft
   b. Subepithelial connective tissue graft
B. Contiguous / pedicle soft tissue autografts:
   a. Rotational flap –
      • Laterally positioned flap
      • Double papillae flaps
   b. Advanced flap –
      • Coronally repositioned flap.

Q.15 What is the ideal thickness of the graft?

Between 1.0 mm and 1.5 mm.

Thinner graft shrivel and expose the recipient site. Thicker graft jeopardizes the circulation and nutrient diffusion.

Q.16 What are the drawbacks of epithelialized palatal graft for the root coverage procedure?

(i) Blood supply to the graft is available on only one surface, rather than two, as with the connective tissue graft.
(ii) Color match of the tissues is a problem between the grafted area and the adjacent tissues.
(iii) Palatal wound is more invasive, more prone to hemorrhage and slower to heal.
(iv) It is technique sensitive and time consuming.

Q.17 What are the various ways for root coverage after graft procedure?

(i) Primary root coverage – found initially after grafting
(ii) Secondary root coverage – through creeping attachment

Q.18 What are the factors associated with incomplete coverage?

(i) Improper classification of marginal tissue recession
(ii) Inadequate root planing
(iii) Failure to treat planed root with citric acid
(iv) Improper preparation of recipient site
(v) Inadequate size of interdental papillae
(vi) Improperly prepared donor tissue
(vii) Inadequate graft size
(viii) Inadequate graft thickness
(ix) Dehydration of graft
(x) Inadequate adaptation of graft to root and remaining periosteal bed
(xi) Failure to stabilise the graft
(xii) Excess or prolonged pressure in coadaptation of the graft
(xiii) Reduction of inflammation prior to grafting
(xiv) Trauma to graft during initial healing
(xv) Excessive smoking.

Q.19 What are the various stages of wound healing after grafting?

(i) Plasmatic circulation (0-3 days)
(ii) Vascularization (2-11days) – Anastomoses are established between blood vessels of the recipient bed and those in the grafted tissue characterized by capillary proliferation.
(iii) Organic Union (11-42 days) – Tissue maturation phase.

Q.20 What is creeping attachment?

In 1964, Goldman et al noted a second mechanism of gaining root coverage by phenomenon of creeping attachment. This occurs between 1 month to 1 year and was the result of coronal migration of newly grafted attached gingiva over the portions of a previously denuded root.

Q.21 What is frenectomy and frenotomy?

(i) Frenectomy is the complete removal of the frenum, including its attachment to the underlying bone.

(ii) Frenotomy is the incision of the frenum.

Q.22 What are the advantages of Miller's surgical technique over Edward's technique of frenectomy?

(i) In Miller's technique, there is contiguous collagenous band of gingiva across the midline rather than scar tissue. The collagenous band provide more of a bracing effect than scar tissue.

(ii) Transseptal fibers are not disrupted surgically in Miller's technique which assures no loss of interdental papilla.

Q.23 Name the procedures used to extend gingiva into vestibule.

(i) Gingival extension using a periosteal fenestration.

(ii) Vestibuloplasty as a modified Edlan – Mejchar procedure

(iii) Gingival extension with a free epithelial graft.

Q.24 What is vestibuloplasty? What is its objective?

Vestibuloplasty is a procedure designed to extend the vestibular fornix.

Objectives:

(i) To enhance plaque control by allowing space for effective use of plaque control aids.

(ii) To gain more retention for removable prosthetic appliances by expanding the prosthesis bed.

F. RESECTIVE OSSEOUS SURGERY

Q.1 What is resective osseous surgery?

The procedure designed to restore the form of preexisting alveolar bone to the level existing at the time of surgery or slightly more apical to this level is called as resective osseous surgery.

Q.2 What is the rationale of resective osseous surgery?

To achieve physiologic architecture of marginal alveolar bone conducive to gingival flap adaption with minimal probing depth.

Q.3 What is Ideal, Positive, Negative and Flat architecture of bone?

(i) Ideal architecture: The bone level is more coronal in the interproximal areas, with a gradual slope around and away from the tooth.

(ii) Positive architecture: The level of radicular bone is apical to the interdental bone.

(iii) Negative architecture: The level of interdental bone is more apical to radicular bone. It is also called as Reverse architecture.

(iv) Flat architecture: The interdental bone is at the same level to that of radicular bone.

Q.4 What is the difference between osteoplasty and ostectomy?

(i) Osteoplasty – It is a procedure to create a physiologic form of alveolar bone without removing any supporting bone.

(ii) Ostectomy – It is a procedure in which supporting bone, i.e. bone involved in the attachment of tooth is removed to reshape deformities.

Q.5 What are the indications of Osteoplasty?

(i) Removing exotoses / ledges

(ii) Tori that interferes with plaque control and lead to persistent pocket formation

(iii) Early Grade I furcation lesion

(iv) To contour alveolar ridges to make room for pontics

(v) Open furcation in tunneling procedures.

Q.6 What are the indications of Ostectomy?

(i) Crown lengthening

(ii) Exposure of sound dentin apical to caries / fractures

(iii) Opening of interradicular spaces for the treatment of furcation involvement.

Q.7 What are the steps in resective osseous surgery?

The resective osseous technique is carried out in 4 steps:-

(i) Vertical grooving / festooning

(ii) Radicular grooving

(iii) Flattening interproximal bone / horizontal grooving

(iv) Gradualizing marginal bone

Q.8 Name instruments used for resective osseous surgery.

(i) Rongeurs:
- Friedman
- Blumenthal

(ii) Files:
- Schulger
- Sugarman

(iii) Chisels:
- Backaction
- Ochsenbein

(iv) Burs:
- Carbide
- Diamond.

Q.9 What is Spheroiding/Parabolizing and Scribbing?

(i) Spheroiding / Parabolizing: It is removal of supporting bone to produce a positive gingival and osseous architecture.

(ii) Scribbing: It is the technique by which high speed rotatory instrumentation is used to outline on the radicular bone, that bone which is to be removed by hand instrumentation.

Q.10 What is widow's peaks?

Schulger 1949 described widow's peaks as the residual pieces of cortical bone left over facial or lingual line angle from the horizontal grooving that form a crater in a mesiodistal direction. They will not be absorbed and will result in immediate postoperative tissue pocketing. Hand instrumentation with Ochsenbein chisel is used to eliminate widow's peak.

Q.11 Which is the most predictable pocket reduction technique?

Resective osseous surgery.

G. REGENERATIVE OSSEOUS SURGERY

Q.1 What are the possible outcomes of surgical periodontal therapy?

(i) New attachment
(ii) Long junctional epithelium
(iii) Root resorption / ankylosis
(iv) Recurrence of pocket.

Q.2 What is repair, regeneration, new attachment, reattachment and periodontal regeneration?

(i) Repair: Healing of a wound by a tissue that does not fully restore the architecture / function of the part.
(ii) Regeneration: It refers to the reproduction / reconstruction of a lost / injured tissue.
(iii) New attachment: It is defined as the union of connective tissue or epithelium with a root surface that has been deprived of its original attachment apparatus.
(iv) Reattachment: It describes reunion of epithelial and connective tissue with a root surface.
(v) Periodontal regeneration: It is defined as the restoration of lost periodontium / supporting tissues and includes formation of new alveolar bone, new cementum and new periodontal ligament.

Q.3 Define Guided Tissue Regeneration (GTR).

The 1996, World Workshop in periodontics defined GTR as "procedures attempting to regenerate lost periodontal structures through differential tissue responses. Barriers are employed in the hope of excluding epithelium and gingival corium from the root surface in the belief that they interfere with regeneration."

Q.4 What is the rationale behind using GTR barrier membranes?

(i) Exclusion of epithelium and gingival connective tissue
(ii) Barriers maintain space between defect and barrier
(iii) Stabilize the clot.

Q.5 What are the various biologic requirements of the membrane used in GTR?

(i) Tissue integration
(ii) Cell occlusivity
(iii) Clinical manageability
(iv) Space making
(v) Biocompatiblity.

Q.6 What is contact inhibition?

George Winter, an English researcher had proposed that specific porosities ingrew with connective tissue, stopped or slowed the migration and pocketing of epithelial tissues. He called this phenomenon as contact inhibition.

Q.7 What are the indications and contraindications for GTR?

Indications:

(i) Narrow 2 or 3 wall infrabony defects
(ii) Circumferential defects
(iii) Class II furcation defects
(iv) Recession defects

Contraindications:

(i) Any medical condition contraindicating surgery
(ii) Infection at defect site
(iii) Poor oral hygiene
(iv) Smoking (Heavy)
(v) Tooth mobility > 1 mm
(vi) Defect < 4 mm deep
(vii) Width of attached gingiva at defect site $\leq$ 1 mm
(viii) Thickness of attached gingiva at defect site $\leq$ 0.5 mm
(ix) Furcation with short root trunks
(x) Generalized horizontal bone loss
(xi) Advanced lesions with little remaining support
(xii) Multiple defects.

Q.8 What are the factors that can adversely affect clinical outcome after GTR therapy?

A. **Barrier dependent factors:**
1. Inadequate root – barrier adaptation
2. Non – sterile technique: plaque / saliva contamination of barrier
3. Instability (movement) of barrier against root
4. Premature exposure of barrier to oral environment and microbes
5. Premature loss or degradation of barrier.

B. **Barrier independent factors:**
1. Poor plaque control
2. Smoking
3. Occlusal trauma: hyperocclusion

4. Suboptimal tissue health: inflammation persists
5. Mechanical habits: aggressive tooth-brushing techniques
6. Overlying gingival tissue:
   (a) Inadequate zone of keratinized tissue
   (b) Inadequate tissue thickness
7. Surgical technique:
   (a) Improper incision placement: excessive loss of marginal tissue
   (b) Traumatic flap elevation and management
   (c) Excessive surgical tissue: tissue / flap desiccation
   (d) Inadequate closure / suturing: Failure to achieve and maintain primary closure.
8. Postsurgical factor:
   (a) Premature tissue challenge
      • Plaque recolonization
      • Mechanical insult
   (b) Loss of wound stability – Loose sutures, loss of early fibrin clot

Factors that can limit regenerative healing after GTR surgery are given above and most important among these are the presence of smoking habit, poor plaque control and premature exposure of barrier material.

Q.9 What are the various materials used for GTR?

A. **First generation material. Non-resorbable**
   (a) ePTFE: expanded polytetrafluoroethylene, GORE-TEX membrane
   (b) dPTFE: dense polytetrafluoroethylene
   (c) Nucleopore
   (d) Millipore filters: ethyl cellulose
   (e) Ultrathin: semipermeable silicone barrier

B. **Second generation material. Resorbable**
   (a) Collagen –
      • Biomend
      • Biomend - extend
      • Periogen
      • Paroguide
      • Biostite
      • Biogide
      • Tissue Guide
      • Biobar
   (b) Polylactide and Polyglycolide –
      • Guidor
      • Vicryl
      • Atrisorb

- Resolut
- Epiguide
- Biofix

(c) Others –

- Periosteum
- Connective tissue graft
- Alloderm
- Emdogain
- Surgicel
- Gelform
- Gengiflex
- Capset
- Hapset
- Cargile membrane

C. **Third generation material**. Resorbable bioactive barrier membranes with added growth factors.

Q.10 Write about the structure of various absorbable GTR barrier material.

(i) Guidor: It is a hydrophobic barrier material from Polylactic acid (PLA) combined with a citric acid ester softening agent. It is a bilayered consisting of an external layer having large rectangular perforations (400-500 /cm^2) and internal layer having smaller circular perforations (4000-5000 /cm^2).

(ii) Vicryl: It is made from copolymer of glycolide and lactide. It is available in two forms:

(a) Knitted mesh: These have large pore size with better handling property.

(b) Woven mesh: These have smaller pore size but tends to fray.

(iii) Atrisorb: It is a polymer of lactic acid, poly (D,L – lactic acid), dissolved in N- methyl – 2 pyrrolidone (NMP). It is prepared as a solution that coagulates or sets to a firm consistency on contact with water / other aqueous solution; this principle is used in forming a barrier that is partially coagulated to a semirigid state in a chairside mixing kit, which can be trimmed to the dimensions of defect.

(iv) Resolut: It is a copolymer of PGA and PLA.

(v) Epiguide: It is a hydrophilic membrane formed from PLA (D, L – form). It contains flexible open cell structure and internal void spaces.

Q.11 What are the various cross linking agents used in collagen barrier membrane?

(i) Physical agents:

- Gamma radiation
- UV radiation

(ii) Chemical agents:
- Formaldehyde
- Diphenylphosphorylazide (DPPA).

Q.12 Why is collagen membrane cross linked?

(i) To extend absorption time
(ii) To reduce antigencity.

Q.13 What are the various advantages and disadvantages of absorbable barrier material?

Advantages:

(i) Elimination of second surgery for barrier removal
(ii) Reduce operatory time
(iii) Increase patient acceptance
(iv) Reduce risk of loss of regenerated attachment owing to reentry surgery
(v) More tissue friendly

Disadvantages:

(i) Instability of barrier against root
(ii) High cost
(iii) Biodegradation rate cannot be controlled
(iv) In case of infection or strong tissue response, if there is a need to remove the membrane, disintegration of the material in its various stages, makes it impossible.

Q.14 Name the agents which are used for Root Biomodification.

(i) Citric acid
(ii) Tetracycline
(iii) Fibronectin
(iv) EDTA
(v) LASERS
(vi) Laminin – glycoprotein
(vii) Sodium Deoxycholate and Human Plasma fraction Cohn IV
(viii) Growth factors – PDGF, bFGF, IGF, TGF.

Q.15 Classify ridge defects.

(i) According to Seibert (1983):
Class I: Loss of buccolingual width but normal apicocoronal height.
Class II: Loss of apicocoronal height but normal buccolingual width.
Class III: A combination of loss of both height and width of the ridge.

(ii) According to Allen et al, modification of Seibert classification:
Type A: Apicocoronal loss of ridge contour
Type B: Buccolingual loss of ridge contour

Type C: Combined loss of ridge contour in both apicocoronal and buccolingual dimensions.

(iii) Can also be classified according to depth of defect
Mild: less than 3 mm
Moderate: 3 to 6 mm
Severe: greater than 6 mm.

Q.16 Classify bone graft materials.

A. According to the graft obtained from:

(i) Autogenous grafts: Grafts transferred from one position to another within the same individual.
  (a) Cortical bone
  (b) Cancellous bone

(ii) Allogenic graft: Grafts transferred between genetically dissimilar members of the same species.
  (a) Undemineralized freeze dried bone allograft
  (b) Demineralized freeze dried bone allograft

(iii) Xenogenic graft: Grafts taken from a donor of another species

(iv) Alloplastic material: Synthetic or inorganic implant materials which are used as substitutes for bone grafts
  (a) Hydroxyapatites (relatively nonresorbable)
  (b) Tricalcium phosphates (resorbable)
  (c) Polymers
  (d) Bioactive glasses.

B. According to their mode of action:

(i) Osteogenetic / osteoproliferative: Means that new bone is formed by bone forming cells contained in the graft.

(ii) Osteoinductive: Means that bone formation is induced in the surrounding soft tissue immediately adjacent to the graft.

(iii) Osteoconductive: Means that the grafted material does not contribute to new bone formation but serve as scaffold for bone formation originating from adjacent host bone.

Q.17 What are the various sites for procuring autogenous bone graft?

A. Intraoral sites:

(i) Healing extraction wounds
(ii) Edentulous ridges
(iii) Exostoses
(iv) Lingual ridge on the mandible
(v) Bone distal to a terminal tooth
(vi) Lingual surface of the mandible at least 5 mm from the roots.
(vii) Maxillary tuberosity

B. Extra oral sites:

(i) Iliac autografts – Posterior iliac crest.

Q.18 What are the problems associated with iliac autografts?

(i) Post operative infection
(ii) Exfoliation
(iii) Sequestration
(iv) Varying rates of healing
(v) Root resorption
(vi) Rapid recurrence of the defect.

Q.19 What is Osseous Coagulum, Bone Blend and Bone Swaging?

(i) Osseous Coagulum: It is a mixture of bone dust obtained by grounding cortical bone and blood. Round carbide bur revolving at 25,000-30,000 rpm is used within the surgical site to reduce donor bone to small particles, which when coated with the patient's blood to become a coagulum.
(ii) Bone Swaging: It is the technique which requires existence of an edentulous area adjacent to the defect. The bone is pushed from edentulous area into contact with root surface without fracturing the bone at its base.
(iii) Bone Blend: It involves removing bone (cortical, cancellous or both) from accessible intraoral donor site by chisel / rongeur forceps, placing it in a sterile plastic amalgam capsule with pestle and then triturating it.

Q.20 What are the advantages and disadvantages of osseous coagulum?

Advantages:

(i) Additional surgical site not required to procure donor material
(ii) Relatively rapid technique
(iii) Complements osseous resective procedures that may be required within surgical site
(iv) Particle size provides additional surface area for the interaction between cellular and vascular elements

Disadvantages:

(i) Cannot be used in larger defects because of inability to procure adequate material
(ii) Poor surgical visibility
(iii) Relatively low predictability
(iv) Inability to use aspiration during accumulation of the coagulum
(v) Fluidity of the material makes it difficult to transfer the coagulum to the defect.

Q.21 Compare freeze dried bone allograft (FDBA) and demineralized freeze dried bone allograft (DFDBA).

	FDBA	*DFDBA*
1	Not demineralized	Demineralized
2	More radiopaque	More radiolucent
3	Breakdown by way of foreign body reaction	Rapid resorption
4	Primary indication: Bone augmentation associated with implant treatment	Primary indication: Periodontal disease associated with natural tooth
5	Osteoconductive	Osteoinductive
6	No bone morphogenetic protein expression	More bone morphogenetic protein

Q.22 What is the advantage and disadvantage of allograft?

Advantage:

- Does not require additional surgical site for the removal of donor material from the same patient.

Disadvantage:

- Can provoke an immune response.

Q.23 What are the various methods to suppress the antigenic potential of allograft and xenograft?

(i) Radiation treatment: 6 Mega Rads of high intensity of Gamma radiation is adequate.

(ii) Freezing: Deep frozen -197° C liquid Nitrogen freezer for a period of atleast 4 weeks.

(iii) Chemical treatment: Through keeping in Merthiolate solution

Q.24 How were bone morphogenetic proteins (BMPs) discovered?

The history of the identification and purification of bone morphogenetic proteins began in 1965, when Marshall Urist demonstrated that the cellular events associated with embryonic bone development could be reproduced in heterotrophic sites by implants of demineralized bone segments. In the late 1960s and early 1970s, it was recognized that dentin also contained bone morphogenetic activity.

Q.25 What is bone morphogenetic protein (BMPs)? What are the types of BMPs?

Bone morphogenetic proteins form a subgroup of a larger family of structurally related proteins known as the transforming growth factor-β superfamily. They are identified by their capacity to induce bone in vivo or extraskeletal sites in mammals.

Types:

Atleast 15 BMPs have been identified up to date.

(i) BMP-1 Protease; not osteoinductive

(ii) BMP-2 Osteoinductive; located in bone, spleen, liver, brain, kidney

(iii) BMP-3 Osteogenin osteoinductive, located in lung, kidney, brain

(iv) BMP-4 Osteoinductive, located in apical ectodermal ridge, meninges, lung, kidney, liver
(v) BMP-5 Osteoinductive, located in lung, kidney, liver
(vi) BMP-6 Not osteoinductive, found in lung, brain, kidney, uterus, muscle, skin
(vii) BMP-7 Osteoinductive, located in adrenal glands, placental, spleen, skeletal muscle
(viii) BMP-8 Osteoinductive
(ix) BMP-9 Osteoinductive; stimulates hepatocyte proliferation; hepatocycte growth and function
(x) BMP-12 and BMP-13 Inhibition of terminal differentiation of myloblasts.

Q.26 What are the advantages and limitations of sclera as non bone graft material ?

Advantages:
(i) Easily sterilized
(ii) Minimal cellularity
(iii) Poor vascularity
(iv) Decrease antigencity.

Limitation:
(i) Sclera has tissue memory and tends to return to its original curvature limits its application to wide and shallow defects.

Q.27 What is Tissucol?

Tissucol is a lyophilized cryoprecipitate of human blood plasma composed of fibrinogen, factor XIII, fibronectin, platelet-derived growth factor (PDGF), plasminogen, thrombin, antiplasmine, aprotinine, calcium chloride and distillated water. The material has the capacity to interact with the coagulation mechanism, stabilizing the clot, accelerating the colonization by the fibroblasts, raising the concentration of growth factors, delaying the clot disintegration up to 6-7 days and helps in wound healing.

Q.28 What is Emdogain?

It is a resorbable, implantable material that consists of enamel matrix proteins extracted from developing embryonic enamel of porcine origin supplied in sterile, lyophilized form. It is a material intended to regenerate the supporting tissues of the teeth-acellular cementum, periodontal ligament, alveolar bone by grafting proteins called amelogenin.

During clinical application, this hydrophyle protein is solubilized in a sterile vehicle of propylene glycol alginate (PGA). Reaching the periodontal lesion, the vehicle from viscid becomes watery and leaves the application site, permitting the amelogenin to lay down as a matrix

on the diseased root surface. The layer is absorbed by the mineral or collagenic component of the exposed root surface in 1-2 weeks. Amelogenin is then degraded by enzymes, liberating natural aminoacids or peptidic fragments, which are eliminated afterwards.

Emdogain has two presentation forms:

(i) 2 small bottles containing the vehicle and the protein powder
(ii) Syringe with gel

The material is stored in the refrigerator, at 2 - 8ºC. It should be used in no more than 2 hours from opening, because it gelifies and hardens.

Indications:-

(i) 1, 2 and 3 walled defects
(ii) Recession defects
(iii) Class II mandibular furcation defects

Q.29 What is Platelet rich plasma (PRP)?

PRP is an autologue thrombocyte concentrate. The thrombocytes contain various components with hemostatic effects, but also factors that stimulate the healing process. It induces neoformation of blood vessels, which is essential in the process of regeneration. Because PRP has an osteostimulant, not osteoinductive effect, in case of using synthetic materials it is recommended to add a small amount of autologous bone. It accelerates the bone maturation, and the bone quality is also enhanced. PRP is obtained through direct centrifugation from the patient's blood and should be applied as quickly as possible.

Components:

(i) Growth factors
(ii) WBC, phagocytic cells
(iii) Native fibrinogen concentration
(iv) Vasoactive and chemotactic agents
(v) High concentration of platelet

Platelet count in PRP often ranges from 5 lakh to 1 million. Thus, PRP is a way to accelerate and enhance the body's natural wound healing mechanism.

Q.30 What are the various methods used to quantify tissue changes after regenerative periodontal surgery?

(i) Clinical evaluation of attachment levels and other soft tissue parameters.
(ii) Clinical evaluation of hard tissue changes.
(iii) Radiographic evaluation of hard tissue changes.
(iv) Histological evaluation of biopsy material.
(v) Ancillary methods. Gingival Bone Count Index (GBCI): Dunning JM and Leach DB (1960).

Q.31 What are the disadvantages of surgical re-entry?

(i) It requires unnecessary second operation
(ii) It does not show the type of attachment
(iii) It is an unethical issue.

Q.32 What are various Star healing?

(i) One—star healing : Control of inflammation
(ii) Two—star healing : Long junctional epithelium formation
(iii) Three—star healing : New attachment occurs
(iv) Four—star healing : Partial regeneration
(v) Five—star healing : Complete regeneration

25 Interdisciplinary Approach

A. PERIO–ENDODONTICS

Q.1 What are various developmental pulpal-periodontal communications?

(i) Dentinal tubules
(ii) Lateral canals
(iii) Furcal canals
(iv) Apical foramina
(v) Developmental grooves
(vi) Cervical enamel projections and enamel pearl.

Q.2 Classify Endo-perio lesion.

I. According to Simon, Glick and Frank:
   (i) Primary Endo
   (ii) Primary Perio
   (iii) Primary Endo with secondary Perio
   (iv) Primary Perio with secondary Endo

II. According to pathogenesis:
   (i) Endodontic lesion
   (ii) Periodontal lesion
   (iii) True combined lesion
   (iv) Iatrogenic lesion

III. According to Grossman (1991), Endo-perio lesions are classified into 3 groups on the basis of therapy:
   (i) Teeth that require endodontic therapy alone
   (ii) Teeth that require periodontal therapy alone
   (iii) Teeth that require both endodontic as well as periodontal therapy.

Q.3 What are the iatrogenic causes of Perio-endo lesions?

(i) Root perforation
(ii) Overfilling of root canals
(iii) Intra-canal medicaments
(iv) Vertical root fractures

Q.4 What is retrograde periodontitis and retrograde pulpitis?

- Retrograde periodontitis: Long-standing periapical lesion draining through the periodontal ligament can become secondarily complicated leading to retrograde periodontitis.
- Retrograde pulpitis: Bacterial and inflammatory products of periodontitis could gain access to the pulp via accessory canals, apical foramen and dentinal tubules and this reverse effect is called as retrograde pulpitis.

B. PERIO-ORTHODONTICS

Q.1 What are the effects of orthodontic treatment on periodontium?

(i) Favor plaque accumulation
(ii) Modify the gingival ecosystem
(iii) Gingival recession
(iv) Excessive force causes -
  - Necrosis of periodontal ligament and alveolar bone
  - Apical root resorption

Q.2 What are the methods to reduce the occurrence of rotational relapse of the tooth?

(i) Complete correction, or overcorrection of rotated teeth
(ii) Long-term retention with bonded lingual retainer
(iii) Fiberotomy.

Q.3 Which periodontal entities influence the stability of orthodontically treated teeth?

(i) Principal fibers of periodontal ligament
(ii) Supra-alveolar fibers.

Q.4 What are the various techniques for correcting gingival discrepancy in anterior teeth?

(i) Gingivectomy
(ii) Intrusion and incisal restoration / porcelain laminate veneer
(iii) Extrusion, fiberotomy and porcelain crown
(iv) Surgical crown lengthening by flap procedure and osteotomy / osteoplasty of bone.

C. PERIO-RESTORATIVE

Q.1 What is preprosthetic periodontal surgery?

The procedure which aims to treat the periodontal condition and preparing the mouth for ensuing an aesthetic, restorative and prosthetic therapy is called as preprosthetic periodontal surgery.

This includes:

- Crown lengthening
- Ridge augmentation

Q.2 How much time after periodontal plastic surgery, the dental restoration can be placed?

Atleast after 2 months, during which gingival health and osseous topography are restored which prevent the restorative procedure to cause the return of inflammation.

Q.3 What is biologic width?

It is defined as the dimension of healthy gingival tissue, which is attached to the tooth coronal to the crest of the alveolar tissue. Average length of connective tissue attachment is 1.07 mm and of junctional epithelium is 0.97 mm which makes total biologic width of 2.04 mm.

Q.4 What is the significance of biologic width?

If the restorative margin is placed into biologic width area there will be:

- Gingival inflammation
- Pocket formation
- Loss of crestal bone to re-establish the biologic width.

Q.5 What should be the least distance between the apical extension of restoration and crest of the alveolar bone?

Atleast 3 mm.

Q.6 What is Crown-lengthening surgery?

The surgical procedure to expose adequate clinical crown to prevent the placement of the crown margin into area of biologic width is called as Crown-lengthening surgery.

Q.7 What are the indications and contraindications of surgical Crown-lengthening procedure?

Indications:

(i) Subgingival caries or fracture
(ii) Inadequate clinical crown length for retention
(iii) Unequal / unesthetic gingival height

Contraindications:

(i) Surgery would create an unesthetic outcome
(ii) Deep caries or fracture would require excessive bone removal on contiguous teeth.

26 Miscellaneous

A. STERILIZATION

Q.1 What are the basic personal protective barrier equipments?

Basic personal barrier protection involves the use of face mask, protective eyewear, gloves and clinical gown.

Q.2 What is the sequence of wearing basic personal protective barrier equipments?

Preparation for the clinical care procedure begins with the positioning of the facemask, followed by the protective eyewear. Then the hands are washed / scrubbed prior to gloving.

Q.3 What is Bacteria filtration effectiveness of mask?

The mask with filtration level of 95-98% of 1-3 micron particles provides high-level protection.

Q.4 What is the duration of wear of a face mask?

Mask should be changed for each patient and not worn longer than 1 hour.

Q.5 Do's and Do not's while wearing facemask.

Do's

(i) Adjust the mask and position of eyewear before scrub / handwash
(ii) Should fit snugly with no gaps
(iii) Change mask each hour or more frequently when it becomes wet
(iv) Use fresh mask for each patient
(v) Grasp side elastic or tie strings to remove.

Do not's

(i) Do not wear mask only over mouth (but also on nose)
(ii) Never place the mask under chin
(iii) Never handle the outside of a contaminated mask with gloved / barehands
(iv) Mask should not be worn longer than 1 hour
(v) Should not leave the treatment area with the mask hanging around the neck.

Q.6 What are the various methods of handwashing?

Three methods:

(i) Short scrub
(ii) Short standard handwash
(iii) Surgical scrub.

Q.7 What are the various types of Gloves?

(i) Exam or procedure gloves: Available as
  (a) Sterile or non-sterile latex
  (b) Vinyl latex-free synthetic
(ii) Over gloves: These are thin vinyl or copolymer gloves placed over exam glove to prevent cross contamination, e.g. to retrieve additional supplies from a drawer, use a pen to make treatment notation or press button during x-ray taking.
(iii) Utility gloves: These are heavy gloves worn during handling of any chemicals or infectious waste; cleaning of contaminated surfaces instruments or materials and environmental surface cleaning and disinfection. Gloves made of nitrite rubber have an increased resistance to instrument punctures and can be autoclaved.
(iv) Dermal under gloves: These gloves are worn to reduce irritation from latex or non-latex.

Q.8 What is the procedure of wearing Gloves?

(i) Right hand grasps inside cuff surface of left glove
(ii) Left glove is pulled into place
(iii) Gloved fingers of left hand are inserted into cuff (outer surface) of right glove
(iv) Right glove is pulled into place
(v) Cuff of left glove is unfolded.

Q.9 What is the procedure of removal of Gloves?

(i) Use left fingers to pinch right glove near edge to fold back
(ii) Fold edge back without contact with clean inside surface
(iii) Use right fingers to contact outside of left glove at the wrist to invert and remove
(iv) Bunch glove into the palm
(v) With ungloved left hand, grasp inner noncontaminated portion of the right glove to peel it off, enclosing other glove as it is inverted.

B. CLINICAL MANAGEMENT OF MEDICALLY COMPROMISED PATIENTS

Q.1 What precautions should be taken during the treatment of a pregnant patient?

(i) Short appointments, served in series because patient fatigues easily.

(ii) Gentle lowering and straightening chair for patient because of genuine awkwardness due to new shape and weight gain.
(iii) Position the patient on her left side and not in supine or trendelenburg position because of discomfort of remaining in one position for long.
(iv) Advice non-alcoholic mouthwash and neutral sodium fluoride rinse.
(v) Advice not to brush right after vomiting to prevent erosion as nausea and vomiting is common in first trimester.
(vi) Recommend less strong flavored dentifrice because of adverse reaction to strong smells and flavor to the pregnant patient.
(vii) Recommend a small toothbrush; take care in instrumentation and radiographic film placement to prevent gagging.
(vii) Ideally, no medications should be prescribed because of toxic or teratogenic effects of therapy on the fetus.
(ix) Use of dental radiographs during pregnancy should be kept to a minimum. When they are required during pregnancy patient is covered with a lead apron, thyroid collar and a second apron for the back to prevent secondary radiations from reaching the abdomen.

Q.2 What should be the position of pregnant patient on dental chair during treatment?

Place the patient on left side or elevate the right hip 5-6 inches by placing pillow or blanket roll underneath. Supine position allows the weight of developing fetus to bear down directly on vena cava, aorta and major vessels. The reduction in return of cardiac blood supply may cause supine hypotensive syndrome with decreased placental perfusion.

Q.3 Clinical management of Breast feeding patient.

(i) Avoid giving tetracyclines, ciprofloxacin, metronidazole, aspirin, vancomycin, barbiturates and benzodiazepines to the breastfeeding patient.
(ii) The mother should take prescribed drugs just after breastfeeding and then avoid for 4 hours or more.

Q.4 Clinical management of menopause patient.

(i) Advice to use extra soft brush
(ii) Dentifrices with minimal abrasive particles should be used
(iii) Mouth rinses should have low alcohol concentration
(iv) Hormone replacement therapy (HRT) or estrogen replacement therapy (ERT) should be advised
(v) Root surfaces should be debrided gently with minimal soft tissue trauma.

(vi) If patient is osteoporotic, advice sodium fluoride, bisphosphonates, selective estrogen receptor modulators and parathyroid after consulting physician.

Q.5 Clinical management of Hypertensive patient.

(i) Stress free, calm and relaxing environment.
(ii) Short appointments.
(iii) Afternoon appointments.
(iv) Local anesthetic solution containing epinephrine concentration not greater than 1:100,000 should be used.
(v) Intraligamentary injection is generally contraindicated because haemodynamic changes are similar to intravascular injection.
(vi) An aspirating syringe should be used since epinephrine in the anesthetic solution may get into blood and may raise blood pressure and precipitate dysrthymias.
(vii) Epinephrine containing local anesthetic should be used cautiously and only in very small amounts in patient taking nonselective β blockers, with careful monitoring of vital signs.
(viii) Postural hypotension is common with patients on antihypertensive drug and can be minimized by slow positional changes in dental chair.
(ix) Gingival retraction cords containing epinephrine should be avoided.
(x) GA should be avoided whenever possible.
(xi) Aspirin may cause sodium and fluid retention and may be contraindicated in severe hypertension or cardiac failure.
(xii) Rofecoxib can increase the risk of myocardial infarction.

Q.6 What should be the concentration of Adrenaline in LA while treating hypertensive patients?

Adrenaline concentration should not be greater than 1:1,00,000.

Q.7 Clinical management of angina pectoris patient.

(i) Preoperative glyceryl trinitrate and sometimes oral sedation are advised.
(ii) Dental care should be carried out with minimal anxiety and oxygen saturation, blood pressure and pulse monitoring.
(iii) If a patient with a history of angina experiences chest pain during the dental surgery, dental treatment must be stopped; the patient should be given glyceryl trinitrate 0.3 – 0.6 mg sublingually and oxygen and be kept sitting upright.
(iv) If chest pain is not relieved within about 3 min, MI is a possible cause and medical help should be summoned.
(v) Pain that persists after 3 doses of nitroglycerin given every 5 min; that lasts more than 15-20 min; or that is associated with nausea, vomiting, syncope, or hypertension is highly suggestive of MI.

Q.8 After how much time period of myocardial infarction dental treatment can be done?

6 months

Q.9 Which are the microorganisms that are commonly found in periodontal pockets and are implicated as causing infective endocarditis?

(i) A.a
(ii) Eikenella corrodens
(iii) Capnocytophaga
(iv) Lactobacillus species

Q.10 In which patients antibiotic prophylaxis should be given before any dental treatment?

(i) Previous history of infective endocarditis
(ii) Prosthetic heart valves
(iii) Major congenital heart disease –
- Tetralogy of Fallot
- Transposition of great arteries
- Single ventricle states
- Surgically constructed systemic pulmonary shunts, or conduits

(iv) Mitral valve prolapse with valvular regurgitation.

Q.11 What is the preferable time gap between appointments for periodontal treatment in patients with infective endocarditis?

10 – 14 days or atleast 7 days

Q.12 What is the recommended antibiotic prophylaxis regimens for periodontal procedures in adults at risk for infective endocarditis?

	Regimen	*Antibiotic*	*Dosage*
1	Standard oral regimen	Amoxicillin	2 g 1 hr before procedure
2	Alternate regimen for patients allergic to amoxicillin, penicillin or both	Clindamycin or Azithromycin or Clarithromycin or Cephalexin or Cefadroxil	600 mg 1 hr before procedure 500 mg 1 hr before procedure 500 mg 1 hr before procedure 2 g 1 hr before procedure 2 g 1 hr before procedure
3	Patients unable to take oral medications	Ampicillin	2 g i/v or i/m within 30 min. before procedure
4	Patients unable to take oral medications and allergic to penicillin	Clindamycin or Cefazolin	600 mg i/v within 30 min. before procedure 1 g i/m or i/v within 30 min. before procedure

Q.13 What precautions should be taken while treating a diabetic patient?

(i) Take proper history
(ii) Consult patient's physician

(iii) Atraumatic procedures
(iv) It is best to give oral glucose just before the appointment.
(v) Drugs that can disturb diabetic control - aspirin and steroids must be avoided as these enhances the effect of oral hypoglycemic agents.

Q.14 What precautions should be taken while treating pulmonary diseased patient?

(i) Take proper history of the patient
(ii) Consult patient's physician regarding medications and status of pulmonary diseases
(iii) Stress free environment
(iv) Preferably afternoon appointment
(v) Position the patient in upright position
(vi) Avoid drugs causing respiratory depression – like GA, narcotics, sedatives
(vii) Avoid bilateral mandibular block anesthesia
(viii) Avoid ultrasonic instrumentation
(ix) For patients with history of asthma, inhaler should be available
(x) Periodontal procedure should not be done until emergency is there with active fungal / bacterial respiratory disease.

Q.15 What precautions should be taken while treating the renal diseases patient?

(i) Consult patient's physician.
(ii) Check blood pressure, blood urea nitrogen, serum creatinine, bleeding time, platelet count, partial thromboplastin time.
(iii) Avoid nephrotoxic drugs like – Phenacetin, tetracycline, aminoglycoside antibiotics.
(iv) Hemodialysis patient who has arteriovenous fistula, prophylactic antibiotic should be given.
(v) Patient on heparin anticoagulant, periodontal treatment should be done a day after dialysis, as the effect of heparin subside.
(vi) If arteriovenous fistula / shunt is present in the arm, blood pressure readings should be taken from the other arm. If A-V fistula is present in leg, patient is asked to avoid sitting with the leg dependent for longer than one hour.

Q.16 What precautions should be taken while treating the patient undergoing chemotherapy?

(i) Consult patient's physician.
(ii) The treatment should be conservative and palliative.
(iii) Periodontal therapy is best done the day before chemotherapy is given, as WBC count is relatively high on that day. It should be done when WBC count are above 2000 / mm^3 with an absolute granulocyte count of 1000 to 1500 / mm^3.

C. RECENT ADVANCEMENTS

Q.1 What is Minimally Invasive Surgery?

According to Harrel, minimally invasive surgery has been defined as the ability to perform a procedure through a substantially smaller surgical wound than had previously been necessary to accomplish the same surgical goals.

It includes:

(i) Using very small incision
(ii) Gentle handling
(iii) Retention of preoperative gingival architecture
(iv) Replacement or coronal positioning of gingival papilla.

Q.2 What is gene therapy?

It refers to the treatment of a disease by means of genetic manipulation. According to Strayer, gene therapy may involve:

(i) Supplying or increasing the expression of a mutant gene that is insufficiently expressed, e.g. to treat genetic enzymatic deficiencies.
(ii) Blocking a gene that is detrimental, e.g. using antisense constructs to inhibit tumor proliferation.
(iii) Adding a foreign gene to treat a situation beyond the capability of a normal genome, e.g. introduce an enzyme into a cell or tissue that allows the tissue to become more sensitive to the effects of a pharmacologic agent.

Q.3 What is Photodynamic therapy?

It involves the use of light activated drugs to kill periodontal pathogens. Toluidine blue when activated by LASER light, can be used to kill periodontal pathogens when instilled within periodontal pocket.

Q.4 What is evidence based dentistry?

It is defined as the integrating individual clinical expertise with the best available external clinical evidence from systemic research.

Q.5 What are biologic modifiers?

These are the materials, proteins and factors that have the potential to alter the host tissue so as to stimulate or regulate the wound healing process.

(i) These agents can act through a systemic route, e.g. hormones
(ii) Can also act at local sites, e.g. growth factors, polypeptide cytokines.

Q.6 What is growth factors?

It is used to denote a class of naturally occurring protein that function in the body to promote the mitogenesis (proliferation), directed migration and metabolic activity of cells.

Q.7 What are the various mode of action of growth factors?

(i) Paracrine: It involves the production of a factor by one cell, with receptors present on another cell in local microenvironment, e.g PDGF, TGF-β.

(ii) Autocrine: Those factors that are synthesized by one cell, secreted in a soluble form outside the cell then binds to surface receptors on the same cell to evoke an effect, e.g TGF-α, BMPs.

(iii) Juxtacrine: The factor produced by the cell of origin is cell surface bound and requires cell contact by target cell to evoke a response, e.g. stem cell factor.

(iv) Intracrine: A factor is produced by one cell and not secreted but acts intracellularly to facilitate its effect, e.g. PTHrP.

Q.8 Name various Growth factors.

(i) Platelet derived growth factor (PDGF -AA, AB, BB)
(ii) Insulin like growth factor (IGF – I, IGF – II)
(iii) Transforming growth factor (TGF-α, TGF-β)
(iv) Fibroblasts growth factor (FGF acidic, FGF basic)
(v) Bone morphogenetic protein (BMP 1 to BMP 15)
(vi) Colony stimulating factors (G, GM and M)
(vii) Parathyroid hormone-related protein (PTHrP)
(viii) Epidermal growth factor

Q.9 What is Nanotechnology?

The science of bioengineering at the molecular level to produce materials of hitherto unknown and unthought properties which will pave the way of future periodontal regeneration and drug delivery in periodontics.

Periodontal microsurgery

Q.1 What is Periodontal microsurgery?

It is defined as refinements in existing basic surgical techniques that are made possible by the use of the surgical microscope and subsequent improved visual acuity.

Q.2 In which periodontal procedure, periodontal microsurgery is applied?

(i) Tissue grafting procedure to correct gingival recession–
  (a) Free epithelial grafting
  (b) Subepithelial connective tissue grafting
(ii) Papilla reconstruction procedure.

Q.3 Why in microsurgery there is great reduction in surgical damage to the tissues?

(i) Excellent visualization of the operative field through microscope.
(ii) Atraumatic surgical approach because of smaller surgical field with lesser injury and bleeding.
(iii) Dexterity of the surgeon.

Q.4 How are microsurgical periodontal instruments different from other periodontal instruments?

(i) Microsurgical instruments are much smaller, often by tenfold.
(ii) Their handles have a round cross-sectional diameter to enhance rotary movements using the precision grip.
(iii) They are made of titanium to reduce weight, prevent magnetization and provide reliable manipulation of needles, sutures and tissues.
(iv) Microsurgical instruments are manufactured under magnification to high tolerance and resist deformation from repetitive use and sterilization cycles.
(v) Opthalamic scalpel and blades are used. Castroviejo microsurgical scalpel and Laschal microscissor - small beak scissor are used.

Q.5 What are the advantages of microsurgery?

(i) Improved cosmetics
(ii) Rapid healing
(iii) Minimal discomfort
(iv) Less invasive
(v) Reduces surgical fatigue and development of spinal and occupational pathology of operator
(vi) Enhanced patient acceptance.

Q.6 How is microsurgical suturing different from conventional suturing technique?

(i) 7- 0 to 9- 0 microsutures are used.
(ii) Needle angle of entry and exit is slightly less than 90°, sutures pass across the incision line at oblique / acute angles rather than perpendicularly.
(iii) Bite size – 1.5 times the tissue thickness.
(iv) Direction of needle passage – perpendicular to the wound.

D. DISCOVERIES IN PERIODONTICS

1.	1535	Paracelsus	Introduced the term tartar and developed doctrine of calculus
2.	1815	Levi Spear Parmly	Invented dental floss
3.	1823	Alphonse Toirac	Gave the term Pyorrhea alveolaris
4.	1844	Gunnel	First reported Pericoronitis

5.	1846	William Sharpey	Described Sharpey's fibers
6.	1868	Paul Langerhan	Described Langerhan cell
7.	1872	Silas Noble and JP Cooley	Patented the first toothpick manufacturing machine
8.	1875	Friedrich Sigmund	Described merkel cells
9.	1877	Wilkerson	First hydraulic dental chair
10.	1882	Metchnikoff	Discovered mechanism of phagocytosis
11.	1884	Robiecsek	Gingivectomy with straight incision
12.	1885	Malassez	First described epithelial rest of Malassez cell
13.	1897	Vincentini	Described Corncob structure of plaque
14.	1898	GV Black	First used the term plaque in dental context
15.	1899	Talbot	Intially proposed Bass method of toothbrushing
16.	1914	Grace Rogers Splading and Gillette Hayden	Founded American Academy of Periodontology
17.	1918	Leonard Widman	Described original Widman flap procedure
18.	1918	Zentler	Described gingivectomy with scalloped incision
19.	1923	Hegedus	Bone Graft for reconstruction of bone defects produced by periodontal disease
20.	1923	Dr Abraham Wesley Ward	Introduced Periodontal dressing Wondr Pak
21.	1923	Gottlieb	Introduced the term diffuse atrophy of alveolar bone
22.	1928	Gottlieb	Introduced the term deep cementopathia
23.	1931	Kirkland	Described modified flap operation
24.	1932	Prinz	Coined the term chronic desquamative gingivitis
25.	1932	Paul R Stillman	Described Stillman tooth brushing technique
26.	1934	Alfred Fones	Described circular tooth brushing technique known as Fones method
27.	1935	William J Charters	Described Charters toothbrushing method
28.	1938	Dupont	Created nylon toothbrush bristle
29.	1938	Wannenmacher	Introduced the term Periodontitis marginalis progressive
30.	1940	Thoma and Goldman	Introduced the term Paradontosis
31.	1942	Orban and Weinmann	Introduced the term Periodontosis
32.	1950	Nathan Friedman	Gave the term Mucogingival surgery

33. 1951	Goldman	Introduced the term Gingivoplasty
34. 1954	Nabers	Developed Apically repositioned flap
35. 1955	Friedman	Introduced the term Osteoplasty
36. 1956	Grupe and Warren	Originally described Laterally displaced flap
37. 1956	Russell	Developed Periodontal index
38. 1957	Ariaudo and Tyrrell	Later modified apically repositioned flap
39. 1959	Ramjford	Introduced Periodontal disease index
40. 1959	Cohen	First described Col
41. 1960	Green and Vermillion	Developed Oral hygiene index
42. 1961	Garguilo	Found biologic width to be 2.04 mm
43. 1962	Friedman	Proposed the term Apically repositioned flap
44. 1962	Schroder	Described antiplaque property of chlorhexidine
45. 1962	Gross Lapiere	Discovered MMPs in the tail of metamorphosing tadpole
46. 1963	Bjorn	Intially described free gingival autograft
47. 1963	Brannstrom	Gave Hydrodynamic theory of Dentin hypersensitivity
48. 1964	Silness and Loe	Developed Plaque index
49. 1964	Green and Vermillion	Gave Simplified oral hygiene index (OHI-S)
50. 1964	Simring and Goldberg	First described relationship b / w periodontal and pulpal disease
51. 1965	Ewen	Introduced bone swaging as autogenous bone grafting
52. 1966	Nabers	Introduced gingival grafts
53. 1967	Marshall Urist	Discovered bone morphogenetic protein (BMP)
54. 1967	Chaput et al	Introduced the term Juvenile periodontitis
55. 1968	Cohen and Ross	First described Double papilla procedure
56. 1968	Podshadley and Haley	Developed Patient hygiene performance (PHP) index
57. 1969	Robinson	Devised Osseous coagulum technique as autogenous bone grafting
58. 1969	L Hench	Invented Bioactive glass
59. 1971	Jones	Coined the term Corncob structure of plaque
60. 1971	Muhlemann and Son	Developed Gingival sulcus index (SBI)

61. 1972	Diem et al	Described Bone Blend as autogenous bone grafting technique
62. 1972	O'Leary, Drake and Naylor	Developed Plaque control record
63. 1974	Ramjford and Nissle	Modified Widman Flap procedure
64. 1974	Ingber	Gave the concept of forced orthodontic eruption for treatment of one wall and two wall bony pockets
65. 1975	Bernimoulin	Gave two step procedure free gingival graft followed by coronally positioned flap
66. 1975	Ainamo and Bay	Developed Gingival bleeding index (GBI)
67. 1978	Dr Paul Keyes	Gave Keyes technique / Keyes salt-out technique
68. 1979	Max Goodson et al	Developed controlled intrapocket antimicrobial drug deliveries
69. 1979	Maynard and Wilson	Coined the term marginal tissue recession
70. 1979	Jan Lindhe et al	First introduced the concept of host modulation therapy
71. 1982	Nyman et al	Pioneered GTR technique
72. 1985	Langer and Langer	Described Subepithelial connective tissue graft
73. 1985	Takei	Gave Papilla Preservation technique
74. 1986	Gottlow	Coined the term GTR
75. 1986	Tarnow	Described semilunar coronally positioned flap
76. 1987	Nelson	Described subpedicle connective tissue graft
77. 1993	Miller	Proposed Periodontal plastic surgery
78. 1999	Prosser	Discovered Quorum sensing in biofilms
79.	McCall and Box	Introduced the term Periodontitis
80.	WH Hanford and CO Patten	Invented periodontal probe
81.	Gibbon and Nygaard	Discovered Interspecies coaggregation of plaque
82.	PiniPrato	Described GTR for root coverage in recession
83.	Edel	Originally described free connective tissue autograft
84.	Klingsberg	Reported the application of Sclera as nonbone graft material
85.	Hammar-strom	Emdogain EMD

86.	Kwan and Lekovic	Described periosteum as a GTR material
87.	Rateitschak	Described Accordion technique of free gingival autografts
88.	Han and associates	Developed Strip technique of free gingival autografts
89.	Friedman	Described Beveled flap
90.	Edlan and Mejchar	Described Vestibular extension technique
91.	David Sackett	Gave the term Evidence based dentistry
92.	Scandivian group	Jens Waerhaug and coworker
93.	Michnigan Group	Ramjford and coworker
94.	Gothenburg Group	Lindhe and coworker

E. FATHERS

1. Father of Medicine - Hippocrates
2. Father of Dentistry - Pierre Fauchard
3. Father of Oral hygiene - Levi Spear Parmly
4. Father of Non surgical periodontal therapy - Isadore Hirschfeld
5. Father of Periodontal Plastic surgery - Dr Preston D Miller
6. First Periodontist in the history - John W Riggs

F. EDITORS

1. Founder of the Journal of Dental Research - William G Gies 1919
2. Former Editor-in-chief – Journal of Periodontology - Robert J Genco
3. Editor-in-chief – Journal of Periodontology - Kenneth S Kornman
4. Editor-in-chief – Journal of Periodontal Research -Isao Ishikawa, Jorgen Slots, Maurizio Tonetti
5. Editor-in-chief – Journal of Clinical Periodontology - Jan Lindhe
6. Editor-in-chief – Journal of Periodontology 2000 - Jorgen Slots
7. Editor-in-chief – International Journal of Periodontics -Myron Nevins and Restorative Dentistry

G. WORDS WITH PREFIX "PERIO" USED IN PERIODONTICS

1. **Perio-Aid:** It is a toothpick holder, which is one of the most effective aids available for cleaning exposed furcation after periodontal therapy.
2. **Perioalert:** Immunoassay to detect serum antibodies to specific bacterial pathogens, monocytes response to LPS and peripheral neutrophil function. Site of sample is peripheral blood.

3. **Periocare:** It is a zinc oxide non-eugenol periodontal dressing available in the form of paste-gel and setting occurs by chemical reaction. Paste consists of zinc oxide, calcium hydroxide, magnesium oxide and vegetable oils; Gel consists of resins, fatty acid, ethyl cellulose, lanolin and calcium hydroxide.
4. **Periocheck:** It is rapid chair side test kit developed to detect neutral proteases in GCF.
5. **Periochip:** Periochip is a small, pale orange chip of baby's thumb nail 4 mm × 5 mm × 350 μm size, weighing 7.4 mg. The prescription chip contains 2.5 mg of Chlorhexidine gluconate, of a biodegradable hydrolyzed gelatin matrix, cross linked with glutaraldehyde and also containing glycerin and water.
6. **PerioCline:** It is subgingival delivery system of 2% Minocycline hydrochloride in syringable gel suspension formulation.
7. **Periodex:** 0.12% Chlorhexidine gluconate.
8. **Periodontometer:** Instrument used for detecting tooth mobility.
9. **Periogard:** Rapid chair side test kit for Aspartate aminotransferase (AST). GCF collected is placed in Tromethamine hydrochloride buffer is allowed to react with mixture of L-aspartic and α-Ketoglutaric acids for 10 minutes. If AST is present the aspartate and α-glutarate are catalyzed to oxalacetate and glutamate.
10. **PerioGard:** 0.12% Chlorhexidine gluconate.
11. **PerioGlass:** Bioactive glass alloplast consisting of sodium and calcium salts, phosphates, silicon dioxide of irregular particles of 90-170 μm.
12. **Periograft:** Nonporous hydroxyapatite alloplast.
13. **Periopac:** It is premixed zinc oxide non-eugenol dressings. It contains calcium phosphate, zinc oxide, acrylate, organic solvents, flavoring and coloring agents, when this material is exposed to air or moisture, it sets by the loss of organic solvents.
14. **Periopaper:** It is blotter on which GCF is collected.
15. **PerioPik:** Tip used for subgingival irrigation. It is a rigid metal cannula inserted into the pocket to release irrigant for subgingival irrigation performed by an oral health professional before scaling, simultaneously with scaling or directly after scaling.
16. **Perioplaner/Periopolisher:** Powered devices for removal of plaque and calculus with reciprocating motion.
17. **Perio-probe:** Electronic probe with a tip diameter of 0.5 mm and uses standardized probing force of 0.3 - 0.4 N.

18. **Perioscan:** It is chair side diagnostic kit using BANA reaction to identify *Tannerella forsythia, P.gingivalis, Treponema denticola* and *Capnocytophaga species*.
19. **Perioscopy:** It consists of 0.99 mm diameter reusable fiber optic endoscope over, which is, fitted a disposable sterile sheath. The new fiber optic endoscope instrument fits into specially designed periodontal explorers with 24-46 power magnification and fiber optic illumination, this device allows clear visualization into deep subgingival pockets and in furcations.
20. **Periostat:** It is subantimicrobial dose of Doxycycline hyclate capsule of 20 mg prescribed for patients with chronic periodontitis twice daily.
21. **PerioTemp:** It is probe, which detects pocket temperature differences of 0.1°C from a referenced subgingival temperature. It consists of copper-nickel thermocouple connected to a digital thermometer attached to metal probe.
22. **Periotest:** It is device for determining tooth mobility by measuring the reaction of the periodontium to a defined percussion force which is applied to the tooth and delivered by a tapping instrument. Periotest scale ranges from – 8 to + 50:
    - – 8 to 9 —— Clinically firm teeth
    - 10 to 19 —— First distinguishable sign of movement
    - 20 to 29 —— Crown deviates within 1mm of its normal position
    - 30 to 50 —— Mobility is readily observed
23. **PERIO-TOR:** These are the instrument tips for scaling and root planing causing minimal removal of tooth structures.
24. **Periotriever:** Highly magnetized instruments designed for retrieval of broken instrument tips from the periodontal pocket.
25. **Periotron:** It is the electronic machine used for measuring the amount of fluid or GCF collected on filter paper.
26. **Perio 2000 System:** Diamond probe is a recently developed instrument, which combines the features of a periodontal probe with the Silver sulfide sensor for detection of volatile sulphur compounds.
27. **Periodontology:** The science that deals with the structures and behavior of the periodontium in health and disease.
28. **Periodontics :** The branch of dentistry concerned with prevention and treatment of periodontal diseases.