TATYASAHEB KORE DENTAL COLLEGE AND RESEARCH CENTRE
NEW PARGAON – 416 113

Tal.: Hatkanangale Dist.:Kolhapur (Maharashtra State)

National Dental Commission

INFORMATION REGARDING INSTITUTIONAL COMPLIANCE

4. Clinical Compliance

4.2 Student clinical work registers are updated regularly.

'SERVICE IS RELIGION'

WARANA
HEALTH MOVEMENT
Est : 1 9 9 2

TATYASAHEB KORE DENTAL COLLEGE & RESEARCH CENTRE, NEW PARGAON

DEPARTMENT OF PEDODONTICS & PREVENTIVE DENTISTRY

Certified that this is a bonafide Record of work done by

~~Mr./~~Miss Kesare Sakshi Rajesh

Roll No. 22

University No. 159214

During the year 2025-26

Staff In-charge

HOD / In-charge

HOD
Department of Pedodontics & Preventive Dentistry
Tatyasaheb Kore Dental College & Research Centre
New Pargaon

Examiners

1) Internal Examiner:-
Dr. Mukul Joshi
10/2/26

2) External Examiner:-
Dr. Shubhendra Khandewale

TATYASAHEB KORE DENTAL COLLEGE & RESEARCH CENTRE
New Pargaon
Tal. Hatkanangale
Dist. Kolhapur

Dr. Harish Kulkarni M.D.S
Principal
T. K. D. C. & Research Centre
New Pargaon, Tal. Hatkanangle
Dt. Kolhapur 416 13

Department of Pedodontist & Preventive Dentistry

RULES AND REGULATIONS

1) Students should maintain strict descipine in the Department.

2) Studets should report in time to the Department in Clean apron with Name plate and Record Book.

3) Dress Code is followed very strictly in the Department and students Should not wear Jeans, T-Shirts, Sports Shoes and Sandles during College Hours.

4) Regulations laid down by MUHS Nashik will be followed regarding attendance.

5) Absence without permission will be considered seriously.

6) Assignments should be completed on time and signature of the incharge Staff Member obtained before the completion of the Clinical Posting.

7) Posting end Examination shall be conducted at the end of each posting (Both Clinical & Viva - Voce)

8) Students must complete the stipulated Quota of Clinical work Compulsorily, if not they will not be permitted to appeat for University Examination.

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WORK SCHEDULE

For IIInd Year

- Case History - Five
- Restorations
  (Silver Amalgam, GIC, Composite) - Twenty
- Extraction - Five
- Oral Prophylaxis - Ten
- Topical fluride application - Five
- Clinical Discussion on
  1. Sterilization & Disinfection in the Dental Clinic
  2. Case - History
  3. Analgesics & Antibiotics
  4. Morphology & Histology of Deciduous & permanent Teeth
  5. Restorative Dentistry
  6. Local Anaesthesia & Techniques
  7. Extraction Techniques

For IV th Year

- Case History - Ten
- Restorations - Twenty Five
- Extraction - Twenty
- Oral Prophylaxis - Ten
- Topical fluride application - Ten
- Space maintainers - Two
- Education & motivation of patients - Thirty
- Clinical Discussion
  1. Behaviour Management
  2. Pulp Therapy & Endodontics
  3. Space Management
  4. Oral Habits
  5. Traumatic injuries to teeth
  6. Flurides

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LIST OF INSTRUMENTS REQUIRED FOR CLINICAL POSTING

Two sets each of

- Mouth Mirror
- Straight Probe
- Explorer
- Small Spoon Excavator
- Tweezer
- Diamond Carver
- Hallenbeck Carver
- Amalgam Carrier
- Parallelogram Condensor
- Round Condenser
- Plastic filling Instrument
- Burnisher Cement Spatula
- Agate Spatula
- Dappen Dish
- Instrument pouch

Matrices & Retainers (No. 9 & 1)

Cotton Holder

Waste Receiver

Stainless Steel Kidney Trays (one large & one small)

Glass Slab

Green cloth

Gloves

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BOOKS RECOMMNDED & REFERENCE

1) Pediatric Dentistry (Infancy through Adolescences)- Pinkham.
2) Kennedy's Pediatric Operative Dentistry - Kennedy & Curzon
3) Occlesal guidance in Pediatric Dentistry - Stephen H. Wei
4) Clinical Use of Fluorides - Stephen H. Wei
5) Pediatric Oral & Maxillofacial Surgery - Kaban.
6) Pediatric Medical Emergencies - P. S. Whatt.
7) Understanding of Dental Caries - Niki Foruk.
8) An Atlad of Glass Ionomer cements - G. J. Mount
9) Clinical Pedodontics - Finn.
10) Textbook of Pediatric Dentistry - Braham Morris.
11) Primary Preventive Dentistry - Norman O. Harris
12) Handbook of Clinical Pedodontics - Kenneth D.
13) Preventive Dentistry - Forrester
14) The metabolism and Toxicity of Fluoride - Garry M. whitford
15) Dentistry for the Child and Adolescence - Mc. Donald
16) Pediatric Dentistry - Damale S. G.
17) Behaviors Management - Wright
18) Pediatric Dentistry - Mathews on
19) Traumatic Injuries - and reason
20) Occlusal guidance in Pediatric Dentistry - Nalata
21) Pediatric Drug Therapy - Tomare
22) Contemporary Orthodontics - Profitt.
23) Preventive Dentistry - Depaola
24) Metabolism & Toxicity of Fluoride - whitford G. M.
25) Endontic Practice - Grossman.
26) Principles of Endontics - Munford.
27) Endodontics - Ingle.
28) Pathways of Pulp - Cohen.
29) Management of traumatized anterior Teeth - Hargreaves.

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CASE HISTORY DETAILS
DEPARTMENT OF PEDODONTICS

Personal Information

Date _____

Patients Name : _____

Registration No. : Age / Sex : Weight :

Date and place of Birth : _____

Religion : _____ Education : _____

Name of person accompanying : _____

with whom does the child live : _____

Occupation of mother & father : _____

Residential address : _____

Contact No. : _____

■ **Chief Complaint :**

Patients complaint in his / her own words

■ **History of present illness :**

1. Pain :

- Nature of pain.
- Spontaneous / Constant / Periodic
- Onset, Duration, Progress

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- Aggravating factors / What causes pain
- Relieving factors - Medications taken ?
- Treatment done - Pain regressed / not aggravated after treatment
- Nocturnal pain - present / absent
- Pain associated with swelling / fever ?

2. Swelling :

- Time of appearance - before pain / after pain ?
- Complete history of swelling including -
  - I / o or E / o swelling,
  - Location, extent, size
  - Treatment done - medications taken ?
  - Swelling regressed / not / increased
  - H / o Hot fermentation ?
  - Swelling associated with pain / fever ?

3. Fever :

- Time of appearance - before pain / after pain ?
- Treatment done - Medications taken ?
- Fever continued / not / increased ?
- Fever associated with pain / swelling ?

4. Trauma :

- Complete medical history
- Neurologic assessment -
  - H / o episode of unconsciousness / amnesia
  - after trauma
  - Other signs - nausea, vomiting, drowsiness,

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- blurred vision
  - H/o previous injury to same area
  - Ask for any bite disturbance

Pre-natal History:

- H/o Illness during pregnancy -
- H/o specific medicines during pregnancy-
- H/o Fluoride supplements during pregnancy -
- H/o high Vitamin/ Calcium supplements -

Natal History:

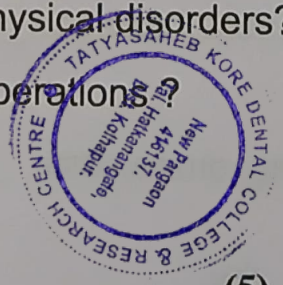
- Type of delivery: Term :
- H/o Birth asphyxia, Jaundice, Blood transfusions -

Post-natal History:

- Developmental milestones:
- H/o Immunization:

Past Medical History:

- Allergic to any food, LA, penicillin or other drugs?
- H/o vitamins, calcium, iron supplements given?
- H/o Major Illnesses during childhood?
- H/o asthama, seizures during childhood?
- Any mental or physical disorders?
- H/o any major operations ?



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In case of Trauma, particular attention given to Drug sensitivity.
Coagulation disorders, Tetanus coverage

- **Family History:**
  H/o Hereditary diseases running in families like haemophilia, D.M., hypertension

- **Past Dental History:**
  Details about Patients first visit to dentist?
  Details about dental treatment done in past

- **Personal Habits:**
  Oral Hygiene methods:
  Method of cleaning teeth: Frequency & Duration:
  Time of cleaning teeth:
  Assisted/ Unassisted:
  Use of other oral hygiene aids:

- **Dietary habits:**
  Source of water:
  Type of diet:
  H/ o bottle feeding in detail:
  Introduction of solid foods:

Details about daily dietary habits:

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Time	Item
8am	breakfast (tea and chapatti)
_____	_____
_____	_____

Details about In between meal snacking:

Frequency:

Time of intake:

Sugar in solid form:

Sugar is solution form:

Difficulty in chewing and swallowing?

Local examination

a) Extra oral Examination:

1) Shape of Head:

Dolichocephalic / mesocephalic / brachycephalic / hyper brachycephalic

2) Facial Form:

Mesoprosopic / euryprosopic / leptoprosopic

3) Face Symmetry: Facial Profile convex/concave /straight

4) TMJ Examination:

Method of examination

Range of movement

Path of closure

Tenderness

Sounds

Locking

5) Lymph nodes examination:

Groups of lymph nodes

Method of palpation

Tenderness,

Consistency - hard / matted / soft

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6) Speech:

7) Lip competency: competent / incompetent / potentially competent

) **Intra oral Examination:**

Swallowing pattern: infantile swallow / mature swallow,

Adverse oral habits:
Thumb sucking
Tongue thrusting
Nail biting
Mouth breathing
Bruxism

Frequency :
Duration :
Intensity

. **Soft tissue Examination:**

- Tongue
- Labial mucosa
- Palate
- Floor of mouth
- Alveolar mucosa - color of pigmentation
- Lip
- Gingiva - presence of draining sinus / healing sinus /l/o swelling
  Fistula / parulis

Labial, Lingual, Buccal Freenal attachment-type

. **Hard tissue Examination:**

Molar relationship: Permanent - Angles CI/CII/CIII malocclusion
Deciduous - Mesial step / Distal step /
Flush terminal plane

Canine relationship : CI/CII/CIII

Incisor relationship : Overjet - mm Overbite - mm

Caries Index: dmft / deft

Type of denotation :

- Teeth present :
- Pit & Fissure caries :
- Smooth surface caries :
- Deep caries:
- Mobility of teeth: Grade I/II/III

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Bleeding on probing (if pulp chamber is involved)
Any other anomaly please specify:
Stains +/++/+++ Calculus: +/++/+++

Dental age:

Provisional diagnosis:

Example - Chronic dentoalveolar abscess
Acute reversible pulpitis
Chronic irreversible pulpitis associated with draining sinus
Pulp necrosis
Chronic hyperplastic pulpitis

Differential Diagnosis:

Periapical cyst/ periapical granuloma

Investigations:

IOPA

- Radiographic Interpretation
  IOPA side,Teeth seen(deciduous and permanent),
  Radiolucency in enamel/ dentin/ approaching pulp/involving pulp.
  Radiolucency in furcation area / periapical area,
  Resorption status of roots

- Other investigations:
- Occlusal / bitewing radiograph / OPG / Lateral cephalogram
- Space analysis
- Pulp vitality tests
- Caries activity tests
- Blood examinations
- Urine examinations
- Advance diagnosis
- Biopsy

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Final Diagnosis:

Example - Acute reversible pulpitis
Chronic irreversible pulpitis
Chronic hyperplastic pulpitis

Treatment plan:

Informed Consent:

Treatment Recommendations:

- Nature of procedures/materials used
- Nos. of appointments / timeframe required for treatment
- Behavioural management techniques required

I Emergency phase:

- Incision & Drainage - E/O swelling cases
- Emegency access opening
- Antibiotics & analgesics
- Antibiotic prophylaxis to avoid bacteremia in congenital heart defects/
- pacemakers patients

II Planned Phase:

A) Preventive phase:

- Oral hygiene counselling
- Diet counselling
- Stopage of habits if any
- Oral prophylaxis
- Topical fluoride application-mention the material used
- P & F sealants

B) Restorative Phase:

- Restorative material to be used should be mentioned specifically
SSC

C) Endodontic Phase:

- Vital pulp therapy / Pulpectomy/Apexogenesis / Apexification

D) Surgical Phase:

- Extraction/minor surgical procedures - excision of mucocele / ranula/ frenectomy/ Apiocectomy

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E) Orthodontic Phase:

- *Space maintainers / space regainers / Removable orthodontic treatment-correction of single tooth crossbite /*

  Fixed orthodontic treatment/ Habit Breaking Appliances

F) Maintainance Phase:

Periodic Recall for all treatments should be mentioned seperately:

Recall after 1 week to check healing of extraction socket
Recall after 6 months for biannual topical fluoride application
Recall after 6 months to check for

- oral hygiene & diet maintenance
- integrity of previous restorations
- detection new carious lesions

- Children who exhibit higher risk of developing caries recalled every 3-4 months

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2.	Morphological diagnosis both primary & permanent dentition & its clinical significance.	6-11	
3.	Chronology of eruption age	10+15	
4.	Eruption & shedding of teeth Theories of eruption	15-17	
5.	Tooth numbering system	18-21	
6.	Development of dentition	22-28	
7.	Development of occlusion	29-39	
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14.	Atraumatic restorative [illegible]	65-68	
15.	Non-pharmacological behaviour management	69-72	

SCOPE OF PEDIATRIC DENTISTRY

Pediatric Dentistry :-

Pediatric dentistry is an age defined speciality that provides both primary & comprehensive, preventive & therapeutic oral health care of infants, children through adolescence including those with special health care needs.

- (AAPD) American Academy of Pediatric dentistry - 1999

- Pedo is a Greek word pais meaning child & dontics is the study of teeth.

- Importance of primary teeth :-

1] Maintain good nutrition by permitting child to chew properly.
2] Involved in speech development.
3] Helps in the eruption of permanent teeth by saving space.
4] A Healthy smile can help children feel good about the way they look to others.

New born - 1 to 28 day
Infants - 1st month to 12 months
children - 1st year to 10 years
Adolescent - 10 year to 19 years
Age of toddler - 1 to 3 years

Father of dentistry - Pierre Fauchard
Father of dentistry in India - Dr. Raffuddin Ahmed (1945)
Father of Pedodontics - Robert Bunan (French dentist)
↳ In India - Dr. B. R. Vacher.

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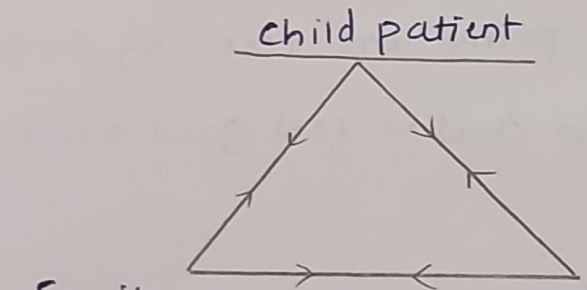
- Aims & Objectives of Pediatric dentistry :-

1] Health of a child as a whole.
2] More specifically concerned with oral health.
3] Early diagnosis & prompt treatment.
4] Restoring the mouth to good oral health.
5] To observe & control the necessary developing dentition of child patient.
6] Relief of pain.
7] Increase the knowledge.
8] Insites a positive attitude & behaviour.
9] Restore the lost tooth structure.
10] Management of special patient.

- Pedodontic Triangle :-

1] Conventional Model - By Wright (1975) :-

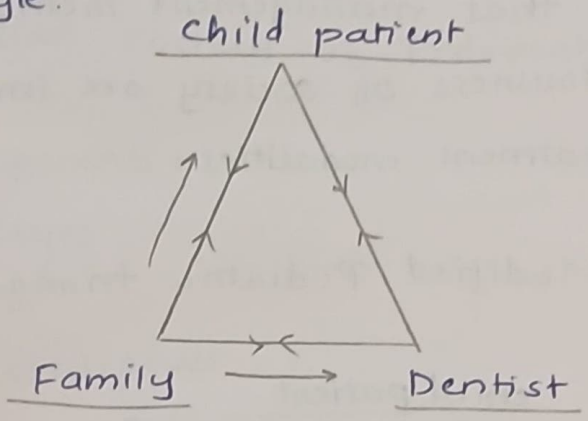


- Patient - doctor relation in adult is linear, but in Pedodont the relation is triangular.
- This is because, in Pedodontics, the parents & the child b are involved & child is at the apex of triangle as he is the focus of attention.

2] Variations :-

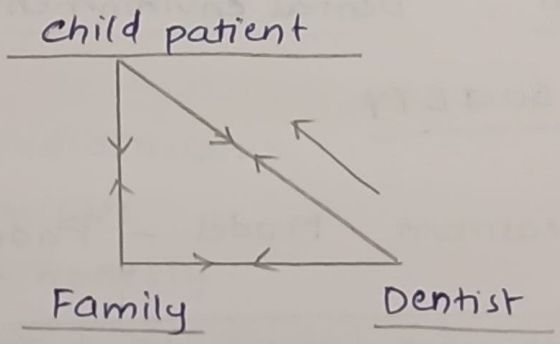
A] Isoceles Triangle :-

- An authoritive or over indulgent parent always tries to interfere in the conversation between the dentist & the child by answering on behalf of the child.
- As a consequence, there is more interaction between the parent & the dentist hence the equilateral triangle is replaud by Isoceles triangle.



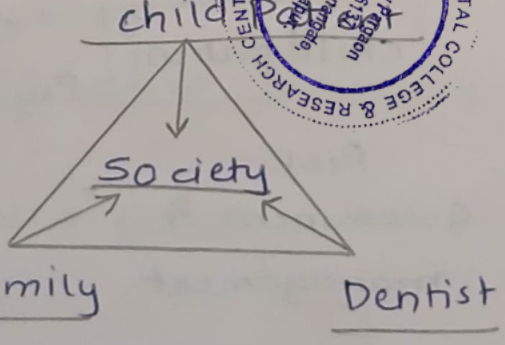
B] Right - angled Triangle:-

- If the parent is negligent, the conversation between the parent & the dentist may not be reciprocal effectively; Hence right-angled triangular replaces the normal equilateral Δle.



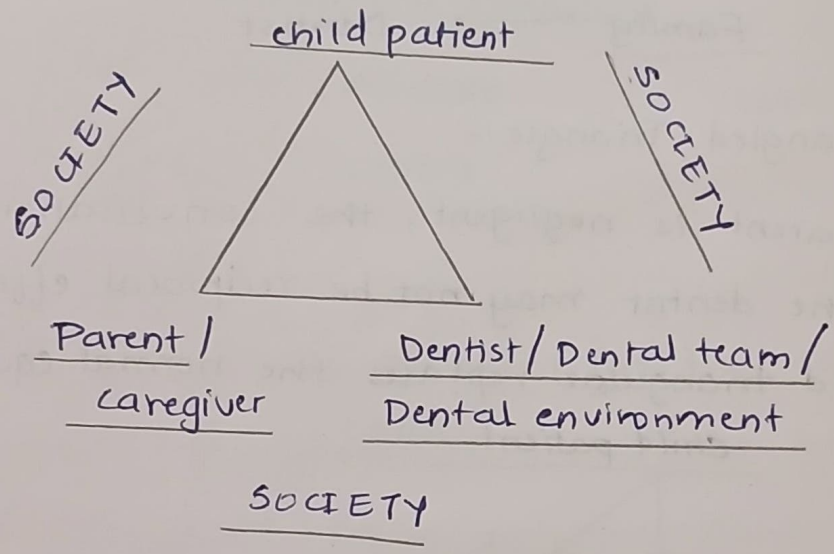
3] Modifications:-

A] Mc Donald - 2004 → Pedigm triangle

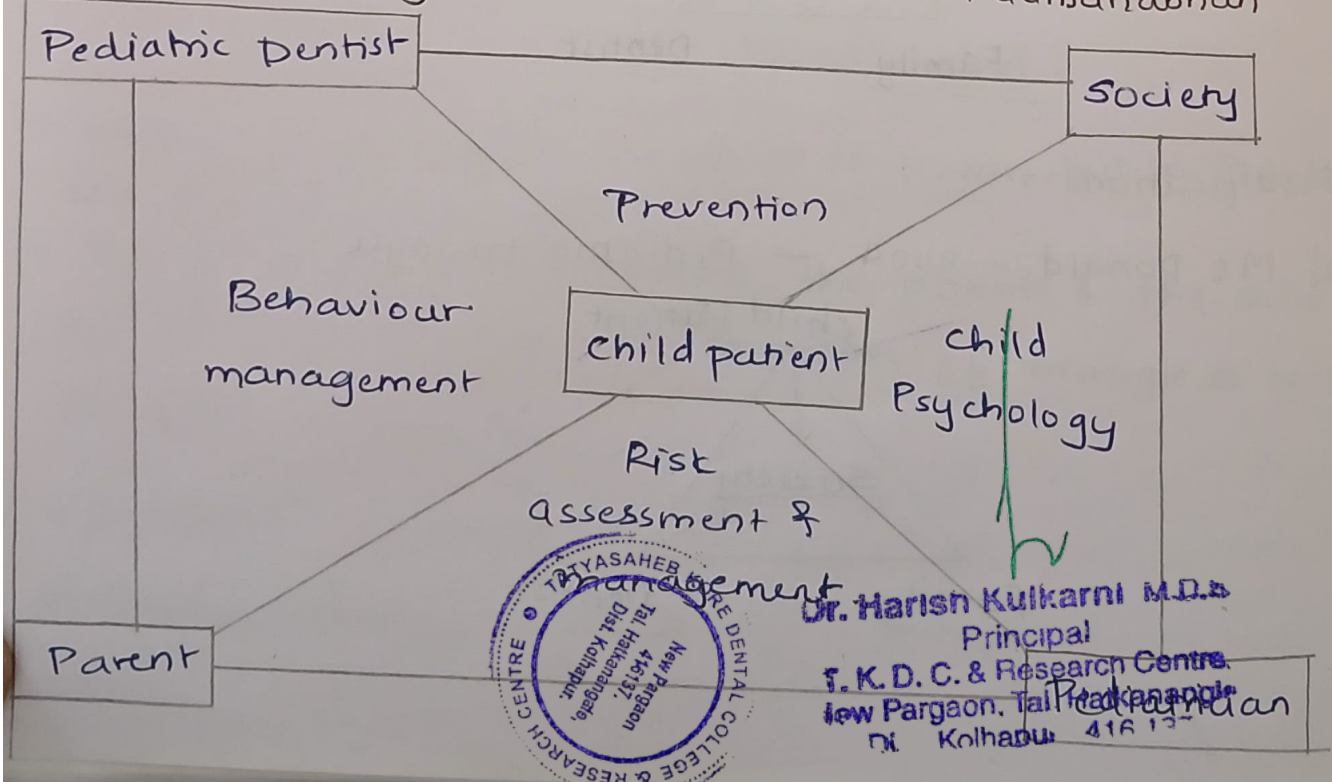


- As community has become a major part of all compon of environment, therefore recently a new parameter ha also been added, i.e society.
- This depiction looked complete with the fact that the comm nication is reciprocal & society came into the centre of the triangle indicating that management methods acceptable n society & the litigiousness of society are important factors influencing the treatment modalities.

B] Wright - 2014 → Modified Pediatric triangle :-



Pediatric Dentistry Treatment Model - Padmanabhan



- It presents the former triangle as a square which has pediatric dentist, pediatrician, Family & society playing important roles & definitely the child patient is the centre of attention.
- Based on four principles → prevention, risk assessment & management, child psychology & behaviour management.

ISPPD :- Indian Society of Pedodontics & Preventive Dentistry (1979)

- Has 5 components -

1] Triad of Keyes
2] Triangles
3] Staff of Aesculapius
4] Serpents
5] Wings

Branches – Two

1] 3 Major
2] 6 Minor

- Attitude of Pediatrician:-
  - Ethical principles
  - Professional honesty.
  - TLt - irrespective of social, economic status.
  - Share knowledge
  - Adopt new techniqu
  - Child's right privileg
  - Seek opinion of allied specialty.

"Every child has a fundamental right to his/her total oral health"

- Tag line of ISPPD

→ SAAPD - South asian academy of Pediatric dentistry.
→ AAPD - American academy of Pediatric dentistry (1947)
→ IAPD - International associations of Pediatric dentistry.

- 1st dental visit :-

Nowark - 1997 → 1st year

AAPD - 1986 → 6 months

- Role of parents :-
  - Ask procedure
  - Plan the visit
  - Talk to child
  - Bring record.

- Pre-appointment behaviour modifications :-
  - Pre-appointment mailing.
  - Pre-appointment modelling.
    - 1. audiovisual
    - 2. live - child sees tt done on others.

- Recommended procedure :-
  - Clinical
  - Radiographic
  - Simple procedure - fluorides, oral prophylaxis, etc.

- Euphemism :-

Use of 2nd language of child.

- Tweezer → चिमटा
- Probe → spoon
- Suction → straw
- Aerotor → special brush
- Air → wind
- Bur → Brush / pen
- Handpiece → wishing

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- Caries → Brown spot, sugar bugs
- Explorer → Tooth counter

Parental separation :- only when child is alone to the communicate with you.

2nd visit :- Infants & toddler.

1. Objective :-
   - Introduction
   - Risk assessment & oral examination
   - Prevention - pit & fissure sealant & fluoride twice a year.

2. Steps of infants :-
   - Pre - appointment
   - Recall shedule

3. Tips :- Read story
   well rested child
   Play dentist
   Given history
   No anxiety to your child
   Allow alone time
   Toy support.

- Dental Home :-

- Relationship between dentist patient
- 1st introduced in 1999.

Characteristics :-

1. Accessible
2. Family centres
3. Continuous
4. Comprehensive
5. Co-ordinate
6. Compassionate

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7. Culturally component

Advantages:-

- Rarely intervation
- Encourage 1st dental visit.
- Periodic preventive care.
- Anticipatory care.

Child friendly dental clinic:-

- Space provision.
- Front desk
- Waiting room
- Attire & presentation of clinical staff.
- Colour, smell & sound.
- Instruction for children & parents.
- Readiness to accept children.
- Visits & reward.
- Audiovisual aids for entertainment.
- Team approach.
- Design of equipment.

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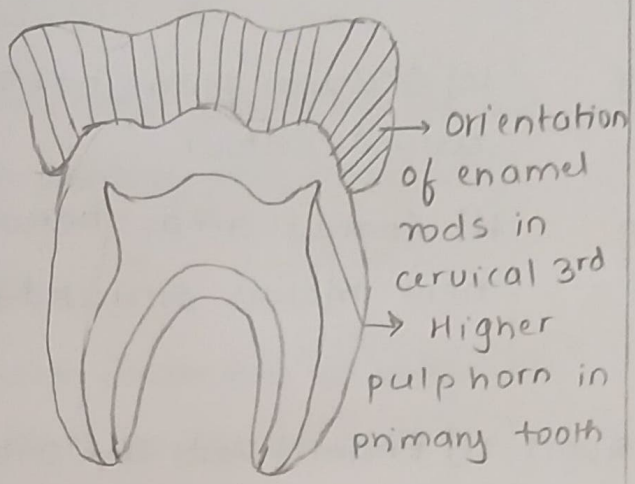
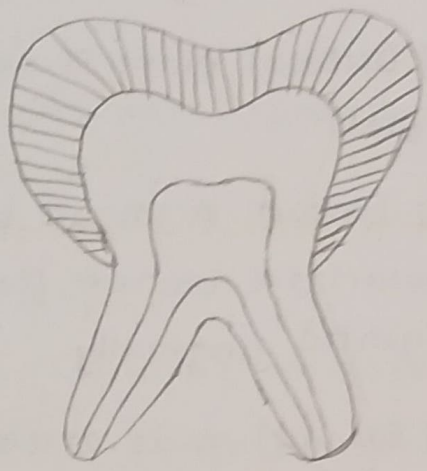
MORPHOLOGICAL DIFFERENCE BETWEEN PRIMARY & PERMANENT TEETH & IT'S SIGNIFICANCE





	PRIMARY TEETH	PERMANENT TEETH
#	CROWN :-	
1]	Smaller in dimensions.	1] Larger in dimensions.
2]	Lighter in colour, bluish white (milky white) called milk teeth as its refractive index same with milk [1.33]	2] Darker in colour, grayish or yellowish white.
3]	No. of teeth → 20 $\frac{2}{2}$ I ; $\frac{1}{1}$ C ; $\frac{2}{2}$ M	3] No. of teeth → 32 $\frac{2}{2}$ I ; $\frac{1}{1}$ C ; $\frac{2}{2}$ PM ; $\frac{3}{3}$ M
4]	Supplemental grooves are more	4] Supplemental grooves are less.
5]	Crowns are wider in mesio-distal dimensions than cervico-occlusal & gives cup shaped appearance to anterior & square shape to molars.	5] Crown of anterior teeth are wider in cervico-occlusal dimension than mesiodistal dimensions & gives tapering appearance to permanent teeth.

7] Mamelons are absent.	7] Mamelons are ~~absent~~ present.
8] Cuspids are slender & tend to be more conical.	8] Cuspids are less conical.
9] cervical ridges more pronounced.	9] Cervical ridges are flat.
10] Occlusal plane is relatively flat.	10] Occlusal plane has more curved contour.
11] Contact areas are between molars are broader, flatter & situated gingivally.	11] Contact areas between permanent molars situated occlusally.
12] Enamel rods at cervical slopes occlusally from CEJ.	12] Enamel rods are oriented gingivally.
13] Eamel is thinner & has a more consistent depth of about 1mm thickness through entire crown.	13] Enamel is thicker & has a thickness of abour 2-3mm.
14] Molars are more bulbous & are sharply constricted cervically.	14] They have less constriction of the neck.
15] First molar is smaller than the 2nd molar in dimensions.	15] First molar larger in dimension than second molar.
ROOT :-	
16] Roots are larger & more slender in comparison to crown size.	16] Roots are shorter & bulbous in comparison to crown
17] Roots are narrower mesiodistally	17] Roots are broader mesiodistal
18] Furcation is more towards cervical area & root trunk is smaller.	18] Furcation placement is apical so root trunk is larger.

19] At cervical region roots of primary molar flare outward & continue to flare as they approach apices to accomodate permanent molars.	19] Marked flaring of roots is absent.
20] Undergo physiologic resorption during the shedding	20] Physiologic resorption is absent
# PULP :-	
21] Pulp chamber is larger in relation to crown size.	21] Pulp chamber is smaller in relation to crown size.
22] Pulp horns are closer to outer surface, mesial pulp horns extends to closer approximately of surface than distal pulp horn.	22] Pulp horns are comparatively away from outer surface.
23] Pulpal outlines follow DEJ more closely.	23] Pulpal outlines follow DEJ less closely.
24] High degree of cellularity & vascularity in tissue.	24] Comparatively less degree cellularity & vascularity in tis
25] High potential for reparr.	25] Less potential for reparr.
26] Comparatively less tooth structure for protection.	26] Comparatively more too structure for protection.
27] Root canals are more [illegible] like & follows branching [illegible]	[illegible] less [illegible]
28] Floor of pulp chamber is porous. Accessory canals in 1° pulp chamber floor leads directly into interradicular furcation.	Floor of pulp chamber doesn't have any accessory canals.

# HISTOLOGIC DIFFERENCES :-	
29] Roots have enlarged apical foramen thus abundant blood supply demonstrates more typical inflammatory response.	29] Foramen are restricted. Reduced blood supply calcified response & healing by calcified scaring.
30] Incidence of reparative dentin formation beneath carious lesion is more extensively & irregular.	30] Reparative dentin formation is less.
31] Pulp nerve fibres pass to odontoblastic area where they terminate as free nerve ending.	31] Pulp nerve fibres terminate mainly among odontoblast & even beyond predentin.
32] Density of innervation is less.	32] Density of innervation is
33] Localization of infection & inflammation is poorer in pulp.	33] Infection & inflammation pulp is localized.
# MINERAL CONTENT:-	
34] Enamel & dentin are less mineralized.	34] Enamel & dentin are more mineralized.
35] Neonatal lines present.	35] Neonatal lines seen only in the 1st permanent molar.
36] Bands of Retzius are less common in enamel & responsible for bluish white colour.	36] Bands of Retzius are more common.
37] Dentinal tubules are less regular.	37] Dentinal tubules are more regular.

38] Dentin forming cells are functionally active by 360 days.	38] Dentin forming cells are functionally active by 700 days.
39] Interglobular dentin absent.	39] Interglobular dentin present
40] Dentin usually less dense & easily cut comparatively.	40] Dentin difficult to cut.
41] Secondary cementum absent.	41] Secondary cementum present.
42] Alveolar atrophy rare.	42] Alveolar atrophy occurs.
43] Gingivitis generally absent in healthy child.	43] Gingivitis common in adults.

CLINICAL SIGNIFICANCE :-

1] As deciduous dentition is lighter in colour when compared to permanent dentition. Thus while restoring primary teeth lighter shades are used.

2] Cervical ridges are more pronounced specially on buccal aspect of 1st primary molars.

3] These cervical bulges have to be reproduced during restoration / crown prosthesis.

4] Sharp cervical constriction has to be kept in mind & specia care should be taken while forming gingival floor during class II cavity preparation.

5] During class II cavity preparation for amalgam, buccal & lingual extensions / wall of class II cavity should be located in self cleansing areas

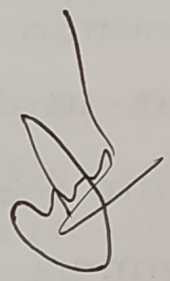
6] Deciduous molars are functionally adapted to withstand less occlusal load.

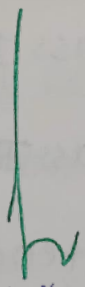
7) Less pressure / force is arequired during cavity preparation of primary teeth, depth of cavity preparation is less.

8) Etching time is prolonged to 90–120 seconds due to differ between their mineralization.

9) Roots of primary molars are flared to accomodate the permanent tooth buds between their roots hence their extraction is difficult.

10) Primary teeth have abundant blood supply & exhibit a poor localization of infection & inflammation & exhibit a more typical inflammatory response.





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CHRONOLOGY & ERUPTION AGE

- Deciduous eruption sequence :-

  Maxillary → A B D C E

  Mandibular → A B D C E

- Permanent eruption sequence :-

  Maxillary → 6 1 2 4 3 5 7 8

  Or

  6 1 2 4 5 3 7 8

  Mandibular → 6 1 2 3 4 5 3 7 8

Primary dentition :-

Maxilla

Tooth	Hard tissue formation begins (month in utero)	Crown completion	Eruption (months)	Root completion (years)
central incisor	4	4	♂ → 11-14 ♀ → 10-14	1.5
Lateral incisor	4.5		♂ → 10-16 ♀ → 10-16	2
canine	5		♂ → 19-24 ♀ → 22-24	3.25
1st molar	5	6	♂ → 18-20 ♀ → 16-20	2.5
2nd molar	6	11	♂ → 28-36 ♀ → 26-32	3

Mandible :-

Tooth	Hard tissue formation begins (months in utero)	Crown completion (months)	Eruption (months)	Root completion (years)
Central incisor	4.5	4.5	♂ → 10-12 ♀ → 10-12	1.5
Lateral incisor	4.5	4	♂ → 12-14 ♀ → 11-14	1.5
Canine	5	9	♂ → 20-29 ♀ → 20-29	3
1st molar	5	5.5	♂ → 18-20 ♀ → 18-20	2.25
2nd molar	6	10	♂ → 26-30 ♀ → 26-32	3

Permanent dentition :-

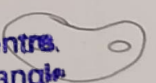
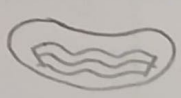
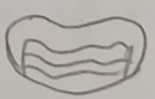
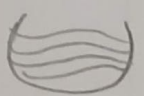
Maxilla :-

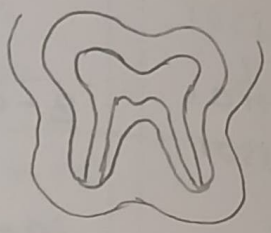
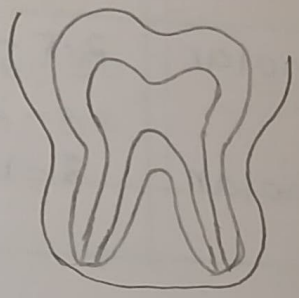
Tooth	Hard tissue formation begins	Crown completion	Eruption	Root completion
Central incisor	3-4 months	4-5 yrs	7-8 yrs	10 yrs
Lateral incisor	10-12 months	4-5 yrs	8-9 yrs	11 yrs
Canine	4-5 months	6-7 yrs	11-12 yrs	13-15 yrs
1st premolar	1.5-1.7[illegible] yrs	5-6 yrs	10-11 yrs	12-13 yrs
2nd premolar	2-2.5 yrs	7 yrs	10-12 yrs	12-14 yrs
1st molar	Birth	2.5-3 yrs	6-7 yrs	9-10 yrs
2nd molar	2.5-3 yrs	7-8 yrs	12-15 yrs	14-16 yrs
3rd molar	7-9 yrs	12-16 yrs	17-21 yrs	18-25 yrs

Mandible :-

Tooth	Hard tissue formation begins	Crown completion	Eruption	Root completion
central incisor	3-4 months	4-5 yrs	6-7 yrs	9 yrs
Lateral incisor	3-4 months	4-5 yrs	7-8 yrs	10 yrs
canine	4-5 months	6-7 yrs	9-10 yrs	12-14 yrs
1st premolar	1.75-2 yrs	5-6 yrs	10-11 yrs	12-13 yrs
2nd premolar	2.25-2.5 yrs	6-7 yrs	11-12 yrs	13-14 yrs
1st molar	Birth	2.5-3 yrs	6-7 yrs	9-10 yrs
2nd molar	2.5-3 yrs	7-8 yrs	11-13 yrs	14-15 yrs
3rd molar	8-10 yrs	12-16 yrs	17-21 yrs	18-25 yrs

Nolla's stage of tooth development

Stage 0	Absence of crypt	
Stage 1	Presence of crypt	
Stage 2	Initial calcification	
Stage 3	One third of crown completed	
Stage 4	2/3rd of crown completed	
Stage 5	Crown almost completed	
Stage 6	Crown completed	

Stage 7	One third of root completed	
Stage 8	Two third of root completed	
Stage 9	Root almost completed Open apex	
Stage 10	Apical end of root completed	

ERUPTION & SHEDDING OF TEETH

THEORIES OF ERUPTION

- Eruption:- Eruption is the process of whereby the following tooth migrates from the intraosseous location in the jaw to its functional position within the oral cavity.

Movements leading to eruption of tooth can be divided into-

Phase 1:- Pre-eruptive phase

Phase 2:- Pre-function eruptive or eruptive phase

Phase 3:- Functional eruptive or post eruptive phase.

- Anatomic stages of tooth eruption:-

Given by Noyes & Schour:-

Stage 1 :- Preparative stage (opening of the bone crypt).

Stage 2:- Migration of tooth towards oral epithelium.

Stage 3:- Emergence of crown tip into oral cavity (beginning of clinical eruption).

Stage 4:- First occlusal contact.

Stage 5 :- Full occlusal contact

Stage 6 :- Continuous eruption

- Stages of tooth eruption:-

Roots begin their formation as a result of proliferation of both the epithelium root sheath & the mesenchymal tissue of the dental papilla & dental follicle.

The erupting tooth moves through the bone of the crypt & the connective tissue at the oral mucosa.

the reduced enamel epithelium covering the crown
oral epithelium.

reduced enamel epithelium of the crown proliferates &
with oral epithelium.

the oral cavity by degenerat
membrane & breaking through the contact centre
of double layered epithelium.

crown erupts further & the lateral border of the oral
mucosa now becomes the dentogingival junction (DGJ)

The reduced enamel epithelium now surrounding like
a cuff becomes known as junctional or attachment ep

Erupting tooth continues to move occlusally as the
result of eruptive eruption, exposing more of
clinical crown.

Shedding of deciduous teeth :-

Three physiologic process resulting in the elimination
of the deciduous dentition is called shedding/exfoliation

Pattern of shedding :-

The shedding of deciduous teeth is the result of
progressive resorption of roots of teeth & their
supporting tissues.

In general, the pressure generated by the growing
& erupting permanent tooth dictates pattern of
deciduous tooth resorption.

- Resorption of Anterior teeth:-
  - The position of the permanent anterior tooth germ is lingual to the apical third of the roots of primary tooth; Hence, the resorption is in the occlusal labial direction, hence which corresponds to the movements of permanent tooth germ.
  - Later, the crown of permanent tooth lies directly apical to the root of primary teeth, which causes resorption to proceed horizontally.
  - This horizontal resorption allows the permanent tooth to eruption into the position of the primary teeth.

- Resorption of Posterior teeth:-
  - The growing crown of the premolars initially are situated between the roots of the primary teeth.
  - The initiation is by the resorption of the interradicular bone followed by resorption of the adjacent surfaces o the root of primary teeth.
  - Meanwhile, the alveolar process is growing to compen sate for lengthening roots of permanent tooth. As th occurs, the primary molars move occlusally; the allo the premolar crowns the more apical.
  - The premolars continue to erupt until the primary molar roots are entirely resorbed & the teeth exfolic The premolars then appear in place of primary molar

- Mechanism of resorption:-
  - Pressure of succeeding permanent teeth.
  - Increase in force of mastication.
  - Action of odontoclast (cementoclast for cementum, & dentinoclast for dentin).

- Action of odontoclast with resorbed bone.
- PDL is resorbed by fibroblast.

- Theories of Tooth Eruption :-

1] Root Elongation Theory :-

According to this theory, the simplest & most obvious mechanism of eruption would be that the crows of the teeth are pushed into the oral cavity by virtue of growth & elongation of the roots.

Evidence for the theory :- Root of the tooth elonga as crown erupts into the oral cavity.

Evidence against the theory :- Rootless teeth often erupt without the concomitant elongation of the roots. Submerged teeth often continue the formation of their roots but do not erupt.

2] Pulpal constriction theory :-
- Growth of the root dentin & subsequent pulp constriction may cause sufficient pressure to move tooth occlusally.
- Evidence → pulp is constricted growth of root dentin.
- Evidence against → Pulpless teeth.

] Growth of Periodontal Theory :-
- Pull by connective tissue (underwood)
- Periodontal fibres are pulled & vice versa.
- Alveolar bone growth (Hemley)

Pressure from muscular action (Berten)
- Action of musculature of cheeks & lips upon alveolar process squeeze crown of teeth out oral cavity.
- Evidence against - unilateral facial paralysis

5] Resorption of alveolar crest :-
- Resorption serves to expose crown.
- Histologically - alveolar crest - most rapid & continuous bone growth.

6] Hormonal Theory (Sir Arthur Keith) :-
Thyroid & pituitary hormones affect eruption.

7] Foreign body - (GotHeib's) :-
Tooth tends to exfoliate by tissue like foreign body.

8] Cellular proliferation (Noyes) :-
Tremendous osmotic pressure & forces from cellular proliferation account for eruption.

9] Vascularity Theory (constant) :-
- Tissue between developing & it's bony swelling surrounding process very rich vascular supply. Blood pressure leading to eruption.
- Evidence - Hyperemia → submerged, periodontitis.

10] Blood vessel thrust :-
Hydrodynamic & hydrostatic force within blood vessels

11] Periodontal contraction theory :-
Contraction of periodontal ligament, collagen constriction due to fibroblast cause eruption.

12] Dental follicle :-
- Bony remodelling to accomodate teeth movement helped by dental follicle.
- Reduced enamel epithelium cascade intracellular.
- It is clear that dental follicle is essential to achieve the bony remodelling required to accomodate tooth movement for it is this tissue that the osteoblast differentiate.

TOOTH NUMBERING SYSTEM

1] Zsigmondy - Palmer System:-

Primary Dentition:-

E	D	C	B	A	A	B	C	D	E
E	D	C	B	A	A	B	C	D	E

Permanent Dentition:-

8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8

- Advantages:-

1] Effective applicability.
2] Easy to use & understand.
3] Easy to record in patient records / radiographic record

- Disadvantages:-

1] Take more space in the patient's files.
2] Very difficult to type in electronic files.
3] Requires special software.
4] Needs private processing editor.

2] Federation Dentaire Internationale System:-

Primary Dentition:-

55	54	53	52	51	61	62	63	64	65
85	84	83	82	81	71	72	73	74	75

Permanent Dentition:-

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28
48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

- Advantages:-

1] Simple to understand & to teach.

2] Easy to pronounce in conversation & direction.

3] Readily communicable in print.

4] Easy to translate into computer output.

5] Easy to adopt to standard charts with used in general practice.

- Disadvantage:-

1] Difficulty in understanding if the number written is in FDI or universal system.

2] Some general practitioners report they were confused by this system.

3] In the case of deciduous dentition, there can be confusion, which is difficult to remember

4] Common mistake in writing was mirror position as far as FDI numbering system is concerned, there is a world of difference between tooth number 32 & tooth no. 32.

3] Universal Numbering System:-

Primary dentition:-

A	B	C	D	E	F	G	H	I	J
T	S	R	Q	P	O	N	M	L	K

Permanent dentition:-

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
32	31	30	29	28	27	26	25	24	23	22	21	20	19	18	17

- Advantages:-

1] Convienient to write.

2] Convenient to record in the patient's record.

3] Every tooth has a separate number.

- Disadvantages:-

1] Need skill & training to build a habit of correct counting.

2] In primary dentition, especially during mixed dentition counting is difficult.

3] Difficult to count tooth without a picture present mainly in the absence of third molars.

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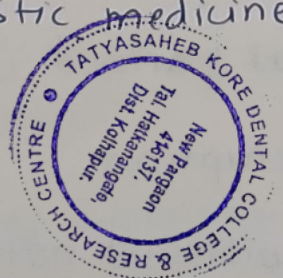
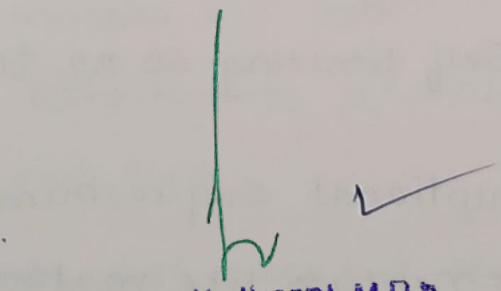
TEETHING

- Definition :-
  It is the process by which an infant's first teeth sequentially erupts & appears in the oral cavity.
- Signs & symptoms:-
  - Pain
  - General irritability / malaise.
  - Mucous membrane inflammation.
  - Drooling / Sialorrhoea
  - Facial flushing / circumoral rash
  - Biting
  - Suckling / gum rubbing
  - Disturbed sleep.
  - Bowel upset
  - Loss of apatite.
  - Ear rubbing on same side as erupting teeth.
- Management :-
  1) Pharmacological
  2) Non-pharmacological
  3) Alternative Holistic medicine.

1) Pharmacological :-
   1) Topical agents.
   2) Lignocaine based products





3) Choline salicylate based products.

4) Systemic analgesics.

2] Non-pharmacological :-

1) Peeled cucumber, Frozen bananas.

2) Hard, non-sweetened musts.

3) Pacifier

4) Teething risk.

3] Alternative Holistic medicine :-

1) Accupressure

2) Armotherapy

3) Homeopathy

4) Massage.

Teething disturbances :-

1] Eruptional hematomal cyst :-

- Bluish purple, elevated area, few weeks before 1% of the permanent tooth.
- Primary second molar, secondary 1st molar are com
- Self limiting so no treatment needed.

2] Eruptional sequestrum :-

- Cementum like material formed within dental folli
- Seen at first molar eruption.
- Spontaneously resolve without noticeable symptoms.

- In case where eruption sequestrum causing local irritation & has surfaced through mucosa it may be easily removed.

3] Ectopic eruption:-
- Arch length discrepancy.
- Retained primary dentition.
- Premature loss of deciduous teeth.
- Non-spaced primary dentition.

Natal & Neonatal teeth :-
- Defined by masseter & savara.
- Natal teeth are present at birth.
- Neonatal teeth erupt during first 20 days of life.
- More in females.
- Natal & neonatal teeth are approximately ratio of 3:1.
- Most affected lower primary central incisor.

• Hebling's classification:-

Category 1 - A shell like crown structure loosely attached to alveolus by rim of oral mucosa not root.

Category 2 - Solid crown loosely attached to alveolar by oral mucosa, little or not root.

Category 3 - Incisal edge of crown just erupted through oral mucosa.

Category 4 - Mucosal swelling with tooth unerupted but palpable.

• Treatment:-

1) CHX 3 times a day.

2) Extraction - Natal :- Vitamin K prophylactic.

Vitamin K synthesizes clotting factors

-Helps in stopping bleeding after extraction.

• Neonatal - Vitamin K may not be needed as gut flora develops within 10 days → synthesize Vitamin K → liver → synthesize clotting factor.

• Extraction indicated - Threat of aspiration & problem during breast feeding.

• Complications :-

- Traumatic ulceration of ventral surface of tongue.
- Riga fede disease as histologically it was described by them also called neonatal sublingual traumatic ulcerati

Non-erupting teeth :-

• etiology - Premature loss of teeth.
• treatment - Incision, gum massage.

Ankylosed teeth :-

- Most common is mandibular primary molar.
- Tooth root become firmly attached to alveolar bone before their normal exfoliation.
- Expensive bony ankylosis of primary tooth prevent normal exfoliation & eruption of 2° successors.

Epstein pearls :-

- Midpalatine raphe are remnants of epithelial tissue trapped along raphe as foetus gravis.

• John nodule :-

- Formed along buccal & lingual aspect of dental ridge & on palate away from raphe.
- Remnants of salivary gland tissue.

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DEVELOPMENT OF DENTITION

- Tooth development begins at 3rd week of intrauterine life
- First sign of tooth development is proliferation of oral ectodermal cell to form epithelial thickening called 1° epithelial band, that projects into underlying ectomesenchyme along future tooth bearing regions of each jaw.
- Primary epithelial band is formed by 6th week of intrauter
- Lingual extension of dental lamina contributes to formation of teeth.
- Successional lamina of central incisor develops at 5th month in utero & 2nd premolar at 7th month of age.
- Permanent molars develops from distal posterior extension of dental lamina, referred to as accesional / parent dental lamina / lamina of permanent molars.
- Permanent ~~m~~ 1st molar buds develop at 4th month of intrauterine life & 2nd molar at 1st year & 3rd molar at 4th / 5th year of life.
- Parts of tooth germ & its derivatives:-

Oral ectoderm

Dental lamina

Enamel organ

Enamel.

Ectomesenchyme.

- Dentin
- Pulp
- Dental papilla

- Dental follicle
- Cementum
- Alveolar socket
- PDL

- Components of tooth germ :-

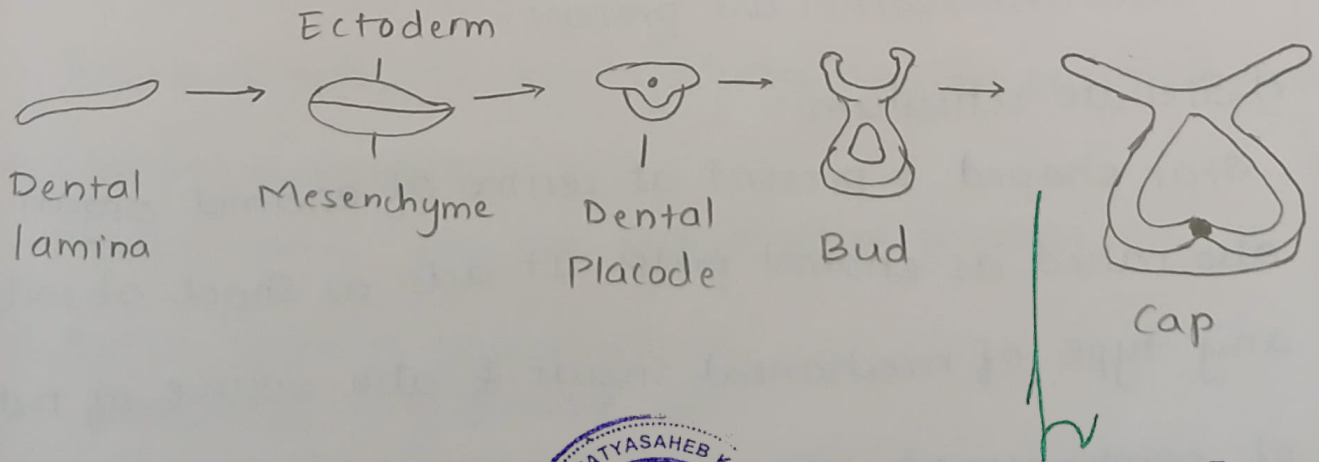
1] Enamel organ - Develops from dental lamina & is ectodermal in origin forms enamel.

2] Dental papilla - Ectomesenchymal in origin.
- Gives rise to dentin & pulp.

3] Dental follide - Ectomesenchymal & forms cementum, PDL & part of alveolar socket.

- Morphology & Physiological stages of Tooth development
  - Morphological stages :-
    1) Bud stage.
    2) Cap stage - Early & late
    3) Bell stage - Early & advanced.

1] Bud stage :-
- Enamel organ - round / ovoid
- Basement membrane present
- cells lining periphery of bud - cuboidal.
- central cells - polyhedral
- Dental papilla & sac can't be differentiated as distinct.



Cap stage :-

- Tooth germ appears like cap of enamel organ sitting on a ball of dental papilla, both enclosed in a sac of follicle.
- Convex surface of cap faces oral cavity.
- Physiologic phases present are proliferation, histodifferen & morphodifferentiation.
- Based on phase of development of stellate reticulum.
  1) Early cap - central cells are polyhedral in shape.
  2) Late cap - central cells form a network of stellate shaped cells.

• Histology of cap stage :-

Enamel organ has 3 different types of cells :-

A] Inner enamel / dental epithelium - low columnar cells.

Desmosomal junction - to attach adjacent cells.

Hemidesmosomal junction - to attach basement membrane

B] Outer enamel epithelium :-

cells lining convex cap portion & are cuboidal desmosom & hemidesmosomes are present.

c] Stellate reticulum :-

star shaped & present at centre of enamel organ. It is also called as enamel pulp. It acts as shock absorbers fr any type of mechanical injury & also source of nutrition of neighbouring cells.

temporary structures of enamel organ :-

1) Enamel knot
2) Enamel chord } Rapid proliferation of enamel organ cells.
3) Enamel septum
4) Enamel navel
5) Enamel niche

- Dental papilla: Main source of nutrition to inner enamelep.
- Dental follicle: Becomes more conspicuous at this stage.

3) Early bell stage :-

- Histodifferentiation & morphodifferentiation occurs rapidly.
- Enamel organ →

1) Inner enamel epithelium: Histodifferentiation.

- membrana perfomative - blueprint of crown, columnar 40 µ length, 4-5 µ width.

migration of centrally placed nucleus away from base to proximal end is reversal of polarity.

2) Stratum intermedium →

- Desmosomes present
- Single functional unit, contributing to enamel formation

3) Stellate reticulum:-

- Desmosomes, alkaline phosphatase present.

4) Outer enamel epithelium :-

Cuboidal → flattened cells.

Desmosomes present

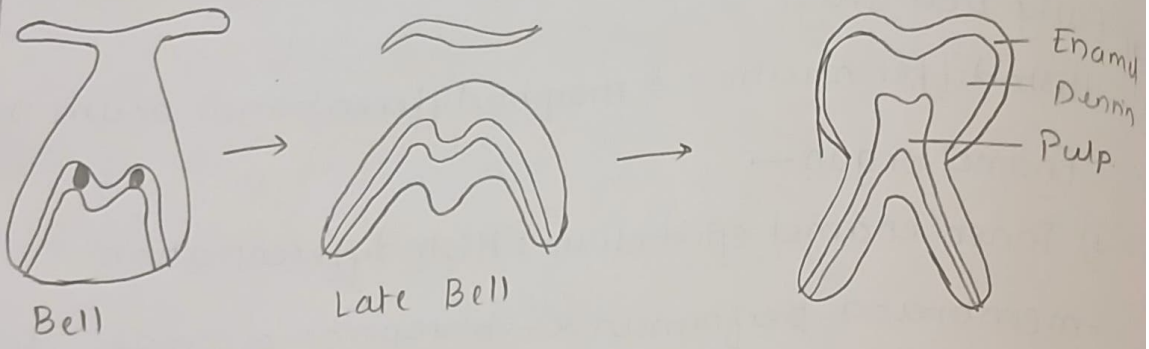
cervical loop or zone of reflection. Stellate reticulum
stratum intermedium absent.

Dental lamina: Attaches enamel organ to oral ectoderm

Successional lamina: forms premordia of permanent s

Dental papilla: peripheral cells change shape.
→ cuboidal to columnar.

Dental follicle/sac → becomes more distinct, Gives r
to cementoblast, osteoblast, fibroblast.



4) Advanced Bell stage:-

- Outer enamel epithelium - more irregular
stellate reticulum collapse.
- Dentin deposition by differentiated odontoblasts, begins at DEJ in region of cusp tip, progresses inwards/ pulpally & cervically.
- Enamel deposition at incisal edge/cusp tip at DEJ & progresses outward & cervically.
- After completion of enamel, columnar ameloblast shorten to cuboidal & merges into reduced enamel epithelium.

-Reduced enamel epithelium - plays important role in establishing dentogingival junction. Elaborates enzymes that have role in tooth eruption.

- Root formation :-

-Enamel organ proliferates to give rise to Hertwig's epithelial root sheath.

-HERS determines number, size, shape of root.

- Inner enamel epithelium → columnar cells - inner layer.

- Outer enamel epithelium → cuboidal cells - outer layer.

- Epithelial diaphragm extends between dental papilla & dental sac & is further apical foramen.

- Radicular dentin formation continues apically & inward while cementum formation continues apically & outward.

Physiological phases :-

1] Initiation :-

Result of epithelial mesenchymal interactions & decides commencement of tooth formation.

2] Proliferation :-

-Help to provide adequate cells for further development of tooth germ & also contribute to determination of shape of crown.

3] Morphodifferentiation :-

- Ensures normal shape & size of developing tooth.

-Make sure that tooth germ is changed from an undifferentiated bud to more differentiated bell stage.

Histodifferentiation:-

- Cells undergo morphologic & functional changes to perfo their function.
- Begins at cap & maximum at bell stage.
- Therefore, appositional activity & differentiated cells lo their capacity to proliferate.

Apposition:-

- Rhythmic deposition of dental hard tissue is apposi
- Once DEJ is established, formative cells start surre deposition of organic matrix which gets mineralized form dental hard tissue.

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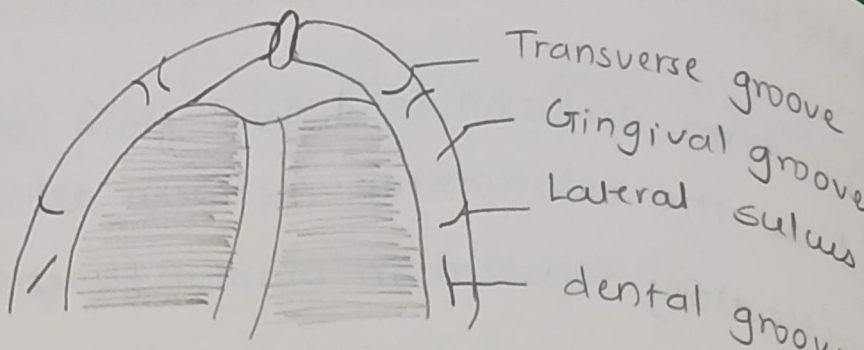
DEVELOPMENT OF OCCLUSION

The term occlusion is derived from latin word "occluso" defined as the relationship between all the components of masticatory system in normal function, dysfunction & parafunction.

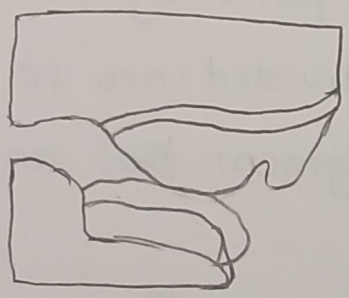
A) Pre-deciduous dentition:-

- This period is soon after birth, during this, the neonate has no teeth. The relation of the gum pads is of equal importance.
- The alveolar process at the time of birth is called the gum pads.
- They are horse-shoe shaped pads that are pink, firm & covered with layer of dense periosteum.
- They are divided into two parts by dental groove.
- The gum pad is further divided into 10 segments by transverse groove, each segment has one developing tooth sac.
- A very important landmark in gum pads is lateral sulcus which is the transverse groove between canine & first molar. This is helpful in predicting interarch relation at a very early stage.



Shape of gum pads.

- The maxillary gum pad is wider & longer than the mandibular. Thus, when they are approximated, there is a complete overjet all around. The only contact that occurs is around molar region. while space exists in anterior region. This is called infantile open bite, which is considered normal & helpful during suckling.



Anterior open bite relation between upper & lower gum pads & birth

Deciduous dentition -

The initiation of primary teeth occurs during 1st week of intrauterine life & 1st primary tooth erupts at the age of 6 months. The individual variations apart, it takes around 2.5 - 3.5 years for all the primary teeth to establish their occlusion.

Spacing :-

- spaced dentition:- It is supposed to be good, as spaces in betn teeth can be utilized for adjustment of permanent successors which are always larger in size compared to the deciduous teeth. The spaces present are of two types.

- Primate spaces -

Exist between maxillary lateral incisor & canine & mandibular canine & first deciduous molar. These spaces are also called anthropoid or simian spaces as they were initially found in our ancestral simian species.

- Physiologic spaces -

Present between all the primary teeth & play an important role in normal development of the permanent dentition. The total space present may vary from 0 to 8mm with the average of 4mm in maxillary arch & 1-7mm with the average of 3mm in mandibular arch.

- Non-spaced dentition:- This dentition is highlighted by lack of space between primary teeth either due to small jaw or larger teeth. This type of dentition usually indicated crowding in developing permanent dentition.

Terminal planes :-

1) Flush terminal plane -

- The distal surface of deciduous mandibular & maxillary molars are in straight plane & therefore situated on same vertical plane.

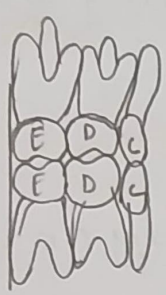
- Further proceed into class I in 74% cases.

Mesial step terminal plane :-

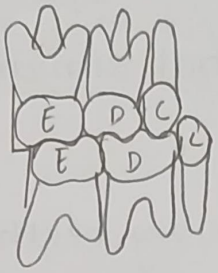
- The distal surface of deciduous second mandibular m
is more mesially to deciduous second maxillary molar
- Class I & class III (14% cases of mandible growing
forward is more).

Distal step terminal plane :-

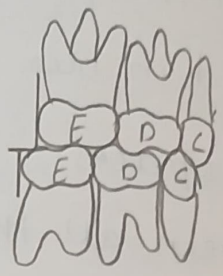
- The distal surface of deciduous 2nd mandibular mo
more distal to that of deciduous 2nd maxillary mola



Flush terminal plane



Mesial step terminal plane



Distal step terminal plane

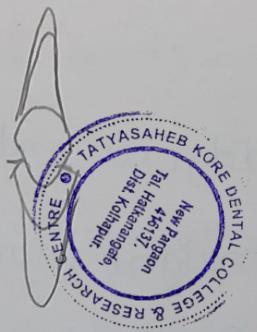
Canine relationship :-

Class I : The mandibular canine interdigitates in the
between maxillary lateral incisor & canine.

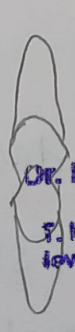
Class II : The mandibular canine interdigitates distal t
embrasure betn maxillary lateral incisor & canine.

Class III : The mandibular canine interdigitates in oth

Class I



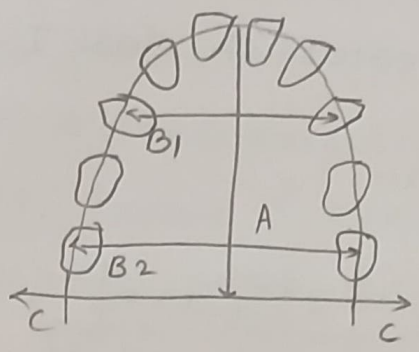
Class II



Class III

- Arch dimension :-
- 1st given by zigmondy in 1890.

A1 → Arch length
A2 → Bimolar diameter.
B1 → Bicanine length
B2 → Arch perimeter.



Mixed dentition period :-

- This phase begins at around 6 years with eruption of 1st permanent molar & last till about 12 years of age.
- It has 3 phases :-

1. First transitional period-
   - Emergence of 1st permanent molar
     - ↳ Early mesial shift
     - ↳ Late mesial shift
   - Incisor transition.
2. Intertransitional period
3. second transitional period
   - Emergence of cuspid, bicuspid & 2nd permanent molars.
   - Establishment of occlusion.

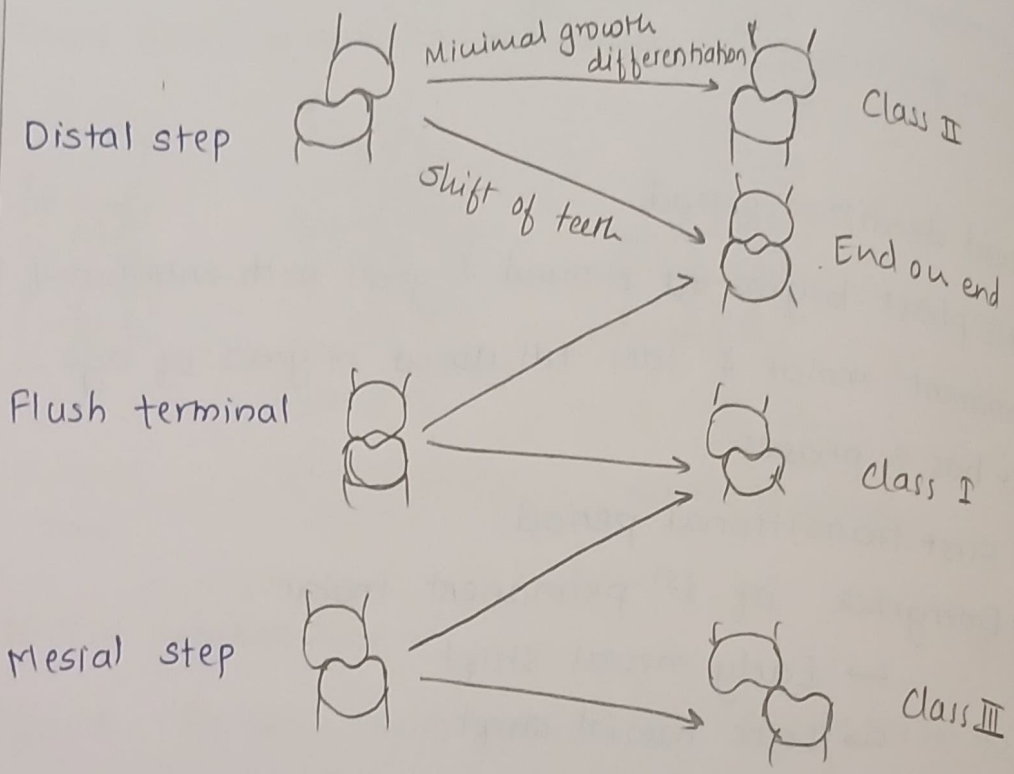
- Early mesial shift :-

The eruptive forces of 1st permanent molar are strong enough to push deciduous molars forward in arch thereby utilizing the primate space & thus establishing class I relationship.

- Late mesial shift :-

Many children lack primate space & have a non-sp
dentition & thus erupting permanent molars are not
to establish class I relation by drifting mesially & u
the leeway space after exfoliation of deciduous m
this is called leeway & establish class I relationship

- Terminal plane prediction :-



- Exchange of incisors :-

- Deciduous incisors replace by permanent incisors.
- This period of transition is from 6.5 to 8.5 years.
- The permanent incisor are larger as compare
their permanent counterparts & thus requird m
space for their permanent. This difference betwe
available & space required is called as liability
- Incisor liability is 10mm maxillary arch & 5mm
mandibular arch.

- Factors that help in alignment of incisor by gaining sp-
1. Utilization of interdental spacing of 1° incisors.
2. Increase in intercanine arch width.
3. Increase in intercanine arch length.
4. change in intercanine angulations.

2] Interpositional period :-
- In this period, the maxillary & mandibular arches consist of permanent incisor & permanent molars that sandwich the deciduous canines & molars.
- This phase lasts for 1.5 years & is relatively stable.
- Only a few changes in the morphology of deciduous teeth are seen because they undergo attrition.

3] Second transitional period :-
- This phase lasts for is characterized by replacement of deciduous molars & canines by premolars & permanent cuspids & the eruption of maxillary lateral & canines.
- This take place around 9-11 year of age.

Replacement of deciduous molars & canine :-
- The combined mesiodistal width of permanent canine & premolars is less than that of deciduous canine & molars. This extra space is called leeway space of Nance. & is utilized by mandibular molars to establish Class I relationship through late mesial shift.
- 1.8mm - maxillary arch & 2.3mm - mandibular arch.
- The dimensions of deciduous molars are more than that of 2nd premolars, this excess space is called 'E' space.

- Eruption of maxillary canine :-

- As the permanent maxillary canine erupts, they dis[...] the roots of maxillary lateral incisor mesially. This fo[...] is transmitted to central incisor & their roots are di[...] displaced mesially.
- Thus, the resultant force causes the distal divergen[...] the crown in an opposite direction, leading to midli[...] spacing. This is called as 'Ugly duckling stage or Broadbent phenomenon'.
- This condition corrects itself after the canines have erupted. The canines after eruption apply pressure on crowns of incisors thereby causing them to shift ba[...] to original position.

Permanent dentition:-

Keys of occlusion-

Andrew in 1970 gave 6 necessary features for an ideal[...]

1] Molar interarch relationship:-

- The distal surface of distobuccal cusp of upper first permanent molar made contact & occluded with th[...] mesial surface of mesiobuccal cusp of lower 2nd mo[...]
- The mesiodistal cusp of the upper first permanent fell within the groove between the mesial & mid[...] cusps of lower 1st permanent molar.
- The canines & premolar enjoyed a cusp-embrasu[...] relationship buccally & cusp-fossa relationshi[...] lingually.

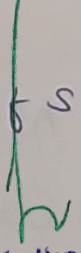
2) [illegible] angulation:-
- Crown angulation refers to angulation of the long axis of crown, not to angulation of long axis of entire tooth.
- The gingival part of long axis of crown must be distal to occlusal part of axis.

3] Crown inclination:-
- It refers to the habitual labiolingual or buccolingual inclination of body long axis of crown, not to the inclination of long axis of entire tooth.
- Crown inclination is determined by the resulting angle between line 90° to the occlusal plane & a line tangent to middle of labial or buccal clinical crown.

4] Absence of rotation:-
- Rotated teeth will occupy more space.
- Rotated molars & premolars occupy more space in the dental arch than nomal, rotated incisors may dental arch than nomal, rotated incisors may occupy less space than those correctly aligned & rotated canines adversely affect esthetics & may lead to occlusal interferences.

5] Tight contact:-
- Permanent dentition should have close contact to optimize space

6] Curve of spee:-
- Occlusal plane should be flat with curve of spee not exceeding 1.5 mm.
- There is natural tendency for curve of spee to deepen with time.



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DENTAL CARIES

"Dental caries is an irreversible microbial disease calcified tissue of teeth, characterized by demineral of inorganic portion & destruction of organic subst of tooth, which leads to cavitation."

Classification:-

A] According to occurence:-

1) Incipient - initial primary caries often reversible
2) Recurrent - secondary caries
3) Residual - caries left due to mistake of dentist

B] According to spread:-

1) Acute - fast spreading
2) Chronic - slow spreading

C] According to location:-

1) Pit & fissure
2) Smooth surface
3) Root surface

D] According to direction:-

1) Forward caries → when caries spread in V-shape base pointed towards DEJ.
2) Backward caries → when more extensive destr towards DEJ with small apex.

E] According to DEJ:-

1) Early childhood caries
2) Adolescent caries
3) Senile caries

F) According to surface :-

1) Simple - one surface involved
2) Compound - two surfaces involved
3) Complex - more than two surfaces involved.

G] According to type of surface :-

1) Occlusal
2) Proximal.

Theories of Dental caries :-

1] Miller's chemicoparasitic theory :-

- A synthesis of ideas that acid & micro-organisms were involved in etiology of dental caries.
- The micro-organisms of mouth, by secretion of enzymes or by their own metabolism, degrade formentable carbohydrate food material so as to form acids.
- The enamel is destroyed by acid of fermentation & disintegrated enamel is subsequently mechanically removed by forces of mastication.
- After penetration of enamel, the dissolution of dentin is brought about by in same manner with organism penetration along dentinal tubules.
- The final breakdown of dentin results from the secretion of proteolytic enzyme that digest the organic part of dentin.

Girtique of theory -

- Unable to explain predilection of specific sites on tooth.
- The initiation of caries on smooth surface was not accounted for by this theory.
- Does not explain why some populations are caries free.
- Phenomenon of arrested caries is not explained.

- The concept of tooth resistance while logical a
any experimental support.

3) Proteolytic theory :-

- The surface coverings found on the tooth, in th
pits are organic in nature also enamel contain
significant amount of organic material.
- They described caries like lesions that were
proteolytic activity at slightly alkaline pH &
The process involved depolymerization & liquefa
the organic matrix of enamel.

3) Proteolytic chelation theory :-

- This theory was proposed by Schatz et al in 19
- According to this theory a simultaneous solubili
of organic components & dissolution of minerals
by process of chelation.

4) Other theories :-

- Legend of worm
- Humoral theory
- Vital theory
- Chemical theory
- Sulphatase theory
- Complexing & phosphorylation
- Sucrose - chelation theory
- Autoimmune theory

Stephan's curve :-

- In 1940's Dr Robert Stephan suggested that
the continuous
of food & bacteria
carbohydrates.

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a) Resting plaque pH :- This describes that plaque has not been exposed to fermentable carbohydrates for approximately 2hr & generally has a pH of between 6 & 7.
- This resting plaque pH value for an individual tend to be stable & may remain so for long periods

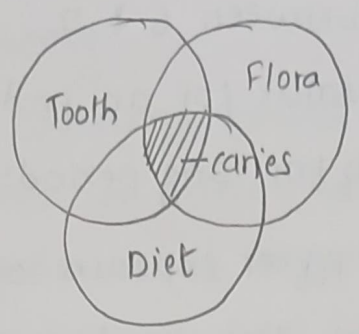
b) Decrease in plaque pH :- After exposure of dental plaque to fermentable carbohydrates, the pH decreases due to the part of composition of dental plaque.
- In general, if more acidogenic, aciduric bacteria is present in plaque, the pH would lower more rapidly. The rate of pH decreases is also dependent on the speed with which the plaque bacteria are able to metabolize dietary carbohydrates.

c) Critical pH:- The pH at which saliva no longer remains saturated with calcium & phosphate, thereby permitting hydroxyapatite in dental enamel to dissolve it is the highest pH at which there is net loss of enamel which is generally accepted to be about 5.5 for enamel.

d) Increase in plaque pH :- The low pH remained for some time, taking 30-60 min to return to its normal pH.
Differences were seen between caries free group & caries active group with latter group having significantly lower plaque pH

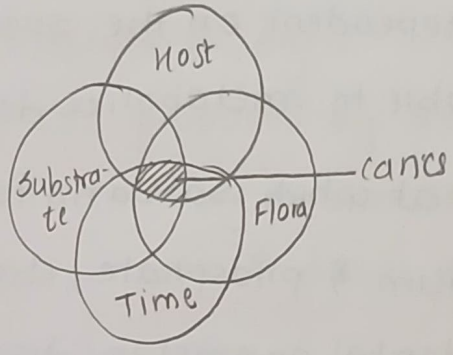
Keye's triad :-
Keyes & Jordan in 1960 gave a model stating that for initiation & progression of caries interaction between three primary factors is essential [illegible] the tooth, microflora with [illegible] potential; a

suitable local substrate to meet the requirements of pathogenic flora.



Keye's triad model

Newman's tetrad model :-



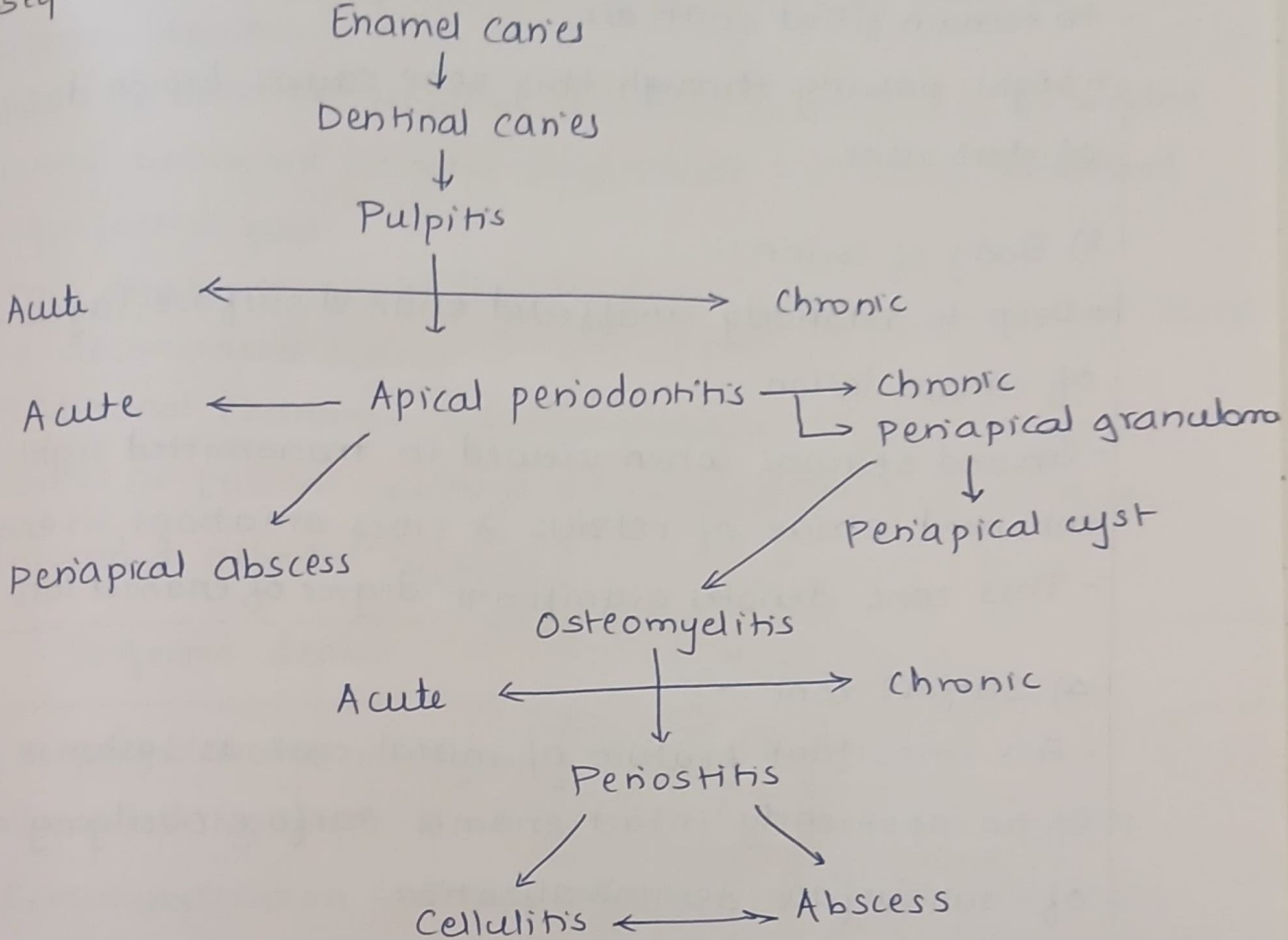
- Newman modified the Keye's triad in 1978 & gave tetrad model where time was added as a 4th fact proposed that dental caries result from the interact oral flora with dietary carbohydrates on the tooth over a period of time.

• Sucrose is called as an arch criminal.

- The dietary sugars all diffuse rapidly into the pl are fermented to lactic acid or other acids can be as intracellular polysaccharide by bacteria prolong fall in pH & promoting suitable environment for aciduric & acidogenic bacteria. Dextran is unique b it is the substrate for production of extracellular

polysaccharide & unsoluble matrix polysaccharide.
- Thus sucrose favors colonization of oral micro-organisms & increase sickness of plaque allowing it to adhere in larger quantities of teeth.

sequelae of dental caries :-



Histology of dental caries :-

1] Histopathology of enamel caries :-

- Light microscopic studies of carious lesions of enamel without cavitation have revealed four distinct zones :-

1) Translucent zone :-

- The advancing front of carious lesion is represented by this
- Enamel alteration / pores at junctional sites such as prism boundaries.

2) Dark zone :-
- Lies deep to body of lesion & just superficial to trans
- This zone is positively birefringent & has a pore volu
of 2-4% this molecular sizing effect permit microp
to remain filled with air.
- Light passing through this zone causes brown discol
of dark zone.

3) Body of lesion :-
- Deep to relatively unaffected enamel surface layers is
of carious lesion.
- Ground sections when viewed in transmitted light re
enhanced striae of retzius & cross striations in enam
- This zone denotes significant degree of enamel loss.

4) Surface zone :-
- An important feature of initial carious lesion is pre
of an apparently intact enamel surface overlying an
of subsurface demineralization.
- The surface zone has been defined as the zone of
birefringencence superficial to positively birefringen
body of lesion.

Histopathology of dentinal caries :-
- As the carious lesion invade dentin, dentinal tubu
becomes involved. This is divided into 3 zones :-
1) Zone of decomposed dentin
2) Zone of bacterial invasion
3) Zone of demineralization.

4) zone of dentinal sclerosis
5) zone of fatty degeneration.

- Translucent zone is identical to sclerosed dentin.
- Narrow zone of demineralization affecting intertubular matrix is seen, occlusion of dentinal tubule is also seen in sclerotic dentin.
- zone of bacterial invasion → lumen of tubule is distended giving ballooned / dilated appearance variously described liquefaction foci.
- This dilations eventually coalesce, forming outermost zone i.e decomposed dentin.
- Additional changes → cleft formation at right angle to tubules or follow contour lines of Owen & dead tracts.

Infected & affected dentin:-

Infected dentin	Affected dentin
1) Superficial dentin layer.	1) Deep layer
2) Invaded by bacteria.	2) Not attacked by bacteria.
3) Remineralization can't occur	3) Remineralization occurs.
4) Lack sensation.	4) Sensitive to instruments.
5) Indistinct collagen bands.	5) Distinct collagen bands.
6) Stained by 0.5% Fuschin or 1% aud red solution.	6) Cannot be stained.
7) Soft necrotic & flakes away with instrument.	7) Softer than normal dentin discolored but does not flake easily.

EARLY CHILDHOOD CARIES :-

"A complex disease involving maxillary primary incisors (month after eruption & spreading rapidly to other pri teeth is called early childhood caries".

[Davis 1998]

- The disease of ECC is the presence of one or more d missing or filled tooth surface in any primary tooth i child > 1 month of age or younger.
- In children younger than 3 years of age, any sign of s surface caries is indicative of severly early childhood cari ages 3-5, one or more activated, missing / filled smooth in primary maxillary anterior teeth or a decayed, mis or filled score of ≥ 4, ≥ 5 surface constitute S-ECC.

- Terminologies for ECC :-
  1) Nursing caries - Winter (1966)
  2) Tooth clearing neglect - Moss (1996)
  3) Infant & early childhood dental decay - Horowitz (19
  4) Maternally derieved streptococcus mutans disease.
  5) Baby bottle syndrome.

- Classification of ECC by Wyne :-

- Type I - Mild to moderate
  - Existence of isolated caries lesion involving molar cause is usually combination of cariogenic semiso food & lack of oral hygiene.
  - seen in 2 to 5 year old age.

Type II - moderate to Severe

- Labiolingual carious lesion affecting maxillary incis

- Mandibular incisors are not affected use of feeding bottle/ breast feeding / combination of both without / with poor oral hygiene seen soon after eruption.

- Type III - severe
- carious lesion affecting all teeth including lower incisors; cause is cariogenic food & poor oral hygiene condition is rampant

- Etiology :-
- Etiology of ECC is similar to other types of coronal smooth surface caries biology may differ in same aspect.

1) Plaque :- When fermentable carbohydrates are present, lactate is mainly produced pH drop in plaque.
- Bacteria & alkaline product provide contribution to pH rise in plaque & base generating metabolism of plaque bacteria considered by many to be significant determinant for cariogenicity of plaque.
- Presence of visible plaque & its early accumulation have been related to caries occurence among children.

) Mutans streptococci :- It possess of wide range of cariogenic traits by that they synthesize $\alpha-1,3$ rich water soluble glycans.
- Laser production of acid driving demineralization irreversible adhere to pellicle through synthesis of glycans medicated by glycosyntransferases.
- Generally requires non-shedding surface to colonize infectn rate increases with age as well as number transmission is likely to be mediated via saliva.

Infant feeding patterns :- Studies have reported bedtime bottle use in children with anterior decay.

Length of contact with bottle at night time is important children also who are exclusively breastfed also appear to susceptible to caries.

4) Tooth brushing:- Major problem confronting the investigation relationship between tooth brushing & ECC is methodological tissue of assessing frequency of brushing, quality of plaq removal, actual level of oral hygiene.

5) salivary factors:- Main host defense against dental caries clearance of food & buffering of acid generated by the den plaque mediate adhesion & colonization as bacteria on toot surface adid to elimination of bacteria.

6) Sugars:- Sucrose, glucose & fructose in fruit juices & vitamin as well as solids are probably main sugars associated with Sucrose only substrate used for bacterial generation of the plaque dextran. Increased sucrose intake increase acidity.

7) Oral clearance of carbohydrates:- sleep time consumption o sugar is another common characteristic increase length of contact between plaque & substrate characteristically localized to maxillary primary incisors.

8) Bovine milk:- Milk decreases solubility of enamel & resul have been extended by intraoral cariogenicity test protect by milk appear to work are decreasing demineralization o enamel increasing calcium & phosphate concentration in pl

9) Human milk:- compared to bovine milk, human breast has lower mineral content, higher conc. of lactose & less pr

10) Fluorides:- At low conc. can affect demineralization proc by decreasing rate of bacterial dissolution enhance deposi by fluoridated

Secondary etiological factors:-

1) Immunological factors:-
- Hard tissue are immunologically inactive, host defense mechanism involved in dental caries is centered on prevention of the colonizatn & pathogenin activity of cariogenic bacteria.
- Immune mechanisms include specific immune factor derieved from saliva / serum.
- secretory immunoglobulin A may inhibit bacterial adherance or agglutination as well as neutralization of the bacterial enzymes.

2) Tooth maturation defects:-
- Tooth is most susceptible to caries in period immediately after eruption & prior to final maturation.
- Combination of recently erupted immature enamel in environmental cariogenic flora with frequent ingestion of fermentable carbohydrate.

3) Race & ethnicity:-
- Increased risk of that could be associated with cultural norms including concern for oral health.
- prenatal diet that contribute to enamel hypoplasia minorities may experience significant barrier to dental care including cost of care & availability of accessible service.

4) Acid fruit drink:-
Acid in fruit juices & soft drinks may decrease oral pH pressure of sugar in drink. this fall in pH likely to enhance fermentation carbohydrates cause more profound enamel demineralization.

5) Stress:-
Stress affects immunology, coping skills or preventive oral health behaviours.

- Early childhood caries
- chronic disease management aims to sustain oral health in lon
This includes parent engagement to facilitate & promote preve
measures while encouraged the identification & reduction o
individual risk factors.
- Active surveillance emphasizes careful monitoring of caries p
& prevention programme in children with incipient lesions.
- Minimal intervention approaches includes caries arrest with
diamine fluoride, interim therapeutic restorations that
temporarily restore teeth in young children until a time
traditional cavity preparation & restoration is possible
use of Hall-Style crowns.

Rampant caries :-

- Massler defined rampant caries as suddenly appearing wide
rapidly spreading, burrowing type of caries resulting in e
involvement of pulp & affecting those teeth which are us
regarded as the immune to decay.
- Winter et al (1966) defined rampant caries as caries of a
onset involving many or all teeth in areas that are us
not susceptible. It is associated with rapid crown destru
with frequent pulpal involvement.

- Etiology :-

A) Salivary deficiency :-

1) Due to radiation therap

2) In stressed children who are trained

3) Xerostomia → acidogenic bacteria, dental plaque, poor
structure susceptible to dissolution, Genetical, Habits.

- feeding sweetened milk through bottles.
- sweetened pacifiers.
- Nursing child at night for long time.
- sucrose in diet.

B) Physiological factors:-

- Emotional disturbance, suppressed emotional fear this cause.

- Clinical features:-

The initial lesion -

- Labial surface of maxillary incisor, close to the gingival margin as a whitish area of decalcification of pitting of enamel surface shortly after eruption.

Soon these lesions:-

- Pigmented to light yellow.
- Extend laterally to proximal surface.
- Extend downward to incisal edge.

Advanced stages:-

- Lesions extends tooth circumference leading to fracture of crown on minimal trauma.
- Decrease in salivary flow rate during sleep & pooling of sweet fluid around tooth like milk, vitamin C syrup, sweetened fruit juices, carbonated drinks result in highly cariogenicity.
- The condition also associated with breastfed infant having prolonged feeding habit or whose pacifiers are frequently dipped in honey sugar syrups.

- Teenagers / Adolescents - Buccal & lingual caries of premolar & molar & proximal & labial caries in mandibular incisors.

• Management:-

A) Initial treatment -

- Provisional restoration.
- Diet assessment.
- Oral hygiene instructions.
- Home & professional final treatment.
- Decreased frequent sucrose consumption with meal.
- Parents should be instructed to record amount & the quantities of foods & beverages consumed during & with meals for three consecutive days.
- Introduce proper brushing technique to patient, under 8 yr Fones / circular scrub technique 11 to 12 years - Bass techn

B) Systemic & topical fluoride treatment (0.3 to 0.7 ppm)

1) Dietary fluoride supplements -

- 0.25 mg F / day for 2-3 yr.
- 0.5 mg F / day for 3-12 yr.

2) Operator applied -

- APF topical soln / gel 1.23% fluoride applied 4 times a for 0-2 years.
- APF topical solution / gel 1.23% fluorides 4 times a day for 2-3 years.
- APF topical solution 1% gel, 23% fluoride 4 times a day for 3-13 yr & >

3) Self applied -

- Self application of gel tray daily for 4 weeks then cont with daily F rinse [0.05% NaF] for 3-13 yrs & >13 yr.

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Age specific prevention of rampant caries :-

1) 0-5 years
- oral hygiene instructions to patients.
- Tooth brushing with prenatal supervision.
- 6 months recall.

2) 5-12 years
- Toothpaste
- Fluoride tablets upto 8 yr if in area w/o water fluoridation
- Mouth rinse.
- Professional topical fluoride application every 6 months.

3) 12 years & above.
- Interdental cleaning with floss.
- Sealants.

Rampant caries	Nursing bottle caries
1) Acute, widespread, rapid	1) Specific form of rampant caries
2) All ages	2) Infants, toddlers
3) Primary, permanent dentition	3) Primary dentition involved.
4) Etiology - multifactorial.	4) Bottle feeding, pacifiers dipped in honey, at will breast feeding
5) T/t - pulp therapy	5) T/t - Topical fluoride.
6) Prevention - dental health education	6) Prevention - dental health education
7) Acute generalized spread of caries & pulpal involvement.	7) Acute generalized spread of caries & pulpal involvement

PULP & PERIAPICAL DISEASES

- Dental pulp occupies centre of each tooth & consist of soft connective tissue.
- Primary teeth have 20 pulp organ their shape confines to roo
- Pulp disease -
- Dental pulp responds to changes in environment in same wa as any other loose connective tissue.

• Etiology of pulp diseases -

- Most common cause of pulp & periapical disease is presen of micro-organisms within involved tooth.

• Other factors :-

A) Physical -

1) Mechanical - Trauma
- Pathological
- Barometric changes
- Crack through body of tooth.

2) Thermal - Heat from tooth preparation.
- Exothermic heat from setting of cement.

3) Electrical - galvanic current.

B) Chemical - Phosphoric acid
- Erosion
- Acrylic monomer, etc.

C) Bacterial - Toxins associated with caries
- Microbial colonization.

Clinical classification of pulp disease -

1) Inflammatory disease of pulp -
- Acute reversible pulpitis.

-chronic reversible pulpitis
-Irreversible pulpitis
① symptomatic irreversible pulpitis
② Asymptomatic irreversible pulpitis
③ chronic hyperplastic pulpitis

2) Pulp degeneration-
① calcific degeneration
② Fibrous degeneration.

3) Pulp necrosis.

classification of periradicular diseases:-
1) symptomatic periradicular disease -
- Primary symptomatic apical periodontitis
- seconday symptomatic apical periodontitis
- symptomatic alveolar abscess.

2) Asymptomatic periradicular disease -
- Asymptomatic apical periodontitis
- Asymptomatic alveolar abscess.
- Condensing osteitis

3) Histological classification:-
- Apical granuloma - apical abscess - apical cyst.

Reversible pulpitis:-

Pulp with reversible pulpitis has mild inflammation & is capable healing once the irritating stimulus has been removed.
ain is felt only when a stimulus is applied to tooth.
in ceases in few seconds or immediately upon removal of mulus. This is due to movement of dentinal fluid. Pulpal tissue. The pain is short & sharp in nature & never ontaneous. No radiographic change is evident.

- Treatment :-

Grossman stated- Best treatment is prevention, removal of th
Stimulus generally is sufficient to allow pulp to return to h

2] Irreversible pulpitis :-

- Pulp is damaged beyond repair & even removal of the
stimulus will not allow its proper healing.
- Pulp generally degenerate progressively causing necros
reactive destruction.
- Classic symptom of irreversible pulpitis is lingering pain
induded by thermal stimuli.
- Initial reaction is very sharp pain to hot cold stimul
followed dull ache or throbbing pain for minutes to
after stimulus is removed.
- Pain increases on bending or lying down position.
- Spontaneous pain is another hallmark feature.
- If periapical tissue is involved, tooth is tender on percus
- Radiographs are not useful in diagnosis but helpful in
identifying possible cause of disease.

- Treatment :- Pulpectomy.

3] Chronic Hyperplastic pulpitis [Pulp polyp] :-

- Hyperplastic pulpitis is productive inflammatory response of
- Involve chronically inflammed young pulp, widely exposed
by caries on its occlusal aspect.
- Characterized by proliferative growth of inflammed conn
tissue rising out of carious crown.
- Tissue is firm, insensitive to touch & occassionally may
cause mild discomfort during mastication.

often covered with epithelium, it resembles pyogenic granuloma of gingiva from which it may easily differentiate.
- Tooth respond to pulp testing which is often delayed.
- No significant radiographic changes

- Treatment :-
- Bodily decayed that restoration is impossible, extraction indicated
- Tooth can be restored, pulpectomy or endotherapy are recommended prior to restoration.

4] Dento alveolar abscess :-
- Symptomatic (acute) apical abscess is an inflammatory reaction to pulpal infection & necrosis characterized by the rapid onset, spontaneous pain, tenderness of tooth to pressure pus formation & eventual sweating of associated tissue.
- Cause - Trauma / chemical / mechanical irritation, bacterial invasion of dead pulp tissue.
- Symptoms - Tenderness of tooth, severe throbbing pain, swelling of overlying soft tissue as swelling extend become more painful & mobile. Pus starts to form beneath swelling, fever, chills, foul breathing, headache & malaise.
- Diagnosis - Clinical examination, subjective history of patient confirmed by means of pulp test & themal test. In an abscess, the concentration of micro-organisms is usually large Streptococci, Staphylococci are remove.
- Treatment - Establishing drainage.
Controlling systemic reaction.

5] Apical periodontitis:-

- It is painful inflammation of periodontal tissue, usually results of microbes spreading from root canal to periapical tissue
- Other reasons include - trauma, irritation to periapical area
- Patient will generally complain of discomfort to chewing
- Sensitivity to percussion is hallmark diagnostic test.
- Tooth not sensitive to hot & cold. Depending on cause of inflammation it may be distinct radiolucency.

• Treatment -

- Determination of cause of relieving symptoms pulpal involvement
- Endodontic treatment is indicated.

6] Periapical abscess:-

- Refers to painful localization of pus in periapical connective tissue
- Characterized by rapid onset, spontaneous pain, pus formation of swelling of associated tissue.
- Depending upon location apices of tooth & muscle attachment swelling will usually develop in buccal vestibule on lingual, palatal or ~~st~~ facial space infection.
- Percussion testing produces response that is usually sensitive
- Palpation| testing may produce a sensitive response.
- Tooth gives negative response to vitality test.
- Radiographically, PDL space may be normal, slightly widened demonstrate distinct radiolucency.

• Treatment -

- Endodontic treatment with drainage & control of systemic manifestations.

PREVENTIVE PEDODONTICS

Oral prophylaxis :- Removal of plaque, calculus & stains from exposed & unexposed surface of tooth by scaling as preventive measure for control of focal irritation.

Plaque :- Dental plaque is defined clinically as structured, resilient, yellow-grayish substance that adheres tenaciously to intraoral hard surface, including removable restoration.

Calculus :- Calculus consists of mineralized bacterial plaque that forms on surface of natural teeth & dental prosthesis.

Stains :- Pigmented deposits on tooth surface are dental stains.

Materia alba :- It is a concentration of micro-organism, desquamated epithelial cells, leukocytes & mixture of salivary proteins, lipids with few or no food practicals & it lacks regular internal pattern observed in plaque.

Oral debris :- Loss of food particles collected above the cervical third & proximal embrasure of teeth.

Fluorides :-

- The term fluorides is derived from word fluore meaning to flow, atomic weight is 19 & atomic number is 9.
- Fluoride is primarily absorbed from stomach.
- In plasma fluoride exists as ionic fluoride & non-ionic.
- Fluoride concentration in most tissue is lower than plasma level exception healthy kidney where because of urine produ tion, on occassion fluoride accumulation may result.
- This is steady state bet^n concentration of fluoride in plasma urine i.e they are parallel to each other very closely.

Metabolism of fluoride

↓

Diet

↓

Digestive tract (faeces)

↓

Circulating plasma fluorides

← Digestive juice
← Milk
← Soft tissue
← Urine.

→ Placenta & foetus
→ Saliva
→ Teeth
→ Bone
→ Sweat

- Water fluoridation:-

- Upward adjustment of concentration of the fluorides ion in public water supply in such a way that concentration of fluoride ion in water may be consistently maintained at 1ppm by weight.
- USPH (1986) fluoride optimum concentration = 0.7 - 1.2 ppm
- Saturation, dry feeder, solution feeder system are equipments

Advantages

- Large no. of people are benefitted.
- Least expensive
- Consumption is regular.
- Systemic & topical effect.

Disadvantages

- Inference with human rights
- Other modes arent considered
- Common source of water
- supply may not be prese

- School water fluoridation:-

Advantages

- Good results in reducing caries.
- Minimal equipment.
- Not expensive.

Disadvantages

- Children do not get benefit un they go to school.
- Not all children go to school in India.
- Amount of water drink can't be regulated.

• salt fluoridation:-

Advantages

- Fluoridated salt is safe.
- Prevent dental caries by systemic & topical action.
- No supervision of set up or distribution of system.
- Low cost.
- Depends on individual acceptance & rejection.

Disadvantages

- No precise control over indicated consumption since intake varies greatly among people.
- International efforts to decrease sodium uptake.
- Lowest when need for fluoride is greatest in early years of life.

• Milk fluoridation:-

- Calcium fluoride, sodium fluoride $Na_2(PO_4)$, disodium silicophosphate are used compounds.
- Less expensive than water fluoridation.

• Dietary fluoride supplements:-

Age	< 0.3 ppm F	0.3-0.6 ppm F	> 0.6 ppm F
1) Birth - 6months	0.0	0	0
2) 6months - 3yrs	0.25 mg	0	0
3) 3yrs - 6 yrs	0.50 mg	0.25 mg	0
4) 6yrs - ≥ 16 yrs	1 mg	0.5 mg	0

• Topical fluoride:-

Professionally applied

- Neutral NaF
- Stannous fluoride
- Acidulated phosphate fluoride
- Amine fluoride
- Fluoride varnish
- Fluoride gas

Self applied

- Tooth brushing dentifrice
- Tooth brushing solutions
- Tooth brushing prophylaxis
- Mouth rinse

1] Sodium fluoride [NaF] :-

- Method of application:-

Cleaning & polishing of tooth

↓

Quadrants are isolated with cotton rolls & teeth are dried thoroughly

↓

NaF is then applied with cotton applicators on one quadrant.

↓

Permitted to dry on teeth for about 4 minutes.

↓

Procedure is required to repeated for the remaining quadrants

↓

Patient is instructed to avoid eating, drinking or rinsing for 30 min, so as to prolong the availability of fluoride to react with tooth surface

↓

2nd, 3rd & 4th applications are given at weekly intervals 6, 7, 11, 13 y

2] Stannous fluoride :-

Stanous fluoride with hydroxyapatite in addition to fluoride & form a new crystalline product stannous fluorophosphate.

↓

Rapid penetration of tin & fluoride in 30 sec therefore continuous reapplication after 15 - 30 sec.

↓

In addition to stannous trifluorophosphate, three more addition products are formed viz stannous hydroxyapatite, CaF_2, calcium trifluorostannate.

3] Acidulated phosphate fluoride :-

After thorough prophylaxis, teeth are isolated with cotton rolls on both lingual & buccal side.

↓

For application of gel, position of patient is upright & provide saliva ejector

↓

place enough gel to fill one third of thorough area of tray so that it is sufficient to cover dental arches.

↓

place loaded tray over arch & squeeze buccal & lingual surface forcing gel between them & allow tray in mouth for 4 min.

↓

Recommended frequency of APF application is semiannual.

↓

After prophylaxis teeth are dried.

↓

DO not isolate with cotton roll as varnish being sticky has tendency to stick cotton.

↓

A total of 0.3-0.5ml varnish is required to cover full dentition

↓

Application is done first on lower arch & then on upper arch with help of single tufted small brush starting proximal surface.

↓

After application patient is made to sit with mouth open for 4 min before splitting.

↓

Patient should be clearly instructed not to rinse or drink anything solid but take liquids & semisolids only till next morning & special emphasis on instructions is needed to maintain contact with varnish & tooth surface for about 18 hr. for prolonging intension between varnish & enamel.

- Fluoride dentifrices :-

) Below 4yrs → not recommended

) 4-6 yrs → once a day with fluoride paste twice without paste.

) 6-10yrs → Twice daily with fluoridated paste without it.

) Above → Thrice daily with fluoridated paste.

- Fluoride Toxicity :-

1] Acute toxicity :- Nausea, vomiting, abdominal pain, excessive salivation, mucosal doses of fluoride at one time, cardiac arrythmia, generalized weakness.

2] Chronic toxicity :- Ingestion of variant doses of fluoride over long time.

- Two types → Dental fluorosis
  → Skeletal fluorosis

- Dental fluorosis :- Developmental disturbance of dental enamel, caused by successive exposure to high concentration of fluoride during tooth development, leading to enamel with lower mineral content & increased porosity.

- It can be hypoplasia / hypomaturation of tooth enamel.

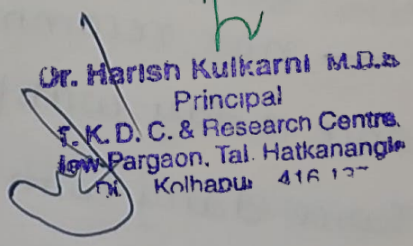
↳ Clinical features :-

- Snow cap phenomenon -
  ① Thin, white striae across enamel surface.
  ② Cloudy, paper white areas are scattered over surface.
  ③ Brown stains.
  ④ Chalky white teeth.
  ⑤ Pits on tooth.
  ⑥ Dark brownish discoloration of tooth.

- Skeletal fluorosis :- may be mild, moderate or severe.

① 3-5 yrs → 500 ppm.
② 5-8 yrs → upto 1000 ppm
③ 8 yrs → above 1000 ppm

} fluoride content in dentifrices.



PIT & FISSURE SEALANTS

Introduction:-
ries potential is directly related to the shape & depth of pit & fissures
e success of fluoride in caries prevention on smooth tooth surfac
ve made caries primarily disease of pit & fissure of teeth.
arrow isolated crevices & grooves that harbour food & micro-
rganisms are the most important anatomical feature leading
o developmental or occlusal caries.

Definition:-
pit & fissure sealant is a material that is placed in pit & fissure
f teeth in order to prevent or arrest the development of caries.
Pit & fissure are enamel tufts, narrow shafts or cracks at some
length whose blind ends are directed more or less towards DEJ.
Pits are small pin point depression located at junction & develop-
mental grooves or at terminal of those groove whereas fissure
are long cleft between cusps or ridges.

Classification:-
1] Based on generations:-
1st generation → polymerized with UV light of 350nm wavelength
Absorbs UV light excessively & prevent complete polymerization.
2nd generation → self cured or chemically cured. Most of
hem were unfilled, increase wear & abrasion resistance.
3rd generation → visible light cured of 430-490nm wavelengt
may be filled or unfilled.
4th generation → Addition of fluoride for added benefit.

According to presence of filler:-
① Filled
② Unfilled.

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C) According to chemical structure of monomer used:-

① Methyl methacrylate [MMA]

② Triethylene glycol dimethacrylate [TEGDMA]

Bisphenol dimethacrylate [BPD]

③ Bis GMA product of Bisphenol A & glycidyl methacrylate

④ Propyl methacrylate urethane [PMU].

D) Based on curing:-

① Autopolymerizing

② Light cure

Indications:-

- Deep retentive pit & fissure, which may cause wedging of explorer
- Stained pit & fissure with minimum appearance of decalcification
- No radiographic or clinical evidence of proximal caries.
- Possibility of adequate isolation.
- Questionable enamel caries in pit & fissures.
- Caries free pit & fissure.
- Patient desire.
- Caries pattern indicative of more than one lesion per year.
- Morphology of pit & risk of caries.
- Routine dental care with active preventive dentistry programme.
- Community based sealant programme.

Contraindications:-

- Well coalesced, self cleansing pit & fissure.
- Radiographic or clinical evidence of interproximal caries, tooth not fully erupted.
- Isolation not possible.
- Life expectancy of tooth is limited.
- Dental caries.
- Lack of preventive practices.

Clinical technique for placement :-

① Tray set up :-

- prior to start of procedure, a tray with all necessary instrument supplies & equipment should be prepared.
- Mouth mirror, slow speed handpiece, explorer, toothbrush, cotton pillers, material isolation device, saliva ejector, arring plrers, etc.

② Isolation of tooth :-

- The tooth should be isolated from salivary contamination by use of rubber dam or by cotton rolls & suction.
- Rubber dam should be used where there is not fully erupted teeth, otherwise cotton rolls are used.
- Moisture control is essential to achieve optimum bond strength.

③ Tooth preparation :-

- There are different methods of enamel surface preparation prior to etching & sealant application. Early concept was to treat surface with slury of pumice & water.

④ Acid etching tooth surface :-

- Apply etchant to tooth surface using a fire brush a minisponge according to manufacture recommendations.
- 37% phosphoric acid is recommended.
- Gently rub etchant applicator over tooth surface including 2-3mm of cuspal inclines & reaching into any buccal or lingual pit & grooves that are present.
- Periodically add fresh etchant to tooth surface.
- Gel form of etchant is preferred as it is more effective & its flow can be controlled.
- Etching time is 15-60 sec.
- Acid etching on surface enamel is removed by etching. In this

plaque & pellides are dissolved fully [illegible] mineral crystal in surface of enamel are also removed, resulting in more reacting surface, increase in surface area & decrease in surface tension that allow resin to wet enamel surface more rapidly.

- This zone is 10± µm in depth.
- The second zone is 20 µm in depth.
- The third zone is quantitative & porous zone with small porosities.

⑤ Rinse & dry etched tooth surface :-

- Rinse etched tooth surface for 30sec with air water spray.
- Dry tooth for 15 sec. with compressed air.
- The dried etched enamel should have frosted white appearance.
- If salivary contamination has occured re-etch for 10 sec & repeat.

⑥ Application of bonding agent :-

- Application of halogenated bonding agent after etching displaces saliva from enamel thereby improving sealant wetting of surface & increase bond strength both in saliva contaminated enamel.

⑦ Application of sealant :-

- The sealant kit have their own dispensers some preloaded that directly apply sealant to tooth structure.
- In mandibular teeth, apply sealant directly & allow it to flow mesially with converse being true for maxillary teeth.
- Then using fine brush / applicators, carry a thin layer of cuspal incline to seal secondary & supplemental fissure.

⑧ Cure the sealant :-

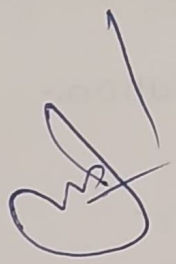
For light cure sealants, polymerization should be initiated, quickly after sealant is placed on the etched surface. to help minimize potential contamination.

(9) Explore the sealed tooth surface & evaluated occlusion:-

- Explore entire tooth surface for pits & voids that may have been seated.
- Evaluate occlusion of sealed tooth surface with articulating paper to determine if any excessive sealant is present.

(10) Recall & re-evaluation:-

- Recall & check patient at subsequent visit.



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GLASS IONOMER CEMENT

- Glass ionomer cement are adhesive tooth coloured, anticariogenic restorative materials which originally used for restoration of the eroded areas.
- The first reliable glass ionomer cement was formulated in 1972 by Wilson & Kent & was known as ASPA.
- It is named glass ionomer because powder is a type of glass & setting reaction & adhesive bonding to tooth structure is due to ionic bond.

Classification:-

A] ISO classification:-

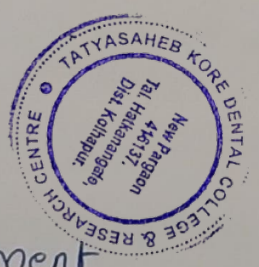
① Luting
② Base & liner
③ Restorations.

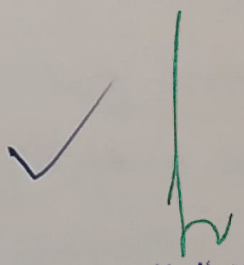
B] According to modifications:-

① Conventional GIC
② Resin modified GIC
③ Metal modified GIC

C] According to application:-

① Luting
② Restorative
③ Base & liners
④ Pit & fissure sealants
⑤ Orthodontic purpose
⑥ Core build up
⑦ Fluoride releasing
⑧ Atraumatic restorative treatment
⑨ Paediatric





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Composition:-

Powder is an acid-soluble calcium fluoro-aminosilicate glass.

1) Silica (SiO_2) → 41.9%
2) Alumina (Al_2O_3) → 28.6%
3) Aluminium fluoride → 1.6%
4) Calcium fluoride → 15.7%
5) Sodium fluoride (NaF_2) → 9.3%
6) Aluminium phosphate ($AlPO_4$) → 3.8%

- Liquid originally was a 50% aqueous soln of polyacrylic acid modern glass ionomer liquids are in form of copolymers

1) Polyacrylic acid – 50%
2) Tartaric acid → 13%
3) Water

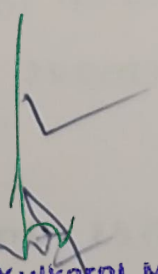
- Mixing time → 4-5 minutes.
- setting time → 7 min. for luting type
  4-5 min for restorative type.

Advantages:-

- Biometric
- Anticariogenic property
- Less mixing time required.
- Better properties due to controlled powder: liquid ratio.
- It adheres well to enamel & dentin.

Disadvantages:-

- Less esthetic compared to composite.
- Less harder than silicate
- Poor mechanical properties.
- Opacity higher than resin.
- Poor wear resistance.



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ATRAUMATIC RESTORATIVE TREATMENT

Definition :-

A dental treatment procedure involving removal of soft mineralized tooth tissue using hand instruments alone followed by restoration of tooth with adhesive restorative material.

Advantages :-

- No need of injection.
- No scary sound making instruments.
- Less chance of bleeding.
- Less expensive.
- Fast procedure for better child co-operation.
- Removal only of caries.

Disadvantages :-

Not long lasting.

Fundamental principles of cavity preparation are not followed.

Low wear resistance & low strength.

Hand fatigue due to hand instruments.

Procedure :-

Tooth is isolated with cotton rolls.

↓

Cleaning of tooth surface

↓

Lesion is slightly widened by hand instrument to remove unsupported enamel using hatchet

↓

Caries are removed by spoon excavator

↓

If necessary, provide pulp protection

↓

Cavity acid etched

↓

Mixed glass ionomer is inserted into cavity

↓

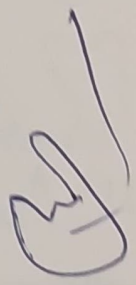
cavity slightly overfilled

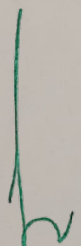
↓

Fit is checked & excess material is removed.

↓

Filling is covered with petroleum jelly.





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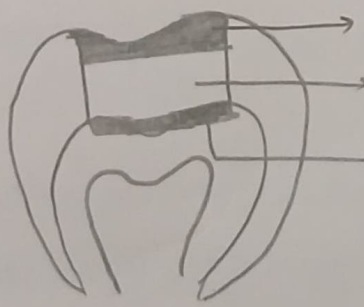
PREVENTIVE RESIN RESTORATION

- They are natural extension of use of occlusal sealants.
- Ingegrates preventive approach of sealant therapy for caries susceptible pit & fissure with therapeutic restorations of incipient caries between the composite resin that occur on same occlusal surface.
- Types of PRR based on extent & depth of caries lesion determin by exploratory, simson has classified them as -

Type A

Type B



Type C

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• Type A :-
- Suspicious pits & fissures where caries removal is limited to enamel.
- Local anesthesia is required.
- Spread of 1/4, 1/2 round bur is used to remove decalcified enamel
- Sealant is placed.

• Type B :-
- Incipient lesion in dentin that is small & confined.
- No LA is needed.
- Appropriate base is placed in areas of dentin exposures, composition is placed & remaining pit & fisiua are covered with sealant.

• Type C :-
- More expensive dentinal involvement & requires restoration with proper composite material.
- Appropriate base is placed over dentin. Pit & fissures are covered with sealant & LA is required.

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NON-PHARMACOLOGICAL BEHAVIOR MANAGEMENT

Means by which dental health team effectively & efficiently perform treatment for child & some time instills positive dental attitu

Factors affecting child's behaviour in dental office :-

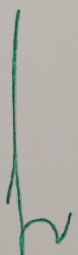
1) Medical history
2) Maternal influence
3) Family & peer influence
4) Dental office environment
5) Growth & development
6) Personal factors
7) Environmental factors
8) Other variables.

Mental attitude :-

1) Overprotective
2) Overindulgence
3) Underaffectionate
4) Rejecting
5) Authoritarian

Classification of child's behaviour in dental office :-

1) Cooperative
2) Tense -operative
3) Outwardly apprehensive
4) Fearful.
5) Stubborn defiant
6) Hypertensive
7) Handicapped
8) Emotionally immature.



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Pre-appointment behaviour modification:-

1) Audiovisual modelling for stimulation of new behaviour.
2) Pre-appointment modelling - contact with parent.

- Communication - may relax the youngster.
- Non-verbal communication - gains patient attention.
- Descriptive phase - emphasize specific co-operative behaviour.
- Signalling - raising hand, finger, etc.
- Use of second language - eg - air wind, rainboat, x-ray machine, etc
- Positive previsit imaginary.
- Tell-show-do - Teach parent of visit & familiarize the patient with instruments.
- Desensitization - Perceived links between stimulus & anxiety response is weakened.
- Direct observation - familarize patient with dental sitting.
- Behaviour shaping - explain necessary for procedure.
- Contigency management - Positive reinforcement
  - Negative reinforcement.
  - Timeout
  - Punishment.
- Externalization - decrease perception of unpleasantness
- Distraction - To relax patient.
  - to decrease anxiety during treatment
  - Placebo effect
  - Audio distraction.
- Assimilation & copying - Behavioural

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15) Parentral pressure or abscence - establish authority.

16) Refraining - for fabricating positive values to replace negative.

17) Memory reconsmucting - Improve patient behaviour
- subsequent dental visits.

8) Relaxation breathing - Benefits fearful patient
- Progressive muscle relaxation.

9) Visual imagery - To establish authority / dreaming

0) Voice control - To establish authority

1) Use of poetry & drawing

2) Hypnosis - To decrease nervousness & apprehension.

) Hand over mouth technique - patient soon begins to gain attention

Protective stabilization - For patient lacking maturity & for physical disable patient.

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CASE HISTORY

Sr. No.	Patient's Name	Date	Signature
1	Karan Sagar Satpute.	9/5/25	
2	Afroza Balgar	5/5/25	
3	Shivany chougule	21/5/25	
4	Yashik Petekar	21/5/25	
5	Shreya Gurav	16/5/25	
6	Samarth Rajesh patil	27/5/25	
7	Kavaya Swapnil kamble	17/10/25	

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WORK DONE

Preventive Treatment

Sr. No.	Work Done	Date	Signature
1	Name:- Fizal Khan	14/5/24	
	OPD No: 8818 Age: 5/M		
	Supragingival scaling is Done		
2	Name: Darshan Bandgar	16/10/24	
	OPD No: 27329 Age: 12/M		
	Supragingival Scaling is Done		

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