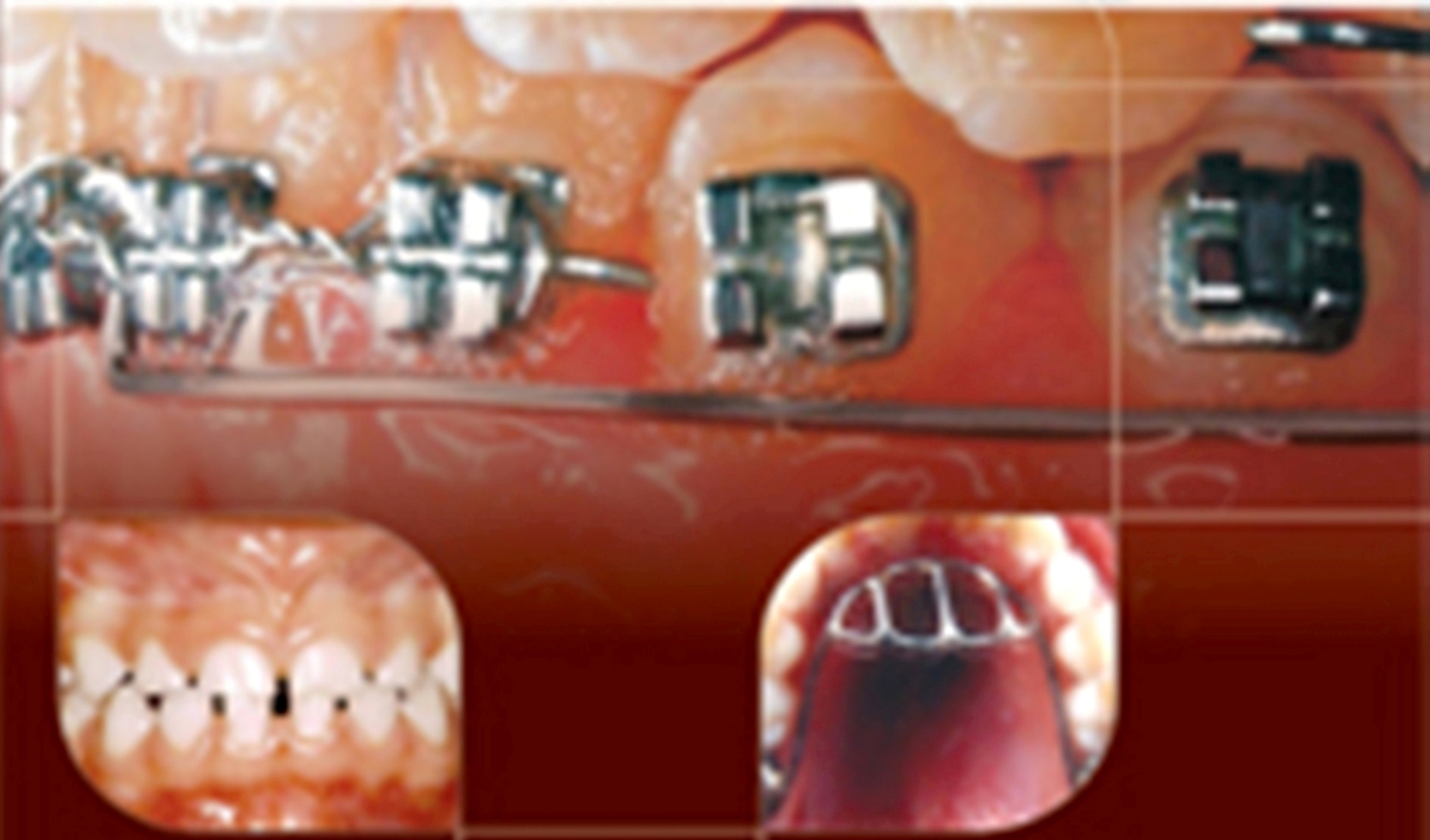
Pedodontics
Practice and Management



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Badrinatheswar GV

Pedodontics

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Practice and Management

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Dedicated to

My grand parents
Late GV Perumal Chetty
Late G Bhagyalakshmi
And
My teachers, family and friends

Foreword

Pedodontics is an ever-developing field of dentistry and every dental student and practicing dentist is required to know its importance and understand the concepts of managing a child patient. Today pediatric dentistry has cast its mantle on every facets of the general dentistry. The book *Pedodontics Practice and Management* gives a complete coverage of all aspects of the pediatric dentistry from the basics to the recent developments in child management. It furnishes a clear coverage of basic anatomy, development, pathology, investigations, diagnosis, and management. Contents of this book are factual, reliable, and scientifically accurate and are based on recent literature. Inclusion of forensic pedodontics, pediatric genetics, and epidemiology will be much informative to the readers. It is well structured with separate sections dealing with each and every aspect in detail. The striking layout, illustrations and photographs reflect the efforts put by the author on making this book.

Dr Badrinatheswar GV is one of the best students from Meenakshi Ammal Dental College, and inspires many in all aspects of his career. I compliment him for authoring a textbook in such a young age and wish all success in his venture.

Dr E Munirathnam Naidu MDS
Former Vice Chancellor
University of Maher
12, Vembuliamman Koil Street
West KK Nagar, Chennai, Tamil Nadu, India

Foreword

I am very happy to write a foreword for this book *Pedodontics Practice and Management* by Dr Badrinatheswar GV a student of mine with a zeal for academic excellence. I am very happy that his ambition of writing a textbook is fulfilled at young age.

I am sure that this book is not only useful to undergraduates but also a reference to postgraduates and for all practicing dental surgeons.

I wish him all success in his endeavor.

Dr D Koteeswaran MDS
Professor and Head
Department of Oral Medicine and Radiology
Meenakshi Ammal Dental College,
Chennai, Tamil Nadu, India

Foreword

I am glad to note that Dr Badrinatheswar GV has authored this book *Pedodontics Practice and Management*. I am sure that this book will be very useful for the dental students as it is informative and has more illustrations and flow charts. In addition it has new chapters like Pedodontics, Orthodontic – Interface, Pediatric Periodontics and Comprehensive Management of Cleft Lip and Palate. I congratulate him on this venture and wish him all success.

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Foreword

I am happy to know that Dr Badrinatheswar GV has compiled this book titled *Pedodontics Practice and Management*. I know him as a hardworking dental student who has topped in the subject of Pedodontics in Final Year from Meenakshi Ammal Dental College and Hospital, Chennai where I had worked as professor and Head of Pedodontics. I knew that he wanted to compile a book on Pedodontics for dental students and I am sure he has presented this keeping in mind the needs of the present-day undergraduate students.

I wish him all success in this project and I hope it will be a useful addition to the needs of undergraduate students of dentistry.

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Preface

The book *Pedodontics Practice and Management* provides with vast information regarding evolving trends and practices in the specialty of pediatric dentistry. I have compiled this textbook covering various topics from introduction, cariology, child abuse, normal and pathologic oro-dental and general health in children with emphasis on growth, behavior management and dental treatment procedures.

Writing this textbook is a dream come true and it has a lot of valuable information and sure to be a worthy dental book for the students of dentistry. This textbook is a compilation of ideas, theories and information about the growing needs for pediatric dental health.

The book follows a sequential diagrammatic representation and enabling easier understanding of the principles and techniques involved in treating a child.

More information on types, indicators and prevention of child abuse and forensic pedodontics, guidelines by American Academy of Pedodontics is of great value and information to students who do not have access to leading pedodontic journals.

I wish every dental student gets his share of information and his quest to seek more information through this textbook.

Badrinatheswar GV

Acknowledgments

The dental institution from which I graduated had taught me both dentistry and ideals of life, all my teachers and professors have always been an unending great source of inspiration and information, which inspired and influenced me in achieving my dream of writing a textbook in the specialty of pediatric dentistry, a dream come true.

I thank the Almighty for having made me achieve my dream.

I take immense pleasure to thank Dr E Munirathnam Naidu, Dr P Jayakumar, my philosopher and guide Dr A Nandakumar and Professor Dr Douglas Alwyn Luke.

I would take pleasure to acknowledge that Dr Sivakumar Nuvvula for his constant support and encouragement and Dr G Sivakumar, Dr Aruna Mohan, Dr Joe Mathew and Dr Roshini Bhaskaran and all the professors of my college who inspired and stunned me with their knowledge in dentistry and were also a source of motivation in my idea of writing this book either directly or indirectly.

I am thankful to all my basic science professors and teachers for their motivation, support and encouragement I received from them, and a few people worth mentioning include Professor Emeritus Cecilia Isaac, Professor Chandrasekhar, Late Professor Sri Karunyam, etc.

There is a never-ending list of great people who have created a great impact on me in this college, sincere salutations to all and I am in debt to "Meenakshi Ammal Dental College"

Last but not the least, I take pleasure to thank my parents who struggled hard to make me a dentist today, my loving wife who helped me throughout this textbook in writing and converting into electronic format, a source of emotional strength and my loving daughters Sankhya Sree, Sruthi Sree and my friends Dr Sujatha, Dr Ayesha, Dr Kiran Koora, Dr Roshan Rayen.

I am thankful to Shri Jitendar P Vij, Chairman and Managing Director, Mr Tarun Duneja, Director-Publishing of Jaypee Brothers Medical Publishers (P) Ltd, New Delhi for having belief in me and providing me an opportunity to publish this textbook.

Contents

1

Introduction to Pedodontics

MEANING

A child is indeed a lovable gift of god; one of gods best creation; dynamic; benevolent; undergoing gradual and remarkable mental, physical, emotional and developmental changes towards its journey of adulthood.

A child is such a precious gift from god; cherished by every parent and is always given great importance; hence special care is to be given to study the complex nature of multidimensional developmental changes of a child from infancy through adolescence. Development and advent in science and technology has led to advent of specialized sciences for the care of children one such branch is "pediatric dentistry"

Thus pediatric dentistry is one such specialized branch of science aimed at identifying; understanding the complexity of factors involving children and also providing both preventive, comprehensive oral health care from infancy till adolescence and those children with special health care needs.

The term pedodontics is made up of two words the "**pedo**" derived from "**pais**" in Greek means "**child**", "**dontics**" refer to "**study of teeth**". There are various great authors, contributors, researchers who have led to the evolution and development of pedodontics as a specialty in the field of dentistry.

DEFINITION OF PEDIATRIC DENTISTRY

According to Stewart, Barber, Troutman, Wei (1982)

"Pediatric dentistry is the practice and teaching of comprehensive preventive and therapeutic oral health care of child from birth through adolescence. It is constructed to include care for special patients who demonstrate mental, physical or emotional problems".

According to American Academy of Pediatric Dentistry (1999)

"Pediatric dentistry is an age defined specialty that provides both primary and specialty comprehensive preventive and therapeutic oral health care for infants and children through adolescence including all those with special health care needs".

Thus pediatric dentistry is a study of comprehensive oral and orofacial health care of an individual from prenatal, natal, postnatal till adolescence; also emphasizing on psychosocial complex of a child and identifying child as an individual and assurance of a child's right for complete health care.

MILESTONES IN EVOLUTION OF PEDIATRIC DENTISTRY

1763 Joseph Hurlock's first book on children dentistry.

1764 Robert Bunon's emphasis on importance of primary dentition.

Father of Pedodontics

1926 Detroit pedodontics study club set up

1927 Detroit pedodontics study club renamed as: "**American Academy for Promotion of Dentistry for children**"

1940 AAPDC renamed as "**American Society of Dentistry for Children**"

1947 "**American Academy of Pedodontics**" founded"

1969 "**International Association of Dentistry for Children**" established.

1984 American Academy of Pedodontics renamed as:
"American Academy of Pediatric Dentistry"

Evolution in India

1950 Introduction of pedodontics
Started as a specialty in Government Dental College, Amritsar

1978 Pedodontics included in syllabus for undergraduates

1979 - "**Indian Society of Pedodontics and Preventive Dentistry**"
Established
–Dr B R Vacher is considered as "**Father of pediatric dentistry in India"**

1982 Indian Society of Pedodontics and preventive dentistry becomes
Affiliated to
"**International Association of Dentistry for Children**"

PEDIATRIC DENTISTRY–ITS SIGNIFICANCE

Pediatric dentistry is an age defined specialty indeed, that supports and emphasizes the fact that "**Every child is recognized as an individual and handled with greater care and respect due to a child's multitudinal complexity which makes a child differ from an adult".**

A child differs from an adult in various aspects like

- Physical
- Emotional
- Mental
- Psychological
- Pharmacological dimensions and, hence a child righteously requires a field or branch of science that provides specialized care and attention.

Pedodontic Triangle

Pedodontics triangle is a pictographic representation of patient-parent-dentist relationship; that dictates the importance of involvement of both parent and dentist to provide comprehensive treatment for the patient; i.e. child and hence rightly called as "**Pedodontic triangle or triad**" (Fig. 1.1).

Pedodontic triangle comprises of:

"**Child**" at the apex which signifies that the pediatric patient or child is given the highest priority who is main recipient of oral health care while both the **"Parent"** and **"Dentist"** are placed at either side of the base which signifies equal role played by parent and dentist in providing specialized dental and general care of the child thereby promoting preventive, therapeutic, psychosocial development of a child.

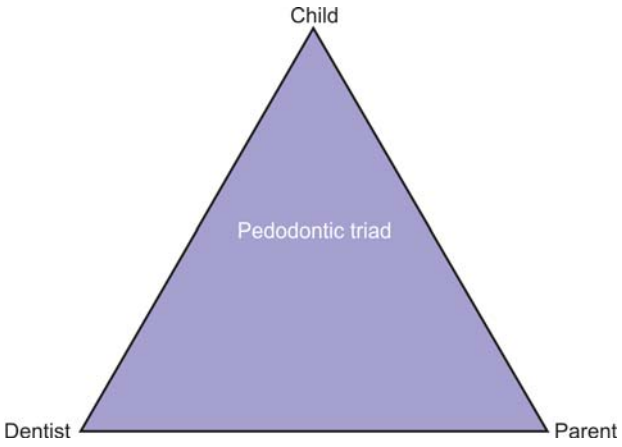


Fig. 1.1: Pedodontic triangle

Pedodontic triangle is commonly expressed as an equilateral triangle signifying equal co-operation, interaction and understanding between child and parent; Dental and child; Dentist and parent to promote optimal success of child's development and oral health care.

EVOLVING TRENDS IN PEDIATRIC DENTISTRY

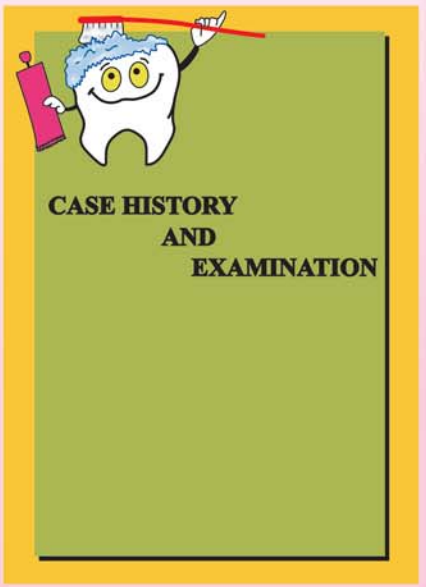
Since inception there has been tremendous increase in trends of pedodontics with day-to-day advances in medical science and technology. More emphasis is laid on preventive and minimal intervention dentistry; and advanced techniques are being advocated. Greater care is advocated in aspect of esthetic; cosmetic dentistry for pediatrics too.

Scope of Pedodontics

Pediatric dentistry includes a variety of facets that are multidisciplinary approach to promote pediatric health care. Such branches include:

1. Preventive dentistry
2. Neonatology
3. Immunology
4. Cariology
5. Oral medicine
6. Dental radiology
7. Restorative dentistry
8. Preventive orthodontics
9. Periodontics
10. Endodontics
11. Child psychology
12. Oral surgery
13. Material science
14. Oral rehabilitation
15. Special care dentistry
16. Allied health sciences
17. Forensic pedodontics
18. Genetics in pedodontics

2



Case History, Examination, Diagnosis and Treatment Planning

OBJECTIVES

This chapter attempts to give an insight into those aspects of history taking, examination, diagnosis and treatment planning that differ in the pediatric patient compared to an adult patient. There are certain considerations that have to be borne in mind with young patients prior to arriving at a definitive treatment plan.

At the first appointment it is necessary to take a detailed history and carry out a full clinical and radiographic examination before a treatment plan can be formulated. It is important to establish the relationship between the child and the adult in order that a valid medical history and consent may be obtained.

Accurate diagnosis can only be achieved by a thorough systematic collection and evaluation of data from both patient and parent. This case history taking and clinical examination is the most critical and vital procedure which provides effective diagnosis of patient's disease/problem and helps the dentist to arrive at an appropriate treatment plan.

SYSTEMATIC APPROACH TO CASE HISTORY DOCUMENTATION

Vital Statistics

Vital statistics hold all useful information about patient for easy retrieval of information, maintenance of records and also for medicolegal communications; storage of data, etc. It includes the following:

Hospital Registration Number/Case Number

- For ease record maintenance
- Billing the individual
- Ease of follow-up for retrieval of data and
- For appointments.

Date

It records the day/month/year of patient. When first reported to clinic and also be used for maintaining and fixing up periodic visits/appointments.

Name

For entry into records with proper first and last name and first name to be included:

- For record maintenance
- To establish a verbal communication and good rapport with patient
- Even nick names can be included to increase a friendly rapport with patient.

Age

Should be recorded with date of birth which aids in calculating exact age of the patient.

- Calculation of age of the patient helps to arrive at various details.

Age of patient is important data because of the following:

- Presence of age related changes of dento-facial structures
- Presence of common age related diseases and lesions
- Useful indications growth spurts, pattern of the individual
- Also helps to use certain behavior management techniques appropriate to that age
- Prescription of drugs when needed
- To compare with the dental, skeletal and mental age of the individual
- To relate with eruption and exfoliation of teeth, etc.

Sex

It is important to differentiate sex related diseases; as certain conditions have sex predilection for, e.g. anorexia is more common in females; hemophilia is more common in males, juvenile periodontitis is common in females

- Timing of eruption sequence is different for males and females
- Healing is faster in males than females
- Probability of traumatic injuries are greater among males than females, etc.

School and Class

- Assess economic status of individual's parent/guardian

- Assess the IQ of the child and his mental age
- For effective communication with the child and assess his/her ability of perception of procedures.

Name of Parent/Guardian; Occupation

- Provides data about socioeconomic status of the parent/guardian which reflects upon the parent/guardians attitude upon dental treatment.

Address

- For communication
- Reflects the socioeconomic status
- Helpful for scheduling appointments for example treatment can be completed within fewer visits for patient's coming from long distance and also scheduling appointments at appropriate timing
- Certain areas are endemic to certain diseases; thus helps in arriving at diagnosis.

Source of Referral

Source of referral is being now emphasized due to increasing trends of multidisciplinary approach in health management. In such cases the queries of the referral should be attended upon first and information about the patient can be obtained from referring physician/consultant and the need for referral.

Chief Complaint

It is the data given either by the child's parent/guardian or the child himself/herself regarding the child's visit to the dental clinic. It is important to note it down in patient's own words which often lead to easy and frank diagnosis. Patient's chief complaint can be a problem of the child which prompted to seek dental treatment or pain, swelling, trauma or referral from other consultants, etc.

History

Taking a good history is the key to accurate diagnosis and to appropriate treatment planning. **Standard forms can be used such as that shown in Table 2.1.** However, these forms do not replace, but rather facilitate, a good interview by acting as an aide-memoir. The time spent taking a thorough history is not only useful in terms of collecting information but is also invaluable in establishing a relationship with the child and the parent.

History of Presenting Illness

The most common presenting illness can be evaluated based on following criterion.

- Type of illness
- Onset of illness
- Duration of illness
- Location
- Quantity, quality, frequency of occurrence
- Aggravating and relieving factors
- Associated symptoms
- For instances of pain- the type of pain experienced will lead to appropriate diagnosis in most of cases.

This information is either provided by parent or patients in the clinic.

Medical History

The importance of taking a thorough medical history lies in its relevance to the safe delivery of routine dental care under both local analgesia and general anesthesia, and in the targeting of preventive advice to children with special needs.

A routine medical history should include:

- Queries about child's recent and past illness and health status
- History of previous medical treatments/surgeries/medication/allergies, etc should be noted
- In case of any previous hospitalization
- Age and cause of admission
- Operations
- Current status should be noted
- A thorough systemic review should be enquired as follows:

i. **Central nervous system:** Epilepsy, Neurological problems, Mental impairment
ii. **Cardiovascular system:** Congenital heart diseases, Subacute bacterial endocarditis, Prosthetic replacements in heart
iii. **Respiratory system:** Asthma, Hay fever, Repeated colds, Rhinitis, Nasal obstructions, etc.

Table 2.1: An example of a standard case history form with some relevant key points

A.	**Social**		
		Name Address Date of birth School Parental occupation	Nick name/preferred name* Age
B.	**Medical**		
	Pregnancy/neonatal	Maternal health Details of birth Childhood illness	Weight, delivery Age, severity
	Systems review	Cardiac Respiratory Hematological Gastrointestinal Diabetes Epilepsy Hepatitis Mental or physical impairment	Subacute bacterial endocarditis risk factors Asthma, hay fever Anemia, bleeding, bruising Bowel habits Control Control
	Medication	Regular prescriptions Recent medication Allergies	Tablet, syrup, inhaler
	Hospitalization	Age and cause of admission Operations General anesthesia	
C.	**Dental**		
	Past history	Regular or irregular attender Previous experiences Experience of local anesthesia Previous co-operation levels	Prevention, restorations, extractions
	Home care	Oral hygiene habits	Frequency of brushing Type and amount of toothpaste Parental assistance
		Dietary habits	Baby bottle habits Favorite snacks and drinks
		Additional measures	Fluoride supplements Water fluoride levels
	Reason for attendance	Routine or emergency Presence of pain any Particular concerns	Duration, nature, relief Color, shape, position of teeth

iv. **Hematological:** Anemia, Bleeding disorders, Bruising, Leukemia, *etc*

v. **Gastrointestinal:** Bowel habits:
- Ulcers
- Gastric bleeding
- Acidity

vi. **Excretory system:** Urinary and renal disorders.

Other things to be noted include conditions like diabetes; hepatitis; jaundice; any acute illness, *etc*.

Prenatal History

Prenatal history includes details of mother's health during the pregnancy like:

- Any nutritional deficiency encountered
- Maternal health status
- Drugs used- some drugs are said to posses teratogenic potential affecting fetal growth and development, e.g. tetracycline

- Any major illness during pregnancy like German measles and leads to cleft lip and palate viral infections
- Diabetes mellitus
- Any trauma/accidents of mother during pregnancy
- Fetal position and any history of previous abortions/ stillbirths, etc.

Natal History

It gives details about child's birth:

- Type of delivery, e.g. forceps delivery
- Presence of any Rh incompatibility may result in tooth discoloration leading to a condition called "Erythro-blastosis fetalis"
- Cyanosis at birth.

Postnatal History

It gives details such as:

- Amount of time the child was breastfed, bottle fed weaning
- Duration and frequency, method of feeding, feeding habits
- Trauma and childhood diseases
- Child's vaccination status (Table 2.2)
- Presence of any habit, if so:
  - Duration
  - Frequency and
  - Intensity to be evaluated
- Behavioral status can be assessed.

Dental History

It is important when treatment planning to try and evaluate both the child's and the parent's attitude to dental treatment. This assessment may be hard to make on the first visit, particularly if the child is in pain or has had an accident. However, gentle questioning concerning past dental experiences can give a good indication of how well the child is likely to cope with future dental treatment knowledge of the attitudes and home habits will facilitate realistic planning of a patient's management.

Likewise, it is useful to anticipate the parent's attitude towards restoration of the primary dentition, it is unrealistic to expect all parents to attach equal importance to dental health and it is appropriate to modify a treatment plan to accommodate these differences.

Table 2.2: Vaccination schedule—Recommended by Indian Academy of Pediatrics

Age	*Vaccine*
Birth	BCG Oral polio vaccine—1st dose Hepatitis B vaccine—2nd dose
6 weeks	DPT—1st dose Oral polio vaccine—2nd dose Hepatitis B vaccine—2nd dose
10 weeks	DPT—2nd dose Oral polio vaccine—3rd dose
14 weeks	DPT—3rd dose Oral polio vaccine—4th dose
6-9 weeks	Oral polio vaccine—5th dose Hepatitis B vaccine—3rd dose
9 months	Measles vaccine
15-18 months	MMR (Measles, Mumps, Rubella) DPT—1st booster dose Oral polio vaccine—6th dose
5 years	DPT—2nd booster dose Oral polio vaccine—7th dose
10 years	TT (tetanus)—3rd booster dose Hepatitis B vaccine—booster dose
15-16 years	TT (tetanus)—4th booster dose
Optional vaccines	Typhoid fever vaccine *Haemophilus influenzae* type-B vaccine Hepatitis A vaccine Varicella (chickenpox) vaccine

Family History

- Provides information of any familial/hereditary diseases if any
- Number and age of siblings
- Health status of the parent, etc.

Personal History

History of oral habits:

- Type of habit
- Duration
- Frequency and intensity, etc.

Oral hygiene:

- Frequency and method of brushing
- Type of brush used and its frequency of replacement
- Use of fluoridated or non-fluoridated dentifrices, etc
- Assessment by recording debris and calculus index.

Dietary status:

- A detailed diet chart has to be recorded
- Usually a 24 hour diet chart is recorded
- Indicates
  - Cariogenicity of diet
  - Frequency of snacking
  - Nutritional deficiency if any

Clinical Examination

It is very important during the clinical examination to bear in mind the child as a whole. The dental practitioner may well see a child more often than any other medical professional and is, therefore, in an excellent position to notice any changes in a child's general health.

Clinical examination begins as soon as the dentist meets the child when an overall impression of the child's health can be formed. Table 2.3 is an example of an examination sheet that can be used in young patients.

General Examination

It is often useful to monitor a child's growth pattern and evaluation of the patient begins with the first appearance of the child and parents themselves. Following factors are to be assessed in general examination:

- Height
- Weight
- Posture
- Gait
- Co-ordination
- Speech.

Local Examination

Extraoral examination

In extraoral examination following features have to be noted:

a. Shape of the head
   - Mesocephalic

Table 2.3: An example of a standard examination sheet with some relevant key points

A.	**Extraoral**		
	General appearance	Height/weight Posture Co-ordination Malaise/fever	
	Head and neck	Skull Hair Ears Eyes Face Temporomandibular joint	Asymmetry Quality, quantity Tags, hearing aids Sclera Lacerations, scars bruises, swellings Opening, deviation, discomfort
B.	**Intraoral**		
	Soft tissue	Mucosa Inflammation Swelling White/red patches Ulcers Frenal attachments	Palate, tongue, cheeks Size, location, texture Number, site, size
	Periodontal tissues	Oral hygiene Inflammation/bleeding Attached mucosa Pocketing/dehiscence Tooth mobility	Plaque, calculus ± deposits Color, stippling
	Occlusion	Skeletal pattern Molar relationship Overjet/overbite Crowding	
	Teeth	Number	Hypodontia, supernumeraries

- Dolichocephalic or
- Brachycephalic

b. Shape of face
- Mesoprosopic
- Euryprosopic
- Leptoprosopic

c. Symmetry of the skull
d. Facial symmetry
e. Facial profile – Straight, concave, convex
f. Facial divergence – Orthognathic, anterior divergent, posterior divergent
g. Hair – Quality, quantity
h. Ears – Tags, hearing aids if any
i. Eyes – Sclera
j. Face – Lacerations, cars, bruises, swellings
k. Temporomandibular joint: Functional examination of TMJ by palpation of each condylar head in various positions is highly recommended to diagnose any:
   - Deviations
   - Path of opening and closure
   - Discomfort
   - Tenderness
   - Auscultation of TMJ might give information about any clicking sound if any present. Which may be early or initial clicking, intermediate clicking or late/terminal clicking?
l. Lymph nodes: Palpation of all lymph nodes of head and neck recommended. However commonly involved submandibular and submental lymph nodes are given importance.
m. Lips:
   - Competence/incompetence
   - Any defects
   - Dryness
   - Texture
   - Tonicity
   - Any ulcers, vesicles, fissures, crusts, abrasions are to be noted if present.

Intraoral examination:

i. Soft tissue examination:
- **Gingiva** - Change in color
  Consistency
  Contour
  - Abnormal swelling
  - Inflammation
  - Abnormal discoloration
  - Bleeding
  - Ulcers
- **Tongue** - Size, shape, color, movements
  - Frenal attachment
  - Any abnormalities if present
- **Mucosa** - Palate, cheeks, tongue.

ii. Hard tissue examination:
- **Teeth** - Number of teeth present
  - Size and shape
  - Teeth numbering is done according to FDI scoring as follows:
- **Primary teeth**

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

- **Permanent teeth**

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

The first number denotes the quadrant and second number denotes the tooth

Primary teeth quadrant

5	6
8	7

Permanent teeth quadrant

1	2
4	3

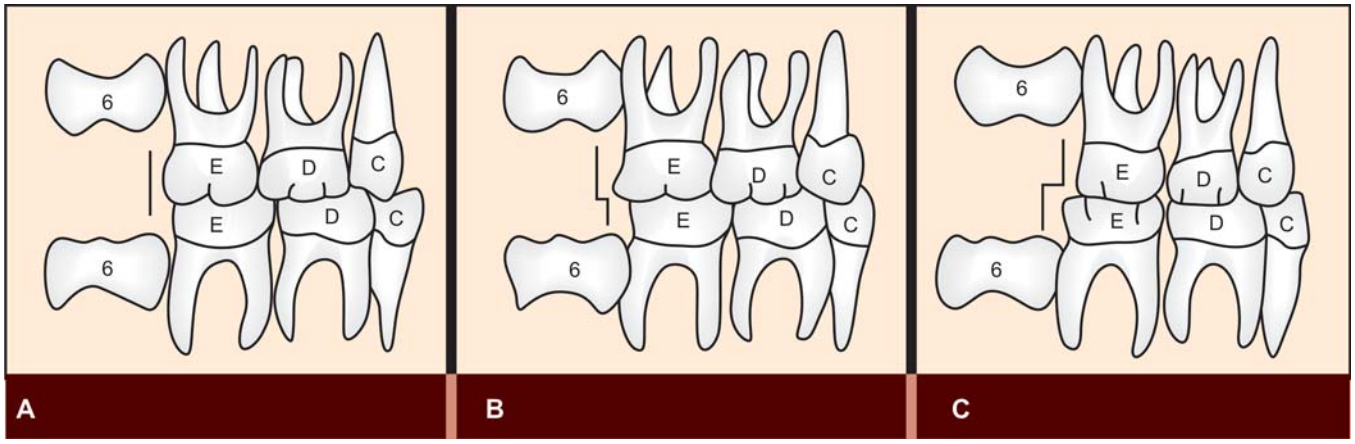
Occlusion

Molar relationship (Fig. 2.1)
- Permanent teeth classified based on Angle's class I
- Deciduous teeth classified based on Baumes terminal plane (distal surface of second deciduous molars)
- Flush terminal plane
- Mesial step
- Distal step

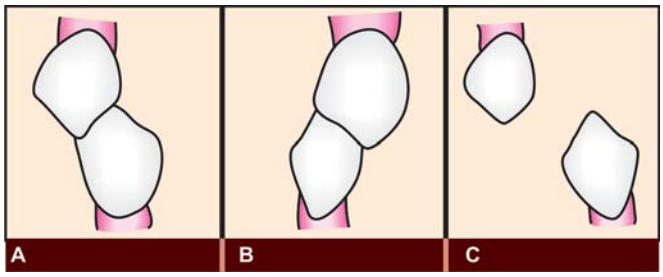
Canine relationship (Fig. 2.2)

Incisor relationship:

i. Overjet (Fig. 2.3)
- Horizontal overlapping of upper and lower anterior teeth
- Measured from labial surface of lower anteriors to incisal edge of upper anteriors



Figs 2.1 A to C: Molar relationship—Primary teeth. (A) Flush terminal plane, (B) Mesial step, (C) Distal step



Figs 2.2 A to C: Canine relationship. (A) Class I, (B) Class II, (C) Class III

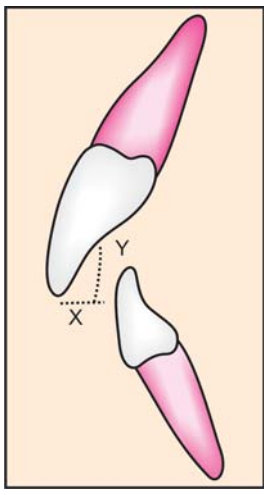


Fig. 2.3: Incisor relationship. X—Overjet Y—Overbite

- Expressed in either 'mm' or '%'
- Normal overjet is 2-3 mm.

ii. Overbite:

- Vertical overlapping of upper anteriors over lower anteriors
- Normal 2-3 mm
- Expressed either in 'mm' or '%'
- It is deep bite when vertical distance is greater than 3 mm
- Open bite is described when there is no contact between incisors.

Curve of Spee

Deciduous teeth normally presents with a steep curve of Spee.

Spacing

Presence of spaced or non-spaced dentition is suggestive of orthodontic management that may be needed in future. Other factors to be assessed in hard tissue examination includes:

- Number of decayed teeth
- Incipient caries lesions
- Rampant caries
- Grossly decayed teeth should be identified
- Staining either extrinsic or intrinsic
- Any tooth dislocation, rotation, missing teeth have to be noted
- Existing restorations.

Provisional Diagnosis

It is a general and primary/preliminary impression of a clinical situation without any appropriate laboratory investigations.

Investigations

Percussion

- Reveals the periodontal status of the tooth
- Percuss lightly either with the handle of mouth mirror or with index finger.

Positive response on vertical percussion indicates:

- Apical periodontitis
- Irreversible pulpitis
- Cuspal fracture, etc.

Positive response on lateral percussion:

- Lateral space inflammation

Radiographic Examination

As per requirement IOPA, Bitewing and occlusal radiographs, OPG's are recommended.

Pulp Testing

The clinical condition of pulp can be evaluated by thermal stimuli, percussion and vitality testing in adults but their value in children is questionable due to difference in perception of pain in children and pain anxiety may lead to false responses and interpretations.

Conventional Pulp Testing Methods

Thermal test—cold and heat test:

- Use of ethyl chloride spray/ice sticks/air blast/hot water spray/heated gutta percha stick are advocated.

Electric pulp testing

- Use of odontometer
- Does not provide information on health, integrity or vascular supply of pulp.

Other novel methods of pulp testing includes

- Laser Doppler flowmetry
- Pulseoximetry in evaluation of vitality
- Dual wavelength spectrometry.

Final Diagnosis

It is the final ultimate and concrete evaluation and confirmed diagnosis of the clinical situation after a thorough and systematic analysis of collected data with adequate laboratory proofs and findings.

Treatment Planning

Having obtained a full history, undertaken a complete clinical examination, and carried out necessary investigations the clinician is armed with all the information required to draw up an appropriate treatment plan. It should be explained in detail to the parent and also to the child in his/her understandable manner to gain his/her acceptance and co-operation. Following factors to be included:

a. Treatment required of their child:
   - Restorative
   - Surgical
   - Preventive or
   - Therapeutic
b. Time duration, number of visits
c. Total cost of the dental treatment
d. Any precautionary measures and home care as per the case.

Definitive Treatment Planning

Definitive treatment planning is achieved based upon 5 basic phases:

Medical phase

- Patients with positive medical history are referred to related consultants for thorough medical evaluation and their consent
- Drug dose modification is done as and when needed.

Systemic phase

- Advocate required pre-medication
- Antibiotic prophylaxis
- Drug modifications are done.

Preventive phase

- Oral prophylaxis, fluoride therapy
- Diet counseling
- Sealant therapy
- Orthodontic intervention to prevent malocclusion, etc.

Corrective phase

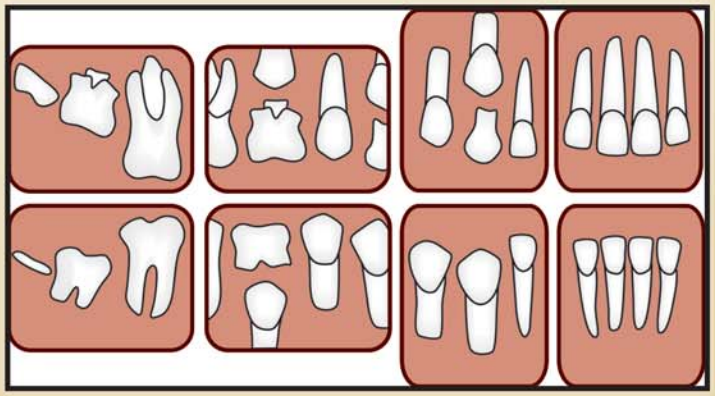
- It's a restorative or treatment phase
- It includes procedures like restorations,
  - Stabilization,
  - Extractions
  - Minor surgical procedures
  - Orthodontic management
  - Aesthetic management, etc.

Maintenance phase

Completion of the initial course of treatment is merely the start of a long-term relationship with the patient. Reassessment and recall are essential to the treatment planning process.

- Establishment of exact time and treatment recall.
- Patients at high-risk recalled at 2-3 months interval will low-risk individuals at 6 month's period.
- Establish appropriate home care program that will work for the particular child and the parent.
- Check the effectiveness of home care program at recall visits and advice necessary reinforcements as per case dictates.
- Also helps in patient and parent motivation.

3



Radiology in Pediatric Dentistry

INTRODUCTION

Although it is often neglected, roentgenography is the most important adjunct to the successful practice of dentistry for children, as an aid to the dentist; roentgenography is one of the most important diagnostic tools available for detection of disease and the interruption of malocclusion. Radiographs are essential if we are to treat children successfully. Evidence indicates that unless carious lesions are discovered early, the primary teeth will not be retained until normal exfoliation.

Early diagnosis of caries prevents the pediatric patient from experiencing dental pain, extraction and emotional stress. In addition eruptive or developmental problems can be discovered and early treatment may reduce the need for prolonged orthodontic procedures. Some restorative procedures require an accurate registration of pulpal outline that only a radiograph can reveal.

DIAGNOSTIC VALUE

Children have perhaps a greater need for roentgenography than do adults; for, in children, one is greatly concerned in every instance, with the problems of growth and development and the factors that alter them:

- Adjunct to diagnosis
- Patient follow-up and record keeping in traumatic injuries cases
- Two-dimensional image of a three-dimensional object and that changes in the soft tissues are not shown
- Facts provided by roentgenograms pertain mostly to the calcified structures.

Radiographic examination of children should occur only after the following conditions are met with:

- Obtainment of previous radiographic history (To asses the availability of recent films from another dentist)
- Clinical examination of the patient
- Determination of appropriate number and size of the films required
- Placement of lead apron and thyroid collar on the child (patient) (Sikorski and Taylor, 1984) and also parent
- Avoid attempts for retakes or duplicate views; it is essential that diagnostic radiographs be obtained on first attempt.

Radiographs of acceptable diagnostic quality should meet particular standards:

1. Follow the appropriate guidelines for the correct:
   a. Size
   b. Number
   c. Type of film used at the appropriate time for the child's stage of occlusion.
2. Include distal surface of canine to mesial surface of the most distal tooth for bitewing radiographs.
3. Include an unobstructed view of all contact areas (for bitewing radiographs) and periapical region (for periapical radiographs).
4. Not be cone cut, overlapped, elongated, or shortened and free of any developing and processing errors.
5. Be of proper exposures.

Myers (1984) lists why children are at higher risk for radiation exposure than adults:

- The tissues of the child are in growth period and are more sensitive to radiation
- Children have a longer life span with greater susceptibility to tumors
- The effects of radiation are cumulative
- Because of their smaller structure, children are closer to the central beam
- Because of carious activity, children may have increased frequency of radiographs.

METHODS OF REDUCING RADIATION EXPOSURE

1. Exposure time can be reduced when there in an increase in:
   - Film speed (E-speed)
   - Kilo voltage
   - Milliamperage are increased.
2. Use of rare-earth screen film system.
3. Use of xero-radiography.
4. Use of long rectangular collimator.
5. Wearing lead apron and thyroid collar.
6. Good dark room procedures thus avoiding retakes
7. Use of panoramic radiography.

Table 3.1: ADA, US Food and Drug Administration. The selection of patients for dental radiograph examination

	PATIENT AGE AND DENTAL DEVELOPMENTAL STAGE				
TYPE OF ENCOUNTER	**Child with Primary Dentition** (prior to eruption of first permanent tooth)	**Child with Transitional Dentition** (after eruption of first permanent tooth)	**Adolescent with Permanent Dentition** (prior to eruption of third molars)	**Adult Dentate or Partially Edentulous**	**Adult Edentulous**
New patient* Being evaluated for dental diseases and dental development	Individualized radiographic exam consisting of selected periapical/Occlusal views and/or posterior bitewings if proximal surfaces cannot be visualized or probed. Patients without evidence of disease and with open proximal contacts may not require a radiographic exam at this time	Individualized radiographic exam consisting of posterior bitewings with panoramic exam or posterior bitewings and selected periapical images	Individualized radiographic exam consisting of posterior bitewings with panoramic exam or posterior bitewings and selected periapical images. A full month intraoral radiographic exam is preferred when the patient has clinical evidence of generalized dental disease or a history of extensive dental treatment		Individualized radiographic exam, based on clinical signs and symptoms
Recall patient* with clinical caries or increased risk for caries**	Posterior bitewing exam at 6-12 months intervals if proximal surfaces cannot be examined visually or with a probe			Posterior bitewing exam at 6-18 months intervals	Not applicable
Recall patient* with no clinical caries and no increased risk for caries**	Posterior bitewing exam at 12-24 months intervals if proximal surfaces cannot be examined visually or with a probe		Posterior bitewing exam at 18-36 months intervals	Posterior bitewing exam at 24-36 months intervals	Not applicable
Recall patient* with periodontal disease	Clinical judgment as to the need for and type of radiographic images for the evaluation of periodontal disease. Imaging may consist of, but is not limited to, selected bitewing and/or periapical images of areas where periodontal disease (other than nonspecific gingivitis) can be identified clinically				Not applicable
Patient for monitoring of growth and development	Clinical judgment as to need for and type of radiographic images for evaluation and/or monitoring of dentofacial growth and development		Clinical judgment as to need for and type of radiographic images for evaluation and/or monitoring of dentofacial growth and development. Panoramic or periapical exam to assess developing third molars	Usually not indicated	
Patient with other circumstances including, but not limited to proposed or existing implants, pathology, restorative/endodontic needs, treated periodontal disease and caries remineralization	Clinical judgment as to need for and type of radiographic images for evaluation and/or monitoring in these conditions				

* Clinical situation for which radiographs may be indicated
** Factors increasing risk for caries

GUIDELINES FOR PRESCRIBING RADIOGRAPHS (TABLE 3.1)

The decision to make radiographs is based on thorough evaluation and examination of the patient. Radiographs should be made only when there is an expectation that disease is present or when undetected condition left untreated could adversely affect the patient's dental health. Therefore, the decision to use ionizing radiation is based upon professional judgment. Two important considerations when deciding whether to perform a radiographic examination for children as summarized by Nowak et al are as follows:

1. Stage of dentition development.
2. Risk of dental caries.

Development of Dentition as Criterion

Primary Dentition

If the proximal surfaces cannot be visually and tactile inspected and the child can be expected to co-operate, then dental radiographs should be made to determine the presence of interproximal caries. If all proximal contacts are visualized, then radiographs are not indicated.

Early Transitional Dentition

After the eruption of permanent first molars or permanent mandibular incisors, or both radiograph are taken to evaluate the presence of:

a. Interproximal caries
b. Developmental teeth anomalies
c. Pathological conditions of hard and soft tissues of mouth, jaw and associated structures.

Early Permanent Dentition

Radiographs are taken to evaluate the same tissues as in the early mixed dentition and to check the position and developmental status of the third molars.

Risk of the Patient for Dental Caries as Criterion

a. High-risk of dental caries:
   - Poor oral hygiene, fluoride deficiencies, prolonged nursing (bottle or breast) high-carbohydrate diet, poor family dental health, developmental enamel defects, developmental disability and acute or chronic medical history and genetic abnormality.
   i. Should have bitewing radiographs made as soon as posterior primary teeth are in proximal contact.
   ii. If interproximal caries detected, follow-up radiographs are indicated semiannually until the child is caries free and therefore classified as having low-risk for dental caries.

b. Low-risk for dental caries.

A child who has low-risk for dental caries may be defined as **"Normal, healthy, asymptomatic patient, exposed to optimum levels of fluoride, who performs daily preventive techniques and consumes a diet with few exposures to retentive carbohydrates between meals".**

i. Posterior bitewing radiographs should be made for the low-risk patients with closed proximal contacts
ii. If no evidence of caries then radiographs may be retaken once in every 12-18 months if primary teeth are in contact or after up to 24 months if permanent teeth are in contact
iii. Bitewing radiographs may be taken more frequently if the child enters high-risk category.

Types of Findings Anticipated as Criteria

Bitewing radiographs are indicated when a clinical examination discloses posterior tooth contact; bitewing examinations are recommended at the first clinical evidence of caries.

Bitewing radiographs are usually taken every 12-18 month in the absence of dental caries with primary tooth contact or 24 months with permanent tooth contact.

By the time the first permanent tooth has erupted, an anterior occlusal radiograph should be made. This will allow detection of condition as supernumerary teeth, missing teeth and dens in dente.

A radiographic examination that includes the tooth bearing areas of the mandible and maxilla is recommended at approximately the time of early transitional dentition to assess the dental age of the patient and to aid in the early diagnosis of congenital and developmental anomalies (Fig. 3.1).

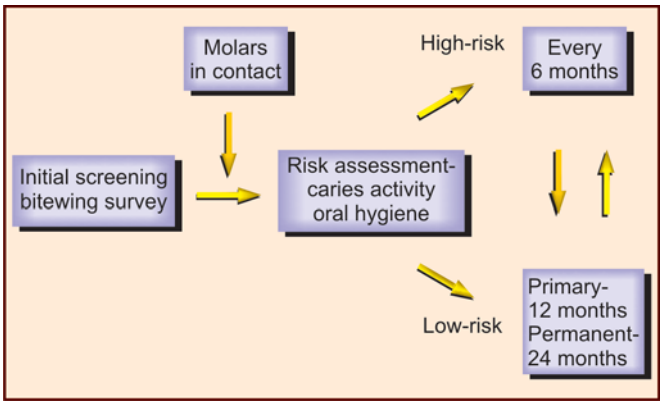


Fig. 3.1: Radiographic indications

CHILD PREPARATION AND MANAGEMENT

Explain the procedure in terms the child can understand use such words as "picture" and "camera" and not "roentgenogram" and "X-ray machine".

In explaining to a young child, it is sometimes wise to bring the "camera" to contact with your own face to help dispel any fares the child may have. Allow the patients to inspect and touch the film packet before it is placed in the mouth. A good idea is to have the X-ray tubes set at the needed angulations and placed next to the child's face, and then all that remains is to gently insert the film.

Tips to Assist in the Radiograph Process

- If a child has a tendency to reject the film, dampen the film packet which takes away some of the taste of the packet.
- Do not insert packet directly but place the film in a horizontal plane. Then, as the film is placed between the tongue and the lingual surface of the teeth, it can be gently rotated into vertical position.
- Before inserting the film; curve it slightly so that it does not impinge on the lingual tissue often, it is also necessary to bend the lower anterior corner to prevent forcing the film into the floor of the mouth. Such forcing is uncomfortable and may cause patient to reject the film.

Radiograph should be employed to supply the following 8 categories of information:

- Incipient carious lesions
- Anomalies
- Alterations in calcification of teeth
- Alterations in growth and development
- Alterations in the integrity of periodontal membrane
- Alterations in supporting bone
- Changes in the integrity of the teeth
- Pulpal evaluation.

TYPES OF EXAMINATION

1. General survey of the mouth
2. Examination of specific areas
3. Special surveys.

General Survey

- Taken as a part of patient's regular visit to the dentist
- Frequency of these surveys should be governed by
  i. Caries susceptibility
  ii. Growth pattern, etc.

Examination of Specific Areas

Specific area examinations consists of examinations for location of bone lesions and objects within the soft tissues, evaluation for multiple roots and pulp canals and examinations of the sinuses and temporo-mandibular joints. In pedodontics, two roentgenograms are important:

i. Cephalometrics
   - To follow the growth and development of the child's skull

ii. Hand wrist roentgenogram
   - Determination of patient's skeletal age.

Special Surveys

When a new patient is seen at the dental office and no previous radiographs are available, it may be necessary to obtain a baseline series of radiographs. These examinations include the following:

Four Film Surveys

- Maxillary anterior occlusal
- Mandibular anterior occlusal
- Two posterior bitewings

Eight Film Survey

- Maxillary anterior occlusal (or periapical)
- Mandibular anterior occlusal (or periapical)
- Right and Left Maxillary posterior occlusal (or periapical)
- Right and Left primary mandibular molar occlusal (or periapical)
- Two posterior bitewing radiographs.

Twelve Film Survey

- Four primary molar-premolar periapical radiographs
- Four canine periapical radiographs
- Two incisor periapical radiographs
- Two posterior bitewing radiographs.

Sixteen Film Survey

- Twelve film survey and the addition of 4 permanent molar radiographs.

Complete Mouth Surveys

Age 1-3 years – Lateral jaw films
- Intraoral film taken as occlusal film in anterior area
- Two bitewings

Age 3-6 years – Six anterior
Four posterior
Two bitewing films

Age 6-12 years – Fourteen film surveys

Age >12 years – Sixteen film survey

TYPES OF FILMS

In radiology; films are broadly classified as intraoral and extraoral films. They are further categorized based upon the criterions like—size (Fig. 3.2), speed, etc.

Based on size intraoral films are classified into:

Intraoral film size	*Measurement*	*Age*
No. 0	-	-
No. 1.0	0.81/1.25 inches	3-5 years
No. 1.1	0.94/1.56 inches	Younger children
No. 1.2	1.22/1.61 inches	Adult sizes
Occlusal film	2.25/3 inches	
Speed films	A, B, C, D, and E	

Based on speed intraoral films are classified into A, B, C, D and E

Contd...

Fig. 3.2: Film sizes

Contd...

Extraoral films

Extra-oral non-screen film used most commonly in pediatric dentistry is 5/7 inch film

- TMJ and lateral oblique view — 1.5 × 7 inches
- Lateral cephalograms, PNS view — 8 × 10 inches
- Orthopantamography — 6 × 12 inches

COMMONLY USED RADIOGRAPHIC TECHNIQUES

Several techniques are commonly used to radiograph a child's dentition. The technique used depends upon the size of the oral cavity, number of teeth and patient co-operation.

Commonly used projections include:

1. Bitewing
2. Periapical
3. Occlusal
4. Panoramic

Radiographic Technique

Available literature strongly suggests that the paralleling principle of intraoral radiography is the technique of choice for periapical radiography, because the images are more anatomically accurate and possess less distortion than radiographs taken using the bisecting angle technique.

Nan Aken, however has demonstrated that even if the film cannot be placed exactly parallel to the long axis of the teeth as long as the film is placed within 20° to the long axis within the beam directed perpendicular

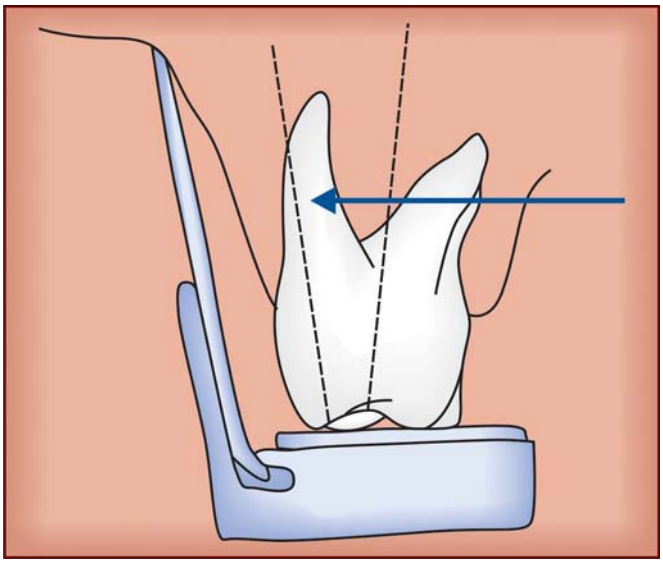


Fig. 3.3: Bisecting angle technique

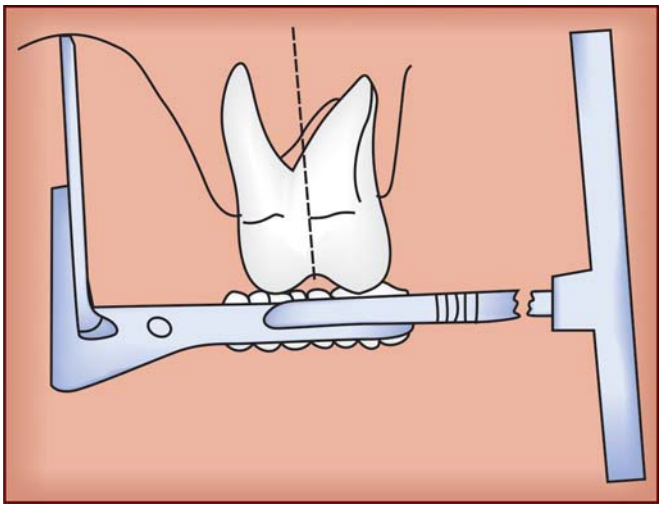


Fig. 3.4: Paralleling technique

to the film, a radiograph will be produced that is clinically superior to bisecting angle radiographs of the same region (Figs 3.3 and 3.4).

Bitewing Radiographs

Indications

- To detect incipient interproximal caries
- To determine pulp chamber configuration and depth of carious lesions
- Record the width of space created by premature loss of primary teeth
- To determine the presence or absence of premolar crowns
- To determine the relation of the occlusal plane to possible tooth ankylosis:

Film sizes used are:

No 0 to 1 – for younger child
No 2 for older children.

Technique (Figs 3.5 and 3.6)

- The head is positioned so that the midsagittal plane is perpendicular and ala-tragus line is parallel to floor
- The inferior edge of film packet is placed in floor of the mouth between tongue and lingual aspect of mandible and the bite tab is placed on the occlusal surfaces of mandibular teeth
- The lower anterior comer of film packet is bent slightly toward lingual to facilitate patient comfort and placement as it lies closer to lingual frenum.
- Ask the patient to close the mouth slowly in centric occlusion
- The central ray enters through the occlusal plane at a point below pupil of the eye
- The vertical angle is +8 to +10 degrees.

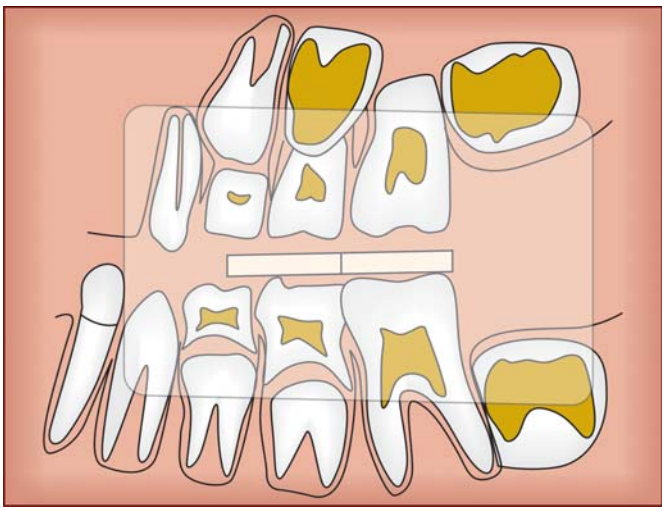
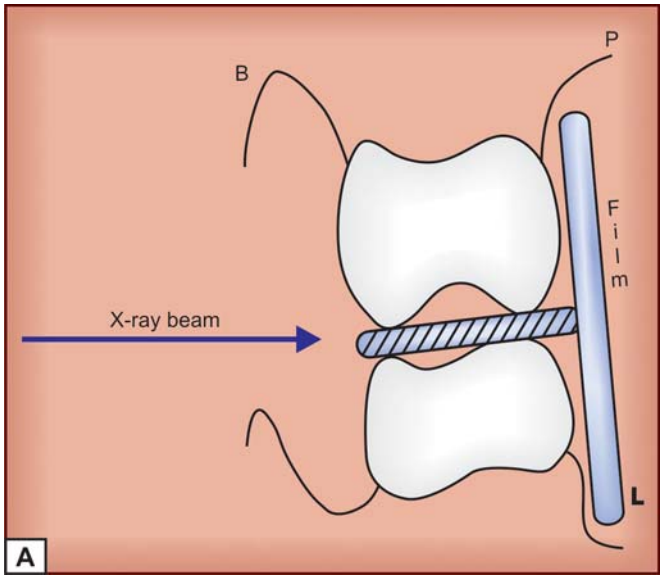
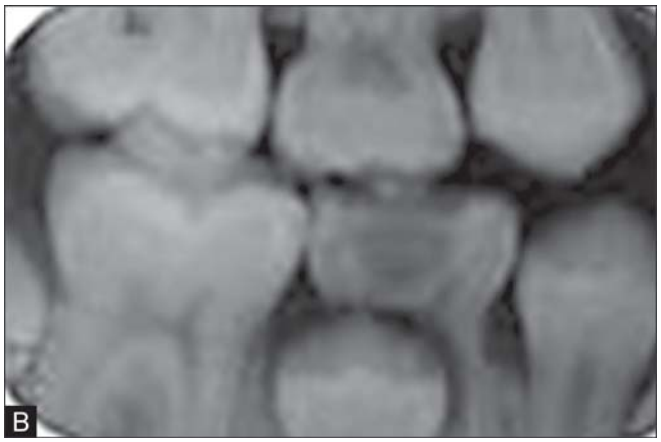


Fig. 3.5: Positioning for bitewing radiograph





Figs 3.6A and B: Bitewing tab placement and radiograph

Periapical Radiographs (Fig. 3.7)

Indications

- To determine root end condition and environment in young permanent teeth
- Evaluate pulp treatment
- Detect developmental abnormalities
- Discover pathologic changes associated with primary teeth
- Detect alterations in the integrity of the periodontal membrane
- Diagnose pulp calcification or root resorption
- Analyses space in mixed dentition
- Assessment of eruption of teeth
- Evaluation of trauma to hard tissues like alveolar bone and teeth, etc.

Technique

There are essentially two methods of taking periapical radiographs:

- Paralleling and bisecting angle techniques; each having benefits and limitations when used with pediatric patient. Regardless of which technique is used, film positioning for the two techniques is identical.

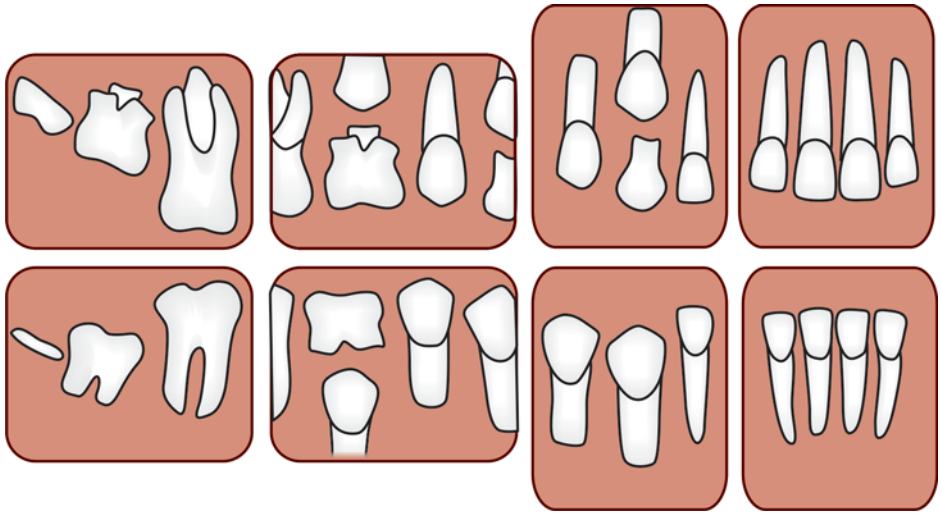


Fig. 3.7: Periapical views

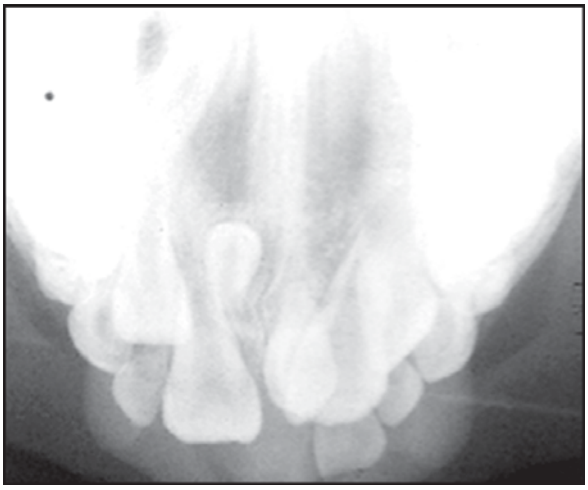


Fig. 3.8: Occlusal radiograph

Occlusal Radiograph (Fig. 3.8)

Indications

Occlusal view-anterior

- Determines the presence, shape and position of midline, supernumerary teeth
- Determine impaction of canines
- Determine the presence or absence of incisors
- Assess the extent of trauma to teeth and anterior segments of the arches after accidents.

Film Sizes

No 2 – for young children through the mixed dentition and anterior occlusal films for children in late mixed and permanent dentition.

Technique

Maxillary occlusal radiograph

- Seat the child with occlusal plane parallel to the floor
- Carry the film to open mouth and place it flat against the maxillary arch, with its long axis side to the side and edge of packet at the incisal edge of the teeth
- Have the patient merely close the mouth to hold the film between the teeth
- Direct the central beam at +60° angle through the tip of the nose.

Mandibular occlusal radiograph

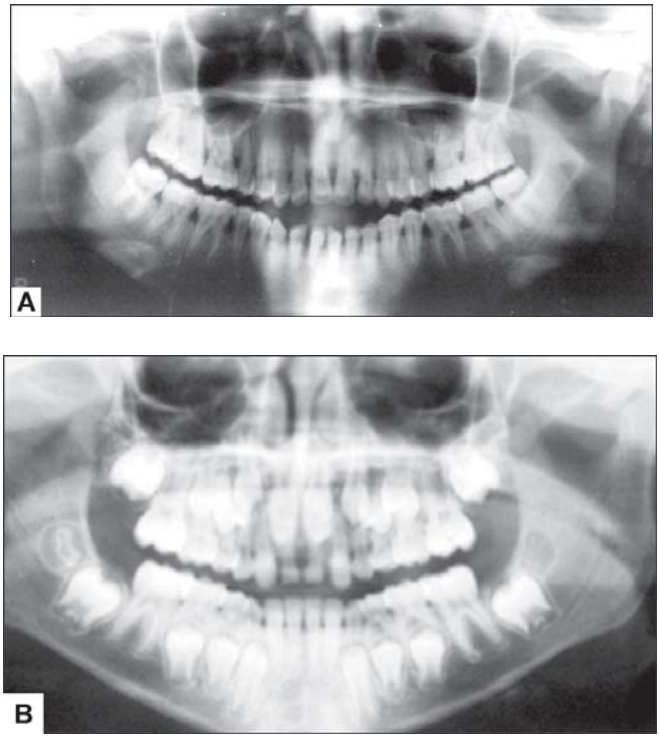
- Position the head so that occlusal plane is at 45° angle to the floor and position the film as usual except with lead foil facing maxillary arch
- Direct the central ray 15 degrees through apices of central incisors, i.e. symphysis.

Combined Occlussal Film

Take the large anterior occlusal film fold it in half with the lead lining inside. Place the film with folded portion in the patient's mouth, position the patient as described for maxillary occlusal view and after exposing; position the patient for mandibular occlusal view having the film in the patient's mouth and expose.

Panoramic Radiographs (Fig. 3.9)

It is an extraoral radiographic technique in which the X-ray source and the X-ray film move around the patient around an axis in opposite directions A diagnostic film includes the teeth, supporting structure, maxillary region



Figs 3.9A and B: Panoramic radiographs

extending up to superior third of orbit, mandible including temporomandibular joints.

- It provides fine details of condylar fractures, traumatic cysts and anomalies that might have gone undetected with the routine periapical series of radiographs
- It is of great value when used for disabled patients and requires very minimal time of 15-20 seconds and reduced exposure
- However, the only inherent drawback is lack of image detail for diagnosing early carious lesions. Adjunct bitewing radiographs and selected periapical radiographs are required.

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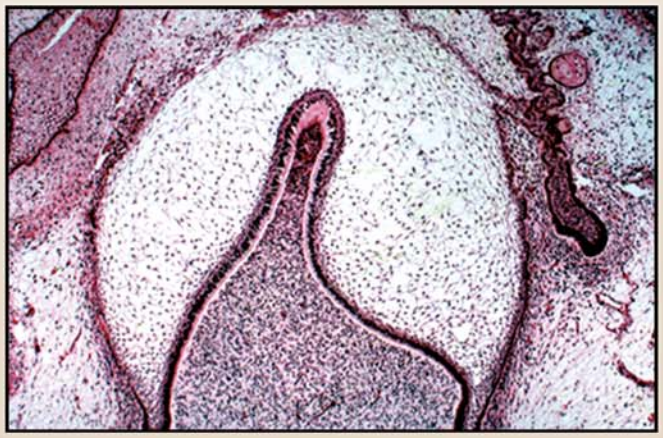


Development and Eruption of Teeth

INTRODUCTION

The primitive oral cavity or stomodeum is lined by stratified squamous epithelium also termed as the oral ectoderm of the foregut to form the buccopharyngeal membrane. At about 27th day of gestation this membrane ruptures establishing a connection between the primitive oral cavity and foregut. Most of the connective tissue cells underlying oral ectoderm are neural crest or ectomesenchymal in origin; which are thought to induce or instruct the overlying ectoderm to initiate tooth development.

How does tooth formation begin?

What initiates it?

Interaction between both epithelium and ectomesenchyme are needed to produce a tooth germ; epithelium and ectomesenchyme can be separated and grown by themselves, but no tooth germ forms. Hence these two tissues must communicate or interact with each other across the basement membrane.

Epithelium and jaw mesenchyme can be separated and then recombined in various ways; the recombinant is grown in anterior chamber of eye (Fig. 4.1 and Table 4.1). The jaw epithelium produces signals such as Bone morphogenetic proteins and Fibroblastic growth factors which activates genes like Lhx-6, Lhx-7 in the ectomesenchyme where the teeth will form. Thereby the tooth development is initiated.

How are different teeth specified in the correct position in the jaw?

How is the dentition patterned?

The ectomesenchyme of the jaw now becomes specialised dental ectomesenchyme whose interaction with different regions of jaw epithelium leads to differentiation or patterning of dentition. Some of the genes involved and Bmp4 and Fgf-8 etc. If the ectomesenchyme interacts with jaw epithelium at the incisor region incisors are formed and likewise in the canine and molar regions leading to the formation of canines and molars (Fig. 4.2). The jaw epithelium probably specifies that incisors form anteriorly and molars posteriorly etc; different genes are switched on in the

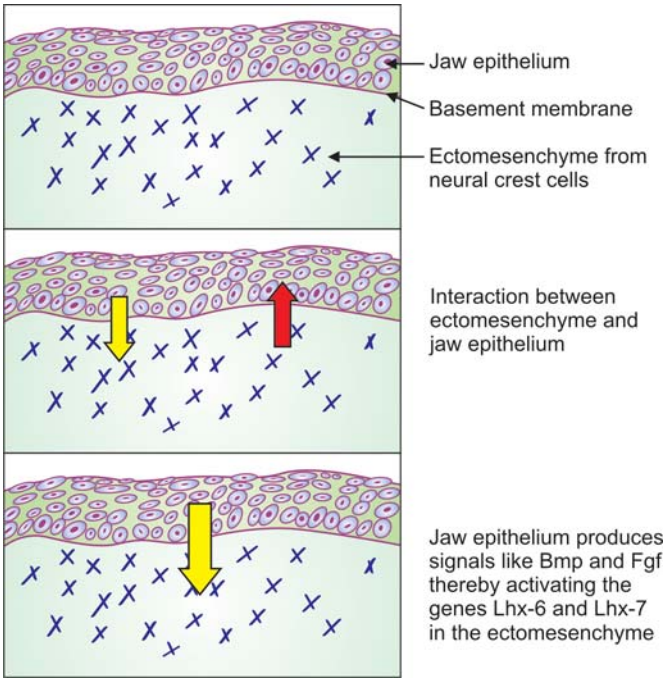


Fig. 4.1: Schematic representation of interaction between jaw epithelium and ectomesenchyme across the basement membrane

ectomesenchyme in relation to the different tooth types; there is not single gene for each tooth type; overlapping gene sites control tooth shape.

Different homeobox genes (Msx-1, Msx-2 etc) are active in the specific regions of the ectomesenchyme and overlapping is noted between these gene sites and its action on the patterning of the dentition.

How is the shape of individual teeth determined (Fig. 4.3)?

The shape of the future developing tooth is determined by inner enamel epithelium. The inner enamel epithelium folds and makes shape of future enamel/dentin junction; cell division in epithelium stops at cusp tips (white) and continues in grooves and at side of cusps (black).

Table 4.1: Stages in tooth growth

Combination	*Teeth*	*Bone*	*Cartilage*	*Neural crest*
Neural crest and mandibular epithelium	+	+	+	+
Neural crest and limb epithelium	–	+	+	+
Neural crest	–	–	+	+
Mandibular epithelium	–	–	–	–

*From Lumsden AGS: In Mederson PFA: **Development and evolutionary biology of the neural crest cells**, Newyork, 1987, John Wiley and Sons

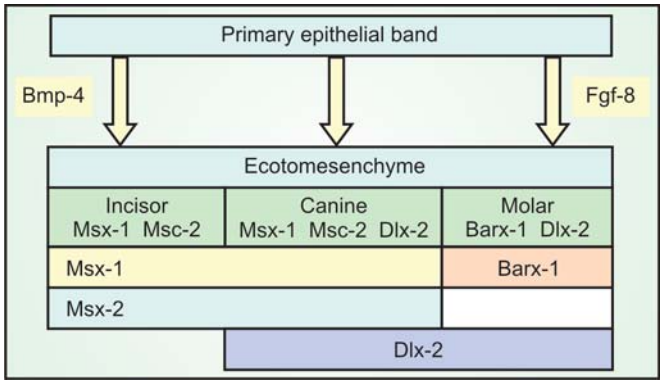


Fig. 4.2: Genes involved in the patterning of the dentition

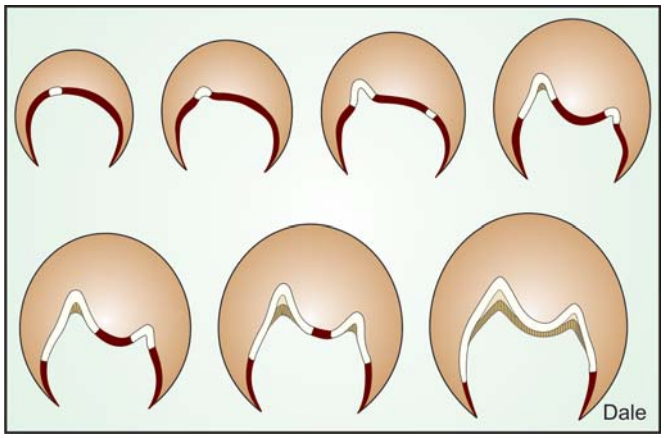


Fig. 4.3: Determination of shape of the tooth

DENTAL LAMINA (FIG. 4.4)

When the embryo is about 6 weeks old certain areas of basal cells of oral ectoderm proliferate more rapidly than do the cells of adjacent areas leading to formation of **"dental lamina".** Dental lamina is a band of epithelium that has invaded the underlying ectomesenchyme along each of the horseshoe shaped future dental arches.

Dental lamina serves as the primordial for the ectodermal portion of deciduous teeth. The development of first molar is initiated at 4th month *in utero;* and second molar is initiated at about 1st year after birth; the third molar at fourth or fifth years.

The distal proliferation of the dental lamina is responsible for the location of the germs of the permanent molars in the ramus of the mandible and the tuberosity of the maxilla. The permanent teeth develop from a lingual extension of the free end of the dental lamina opposite to the enamel organ of each primary tooth.

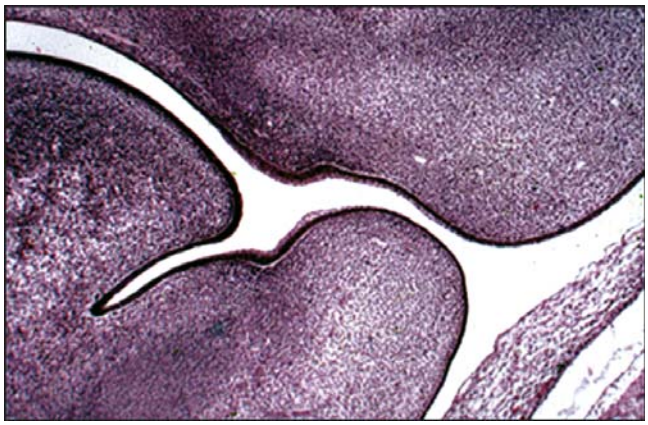


Fig. 4.4: Photomicrograph showing dental lamina

VESTIBULAR LAMINA

Labial and buccal to dental lamina develops another epithelial thickening which is termed as vestibular lamina or lip furrow band. It subsequently hollows to form oral vestibule.

TOOTH DEVELOPMENT (TABLE 4.2)

The development of teeth starts at about 5-6 weeks in utero. Tooth development progresses as a continuous process; the developmental history of the tooth is divided into several morphologic and histologic stages for descriptive study and understanding.

Table 4.2: Stages in tooth growth

Morphologic stages	*Physiologic processes*
Dental lamina	Initiation
Bud stage	Proliferation
Cap stage	Proliferation
Bell stage (early)	Histodifferentiation
Bell stage (late)	Morphodifferentiation
Formation of enamel and dentine matrix	Apposition

Based upon morphologic tooth developmental changes; it is classified as

- Bud stage
- Cap stage
- Bell stage
- Advanced Bell stage

Histophysiologic processes involved in tooth development and growth includes

- Initiation
- Proliferation
- Histodifferentiation

- Morphodifferentiation
- Apposition

Bud Stage (Fig. 4.5)

The bud stage is represented by the first epithelial incursion into the ectomesenchyme of the jaw. This stage is characterized by localized proliferation of cells of the dental lamina at 10 different points representing the position of deciduous teeth. The cells continue to proliferate than the adjacent cells and has mitotic index at localized areas. These localized thickenings in the form of tiny buds constitute the enamel organ.

The enamel organ comprises of peripherally located low columnar cells and centrally located polygonal cells. As a result of increased mitotic activity and migration of neural crest cells into the area the ectomesenchyme surrounding the tooth bud condense leading to formation of dental papilla. The condensed ectomesenchyme that surrounds the tooth bud and dental papilla is termed as dental sac.

Cap Stage (Fig. 4.6)

As the tooth bud continues to proliferate; there is unequal growth in different parts of the tooth bud leading to the characteristic appearance of a "cap" with shallow invagination on the deep surface of the bud shaped enamel organ. Enamel organ in cap stage has the following characteristic features.

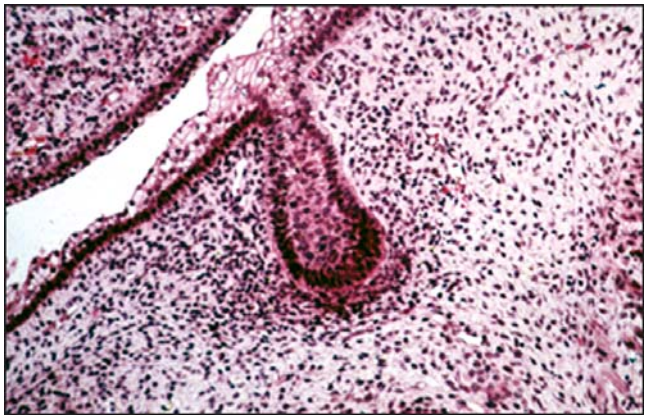


Fig. 4.5: Bud stage

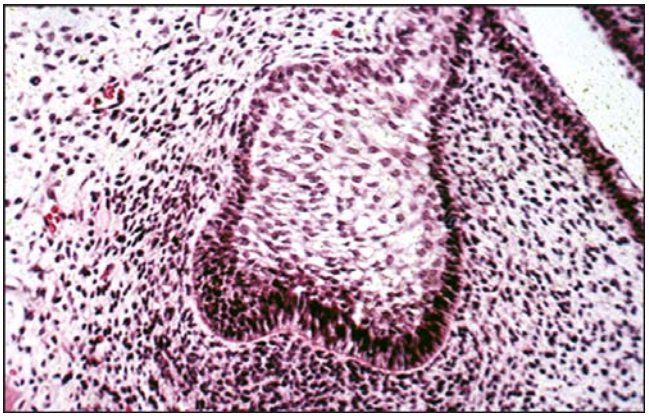


Fig. 4.6: Photomicrograph showing cap stage with outer enamel epithelium

Outer Enamel Epithelium

- Seen in the periphery covering the convexity of the cap
- Cuboidal epithelial cells
- Separated from dental sac by delicate basement membrane.

Inner Enamel Epithelium (Fig. 4.7)

- Seen in the concavity of the cap
- Tall columnar cells
- Separated from dental papilla by a delicate basement membrane

Stellate Reticulum

- Polygonal cells located in center of enamel organ between outer and inner enamel epithelium

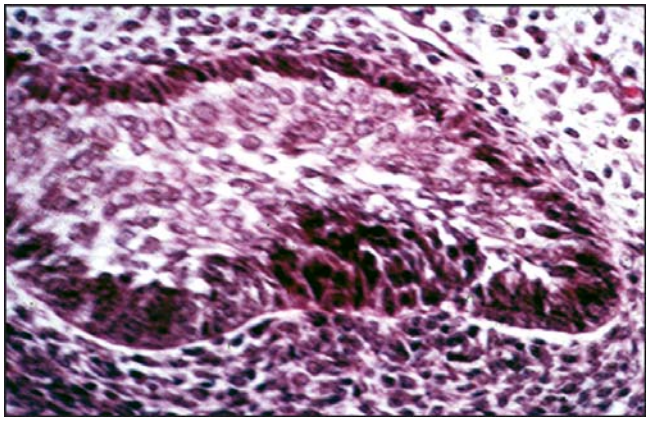


Fig. 4.7: Photomicrograph showing inner enamel epithelium and stellate reticulum

- Cells have branched reticular form
- Spaces filled with mucoid fluid rich in albumin.

Enamel Knot (Fig. 4.8)

The cells in the center of enamel organ are densely packed and form the enamel knot.

It projects towards dental papilla.

It is hold by a vertical extension of densely packed cells called as **"enamel cord".** The enamel knot produces a great variety of signalling molecules e.g.: Shh (sonic hedgehog), Bmp, Fgf which control cell division in the inner enamel epithelium.

Signalling molecules stop growth of the inner enamel epithelium at the future cusp but stimulate growth of inner enamel epithelium away from the cusp; so the cusp increases in height and the cervical loop continues to grow.

The enamel knot has its control over cusp formation.

Fgf-4 from enamel knot stimulates proliferation of epithelium surrounding the not to make cusp higher (Fig. 4.9).

Enamel knot then produces factors that lead to death of its cells (apoptosis).

In multicusped teeth, one or more secondary enamel knots develop after the primary one.

Dental Papilla

- It shows condensation of ectomesenchymal cells
- Proliferation of capillaries and other cells
- Peripheral cells adjacent to inner enamel epithelium differentiate into odontoblasts.

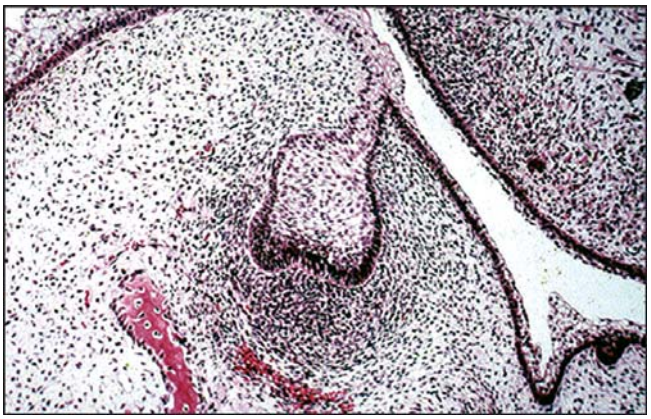


Fig. 4.8: Photomicrograph showing enamel cord

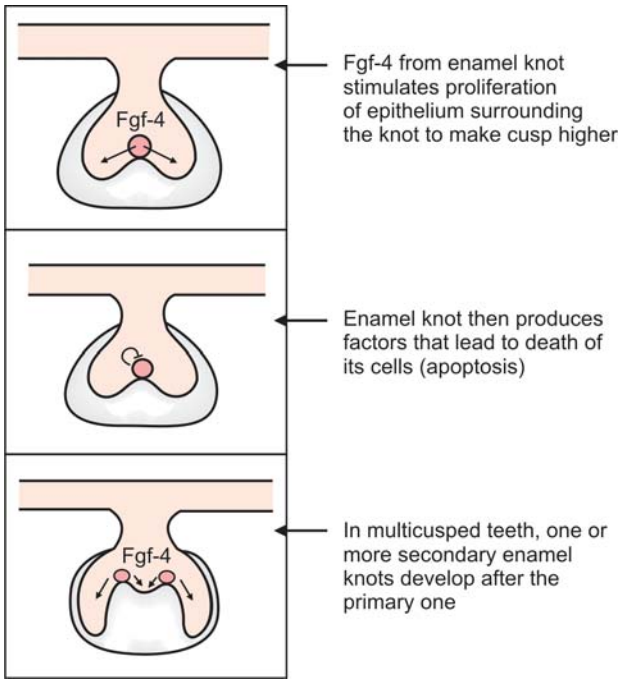


Fig. 4.9: Functions of enamel cord

Bell Stage (Fig. 4.10)

This stage is characterized by increased invagination of the epithelial surfaces of enamel organ into dental papilla forming characteristic **'Bell shaped appearance'.** At this stage; 4 different types of epithelial cells can be distinguished, which includes:

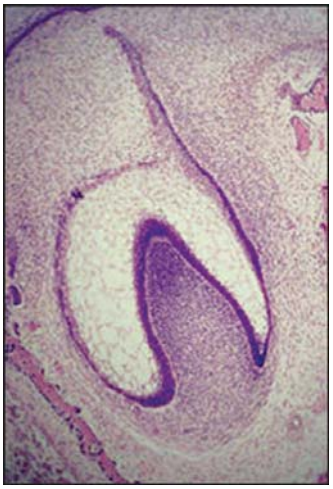


Fig. 4.10: Photomicrograph showing "Bell stage"

Inner Enamel Epithelium

- Single layer of tall columnar cells that differentiate prior to amelogenesis as ameloblasts
- 4-5 μm in diameter and 40 μm high
- Posses desmosomal attachments
- Posses an organizing influence on underlying mesenchymal cells to differentiate into odontoblasts.

Stratum Intermedium

- Layers of squamous cells that are essential for enamel formation
- Absent in part of the tooth germ that outlines the root portion of the tooth

Stellate Reticulum

- Presence of increased amount of intercellular fluid
- Star shaped cells with long processes that anastomoses with adjacent cells
- Before enamel formation begins this layer collapses reducing the distance between ameloblasts and nutrient capillaries near outer enamel epithelium.

Outer Enamel Epithelium

- Low cuboidal cells
- Lie in close proximity to dental sac that contains proliferating capillary loops.
- Provide a rich nutritional supply for the intense metabolic activity of the avascular enamel organ.

Dental Papilla

- Enclosed in the invaginated portion of the enamel organ
- Before enamel production, the peripheral cells differentiate into odontoblasts under the organizing influence of the epithelium
- Basement membrane that separates enamel organ and dental papilla is termed as **"Membrana preformativa".**

Dental Sac

- Shows circular arrangement of its fibers resembling a capsular structure
- With the development of root, the fibers of dental sac differentiate into periodontal fibers that become embedded into developing cementum alveolar bone.

Advanced/Late Bell Stage (Fig. 4.11)

In this stage, the boundary between inner enamel epithelium and odontoblasts outlines the future dentinoenamel junction. Ameloblasts lay down enamel on superficial surface of basement membrane. The odontoblasts lay down dentine on its deeper surface. The process of laying down of enamel and dentine is similar to that of formation of bone by osteoblasts. As layer after layer of enamel and dentin are laid down, the layer of ameloblasts and odontoblasts move away from each other and later the ameloblasts disappear leaving a thin membrane; **'The dental cuticle'**. However the odontoblasts continue to separate the dentine from the pulp throughout the life of the tooth.

In addition, the cervical portion of the enamel organ gives rise to the epithelial root sheath of Hertwig.

ROOT FORMATION

- Begins after enamel and dentine formation has reached the future cementoenamel junction
- Hertwig's epithelial root sheath comprises of outer enamel epithelium, cells of inner enamel epithelium does not produce enamel **(Fig. 4.12)**
- It bends in horizontal plane at future cementoenamel junction to form epithelial diaphragm and mould's the shape of the root and initiates radicular dentine formation

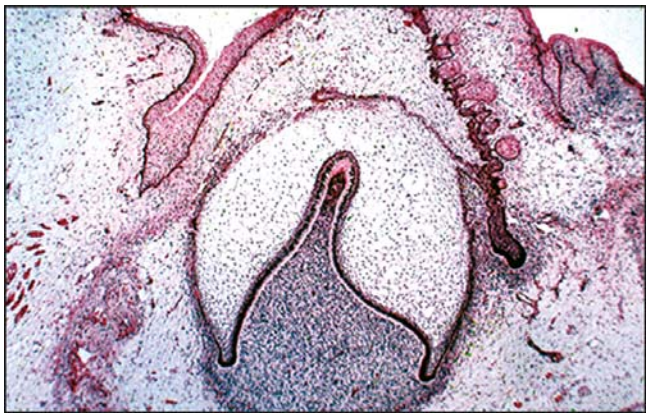


Fig. 4.11: Photomicrograph showing "Advanced bell stage"

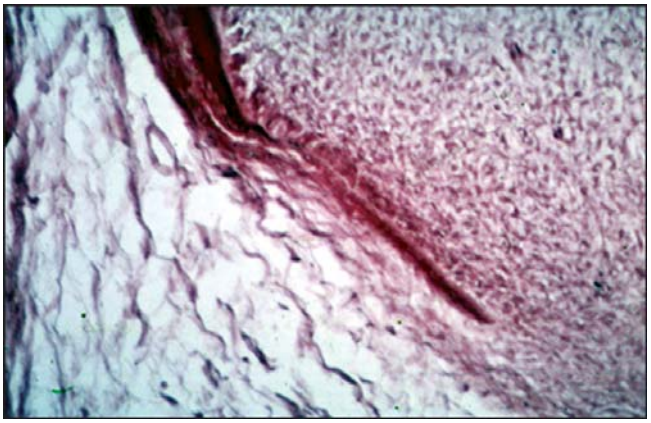


Fig. 4.12: Photomicrograph showing Hertwig's epithelial root sheath

- Cells of root sheath loose their structural continuity and expose the dentine to mesenchymal cells of dental sac to induce and initiate formation of cementoblasts to produce cementum
- At the advanced stages; the wide apical foramen is reduced first to the width of diaphragmatic opening and later narrows by apposition of dentine and cementum at the root apex
- Differential growth of the epithelial diaphragm in multi-rooted teeth causes the division of the root trunk into two or three roots.

Table 4.3 summarizes dental growth and development.

Figure 4.13 shows the "Nolla's stages of tooth development" a histologic classification of development of tooth from its follicle till the root completion from stages 1 to 10.

Figure 4.14 shows diagrammatic representation of development of dentition in primary teeth.

Figure 4.15 shows diagrammatic representation of development of dentition in permanent teeth.

Table 4.3: Dental growth and development

Maxillary (upper) teeth					
Primary teeth	**Central incisor**	**Lateral incisor**	**Canine**	**First molar**	**Second molar**
Initial calcification	14 wk	16 wk	17 wk	15.5 wk	19 wk
Crown completed	1.5 mo	2.5 mo	9 mo	6 mo	11 mo
Root completed	1.5 yr	2 yr	3.25 yr	2.5 yr	3 yr
Mandibular (lower) teeth					
Initial calcification	14 wk	16 wk	17 wk	15.5 wk	18 wk
Crown completed	2.5 mo	3 mo	9 mo	5.5 mo	10 mo
Root completed	1.5 yr	1.5 yr	3.25 yr	2.5 yr	3 yr

Maxillary (upper) teeth								
Permanent teeth	**Central incisor**	**Lateral incisor**	**Canine**	**First premolar**	**Second premolar**	**First molar**	**Second molar**	**Third molar**
Initial calcification	3-4 mo	10-12 mo	4-5 mo	1.5-1.75 yr	2-2.25 yr	at birth	2.5-3 yr	7-9 yr
Crown completed	4-5 yr	4-5 yr	6-7 yr	5-6 yr	6-7 yr	2.5-3 yr	7-8 yr	12-16 yr
Root completed	10 yr	11 yr	13-15 yr	12-13 yr	12-14 yr	9-10 yr	14-16 yr	18-25 yr
Mandibular (lower) teeth								
Initial calcification	3-4 mo	3-4 mo	4-5 mo	1.5-2 yr	2.25-2.5 yr	at birth	2.5-3 yr	8-10 yr
Crown completed	4-5 yr	4-5 yr	6-7 yr	5-6 yr	6-7 yr	2.5-3 yr	7-8 yr	12-16 yr
Root completed	9 yr	10 yr	12-14 yr	12-13 yr	13-14 yr	9-10 yr	14-15 yr	18-25 yr

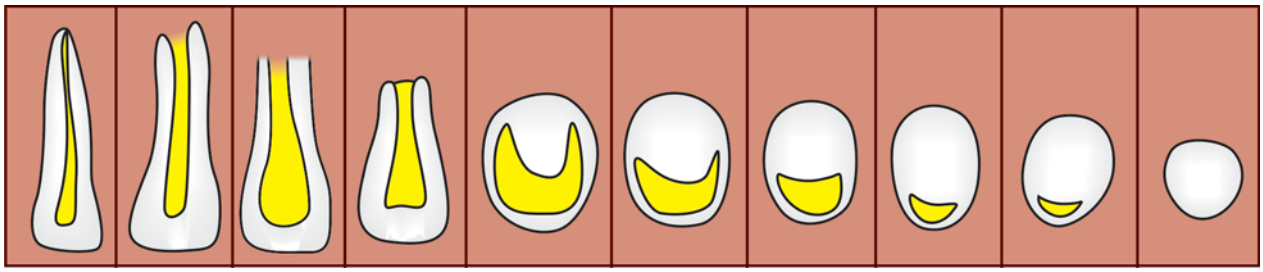


Fig. 4.13: Nolla stages of tooth development

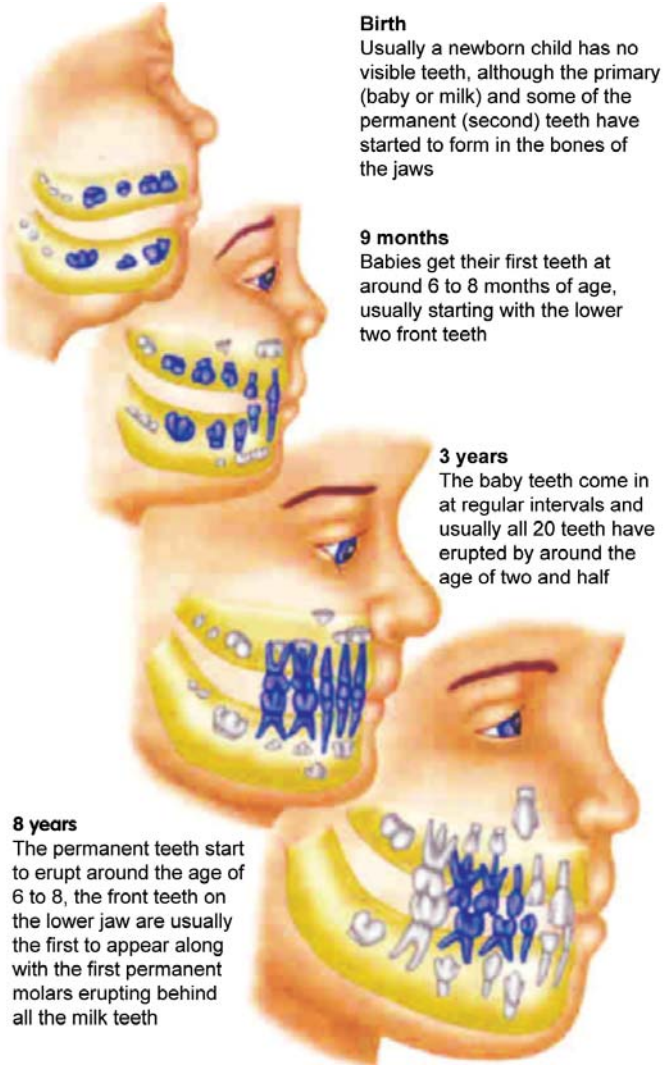


Fig. 4.14: Massler's chart for primary teeth

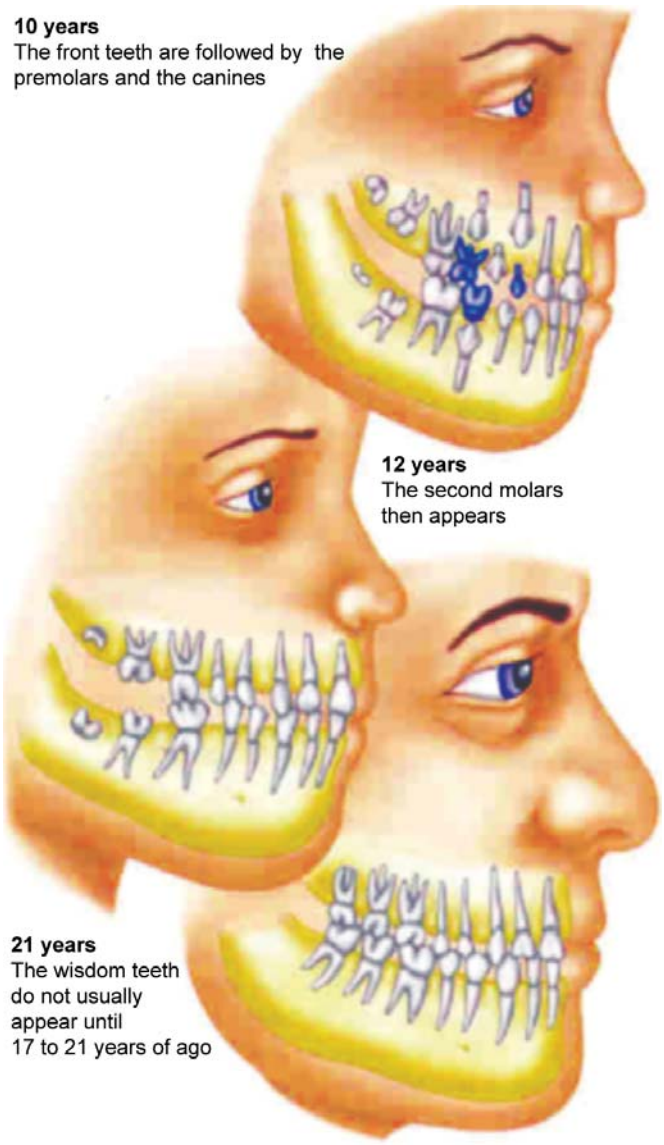


Fig. 4.15: Massler's chart for permanent teeth

ERUPTION OF TEETH

Eruption, a Latinized term **'erumpere'** meaning **"to break out"**; is a complex process. It is considered to be the axial or occlusal movement of the tooth from its developmental position within the jaw to its functional position in the occlusal plane. However, eruption does not only involves axial movements but also other complex movements related to maintaining tooth position in the growing jaws and compensating for masticatory wear.

There are various factors that influence the timing of eruption which are summarized in **Figure 4.16.**

Tooth eruption: what is the origin of the eruptive force?

Tooth eruption needs a force to push or pull a tooth; force could come from either of the following factors:

- Growth of root
- Growth of pulp
- Growth of bone in bottom (fundus) of socket and/or alveolar margin
- Some constituent of periodontal ligament
  - Collagen
  - Fibroblasts

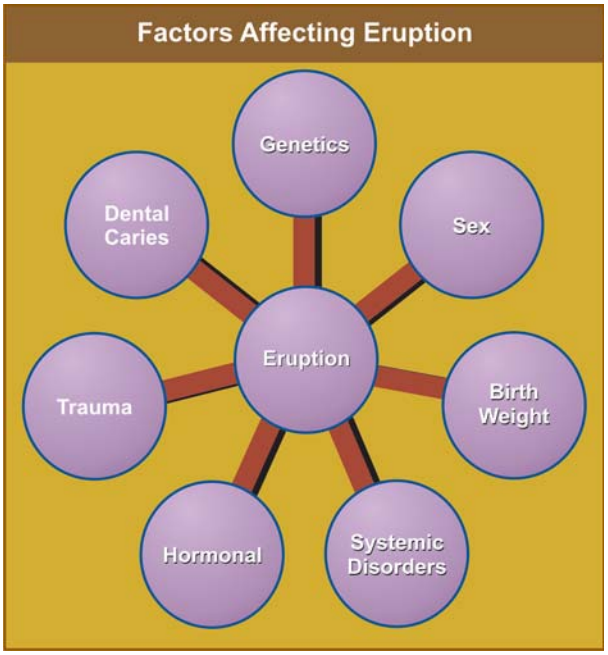


Fig. 4.16: Factors affecting eruption

- Proteoglycans
- Tissue fluid pressure
- Blood supply (Usually Proteoglycans, tissue fluid pressure and blood supply are linked together as the causative factor)

Theories of Tooth Eruption

There are various theories propounded on mechanism of tooth eruption which are considered to be based on various factors. It is thought that eruption of teeth is a multifactorial process. Following are some of the proposed theories of mechanism of tooth eruption.

a. Papillary constriction theory
b. Bone remodeling theory
c. Epithelial path theory
d. Cushion Hammock theory
e. Root growth theory
f. Hydrostatic/vascular pressure theory
g. Ligament traction theory.

Although many possible causes have been proposed, only four are merited serious consideration.

Bone remodeling theory: This theory states the role of selective resorption and apposition of bone along the erupt wall of the developing tooth in exerting an eruptive force to the tooth along with the periodontal ligament between the tooth and bone.

Root remodeling theory: Root formation yields an overall increased length of tooth along with bony changes of the jaw in height leading to occlusal movement of teeth. According to this theory root growth produces an inherent eruptive force that aids in axial movement of the tooth towards oral cavity and concomitant resorption and apposition of bone apically.

This theory was disproved later due to clinical facts like eruption of rootless teeth; experimental resection preventing further root formation does not stop eruptive tooth movement, alveolar bone at base of socket cannot act as a fixed base because pressure on bone results in resorption and teeth like permanent canines, erupt further than the length of their roots.

The root/pulp growth theory by itself cannot explain how teeth erupt because teeth with fully grown pulps can erupt (otherwise eruption would stop in permanent first molars at 9 years of age, in second premolars 14 years and in second molars at 15 years etc), eruption rates are about same whether root growing or not growing, some normal and abnormal teeth erupt longer distances than root length as already stated.

Vascular pressure theory: It is already cited that teeth move in their socket in synchrony with arterial pulse, hence it was said to be the cause for movemnt of tooth during eruption. But whether such pressure are the prime factors to move teeth is debatable because of surgical excision of root & therefore the local vasculature, does not prevent tooth eruption.

Periodontal ligament traction: This is the most supported theory with clinical evidence of the presence of eruptive force in the dental follicle periodontal ligament complex. This theory is well supported by lot of clinical evidence and experiments contributed by Berkovitz resection/transection (1969) experiment in the rodent incisor. Berkovitz had removed pulp and there was no bone formation in socket fundus; later Berkovitz repeated experiment adding a barrier of plaster of Paris between the end of the root and the transected distal part of the

tooth–the distal part still erupted; so the growing root apex was not the cause; if pulp growth, root growth, bone growth all stop in rodent incisor, eruption continues. It is the only source when the other explanations have been shown to be unsatisfactory thereby he concluded that periodontal ligament as the source for eruptive force.

Several constituents of periodontal ligament could generate an eruptive force:

- Collagen contraction
- Fibroblast traction
- Tissue fluid pressure (including blood supply and proteoglycans which can affect tissue fluid pressure)

Collagen contraction in periodontal ligament:

Thomas (1965) disturbed the production of collagen in the pdl by giving the lathyrogen aminoacetonitrile; this prevents cross-links between collagen molecules; in the rat incisor, the impeded and unimpeded eruption rates were reduced compared with controls. Thereby Thomas concluded that pdl collagen was important in tooth eruption; but his experiments failed to emphasize the role of collagen contraction.

Collagen of the oblique principal fiber groups seems to be arranged so that it could pull tooth away from apex of socket; turnover rate of collage in pdl is high; but collagen cannot contract under physiological conditions.

Later experiments using lathyrogen aminoacetonitrile by Berkovitz *et al*, used smaller doses that Thomas has done and Berkovitz found that the eruption of the rat incisor, provided it was cut out of occlusion, (unimpeded) was not affected by this lathyrogen; so Berkovitz concluded that collagen traction was not a cause of tooth eruption and this is now generally agreed.

Fibroblast traction/movement:

The eruptive force from periodontal ligament resides in the contractile property of the fibroblasts and its cell-to-cell connectivity of adherence type and its close relation to collagen. The contractile property and orientation of collagen fiber bundles generate an eruptive force.

Fibroblasts can move, perhaps 'pulling' the tooth along with them; some fibroblasts ('myofibroblasts') contain contractile proteins; (these are important in wound contraction). Colchicine disrupts contractile microtubules and slows eruption; (but colchicine also stops cell division).

There are also some evidences against these two factors:

- Collagen does not contract
- Collagen fibers in pdl usually seen to be wavy
- Collagen synthesis in pdl can be stopped but eruption continues
- Not many myofibroblasts in pdl
- No good experimental evidence in favour of myofibroblasts

Tissue fluid pressure (including blood supply and proteoglycans which can affect tissue fluid pressure)

This factor is well supported by following evidences:

- Drugs that decrease tissue fluid pressure (e.g. noradrenaline), slow eruption (Shimada et al 2006: effects of vasoactive drugs on eruption rate of rat incisor)
- Proteoglycans present in large amount in pdl and swell if take up fluid;
- Pdl is highly vascular and capillaries are fenestrated (have pores, closed only by cell membrane, in their walls).

This is a currently favoured theory of how the eruptive force is generated.

Tooth eruption as a multi-stage process

- Positive blood/tissue fluid pressure in follicle around root/pdl maintains an eruptive force.
- If tooth moves (erupts) slightly, proteoglycans are synthesised and these attract more fluid.
- After a few days, collagen is resynthesised and now holds tooth in new (erupted) position.
- Bone in depth of socket and alveolar margins remodels to adapt to new position of tooth.

It is supported by a simple anology of a sailor (fibroblast) pulling on a rope (collagen) attached to sail (tooth). To move the sail the sailor must remain stationary and pull on the rope (contraction) and coil it on the deck (collagen remodeling). Thereby eruption is a co-operative activity of the supporting tissues of a tooth, each factor may mainly operate at one time and dental follicle is important in pre-emergent phase whereas root growth may play a part in pre-emergent phase.

Eruption Pattern

Physiologic tooth movement is subdivided into following phases:

1. Pre-eruptive tooth movement
2. Eruptive tooth movement
3. Post-eruptive tooth movement

Pattern of Tooth Movement

Eruption pattern on stages can also be classified for ease of understanding every stage as following:

- Pre-eruptive movements
- Intra-osseous movements
- Entry into oral cavity
- Pre-occlusal movements
- Occlusal movements

Stages of tooth eruption:

- Pre-emergent phase/stage:
  - This phase comprises of all events before the tooth enters into oral cavity.
  - In this phase the developing dental follicle plays an important role.
  - During the eruption of secondary/permanent dentition there is a need for removal of deciduous roots and/or bone and gingival collagen as well as eruptive force.
- **Post emergent phase/stage:**
  - This phase comprises of all events that include after the eruption of tooth into oral cavity.
  - Periodontal ligament plays a vital role during this phase.
  - This stage requires the basic eruptive force in instances of both primary and secondary dentitions.

Pre-eruptive Tooth Movement

It includes the movements of the tooth germ within the jawbone until it is ready to erupt into the oral cavity. When deciduous tooth germs first differentiate, they are very small and a good deal of space exists between them which soon gets used because of the rapid growth of the developing tooth germs resulting in crowding, especially in the incisor canine region. This crowding is gradually relieved by growth of jaw in length, which permits drifting of the tooth germs.

Permanent teeth with deciduous predecessors also move before they reach the position from which they will erupt at later stage. It includes two characteristic movements of the tooth germ.

a. Total bodily movement
b. Differential growth of the tooth germ in accordance to maintenance of a fixed position.

Eruptive Tooth Movement (Fig. 4.17)

Eruptive tooth movements coincide with the initiation of root formation. During the phase of eruptive tooth movement the tooth moves from its position within the bone of the jaw to its functional position in occlusion, and the principal direction of movement is occlusal or axial. During this phase significant developmental events; like formation of the roots, the periodontal ligament and the dentogingival junction. Root formation is initiated by growth of Hertwig's epithelial root sheath which in turn differentiates dental papilla to form odontoblasts to produce radicular dentine.

Formation of root dentine is followed by deposition of cementum along root dentine and formation of periodontal ligament. Fibroblasts have a cytoskeleton that comprised of contractile proteins, which exhibit frequent cell-to-cell contacts of adherence type and is in close

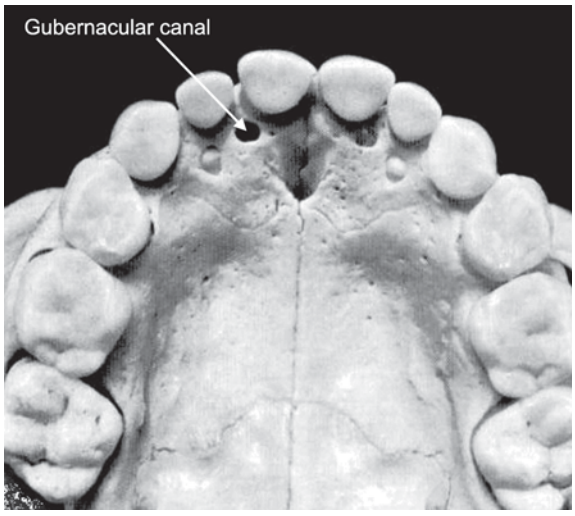


Fig. 4.17: Diagram showing the presence of Gubernacular canal lingual to the deciduous incisors

relationship to collagen of periodontal ligament. As eruptive movements proceed the enamel of erupting tooth crown is covered by reduced enamel epithelium. The bone covering the erupting teeth gets resorbed exposing the tooth to pass through the connective tissue of oral mucosa during which the reduced enamel epithelium fuses with oral epithelium to form a band of cells; piercing which the crown erupts into oral cavity. These bands of epithelia later form the dentogingival unit.

Bone removal is necessary for permanent teeth to erupt. In case of those teeth with deciduous predecessors; the successional tooth germ first develops within the same crypt as its deciduous predecessor, bone surrounds the tooth germ completely except for a small canal that is filled with connective tissue. Comprising of epithelial remnants of dental lamina which is termed as **"Gubernacular cord"** which aids in guiding the permanent tooth as it erupts and the canal is termed as **"Gubernacular canal".**

Gubernacular canals are not seen in newborn because the jaw has to grow before deciduous and permanent teeth can occupy separate bony sockets/crypts. Gubernacular canals of permanent incisors (Fig. 4.20) and canines open through bone lingually to deciduous predecessors; Gubernacular canal for premolars opens between roots of deciduous molars.

Posteruptive Tooth Movement

It comprises of movements of the tooth after the tooth has reached its functional position at the occlusal plane. It compensates for accommodating the changes during jaw growth, continued occlusal wear and interproximal wear of the teeth. Forces seen during posteruptive movements include axial/occlusal; mesial and distal movements.

Shedding or Exfoliation of Primary Teeth (Fig. 4.18)

Exfoliation of deciduous teeth involves the resorption of their roots by osteoclasts.

Root resorption is aided by:

- Presence of underlying permanent successor tooth and its follicle

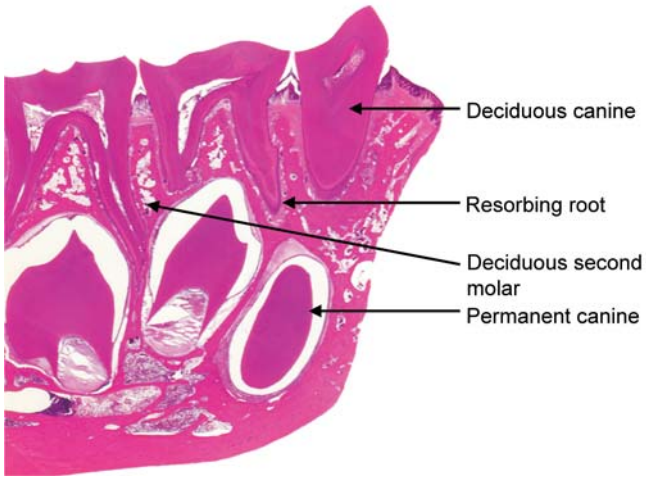


Fig. 4.18: Exfoliation of primary teeth

- Increase of muscles and chewing force as child grows which leads to the damage of the periodontal tissues and encourages osteoclastic root resorption.
- Osteoclasts are attracted to a bare mineralised surface.

Theories and experiments supporting the cause of exfoliation and pre-emergent eruption

- Fixation of erupting premolars of dogs by transmandibular wire prevents tooth movement but not bone resorption that creates the eruptive pathway and exfoliation of deciduous tooth (Cahill, 1969).
- Removal of coronal follicle prevents eruption but root growth continues; removal of radicular (basal) follicle stops bone formation in crypt but not resorption around crown (Marks & Cahill, 1972).
- Cahill & Marks (1980, 1984) replaced unerupted premolars of dogs with plastic and metal replicas, provided that the dental follicle was still present around the replicas, the "premolars" erupted.

The above results of Cahill and Marks suggest that the dental follicle is the most important requisite for shedding of deciduous teeth and pre-emergent stage of eruption;

Role of reduced enamel epithelium in resorption:

- Reduced enamel epithelium is protective to enamel and prevents resorption of the mineralised surface.
- Epidermal growth factor from follicle stimulates formation of TGF-β by reduced enamel epithelium.
- TGF-β stimulates collections of osteoclasts precursors

- It produces cytokines e.g. Interleukin 1-α which is a powerful promoter of bone resorption.
- Produces proteases that help to remove overlying tissues and aid the fusion of REE with oral epithelium.
- These proteases include matrix metalloproteinases (MMPs) e.g. collagenase.
- Also proteinases that digest a sialoprotein (DF-94) that is possibly an enamel organ protein in the dental follicle.

Histological Features of Root Resorption (Fig. 4.19)

As already discussed osteoclastic activity is responsible for the resorption of the roots of deciduous teeth which is promoted by various factors. The osteoclasts do not differentiate the developing secondary dentition or primary dentition, all it does is resorbs the mineralized tissue and hence the role of reduced enamel epithelium protecting the developing secondary dentition is again emphasized here.

The osteoclastic activity is seen over the mineralized surfaces of the primary teeth adjacent to the developing or erupting secondary teeth. The photomicrograph shows the removal of mineralized tissue namely cementum and dentin leading to concave shaped areas all over the surface of dentin and cementum (Fig. 4.20)

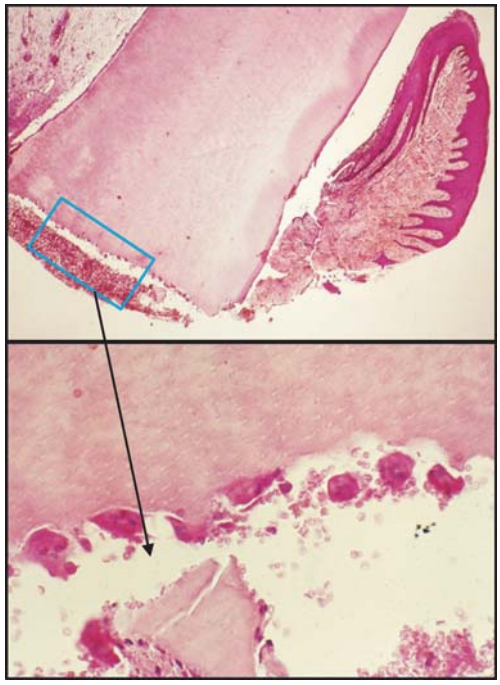


Fig. 4.19: Photomicrograph showing the presence of osteoclasts over the dentinal surface of a shed deciduous teeth

Teething

Teething though not a scientific terminology; it refers to the eruption of teeth into the oral cavity in a child. When the baby begins teething, there is no set pattern on when it will begin? How long it will take? And how painful it will be? The process of teething often follows hereditary patterns.

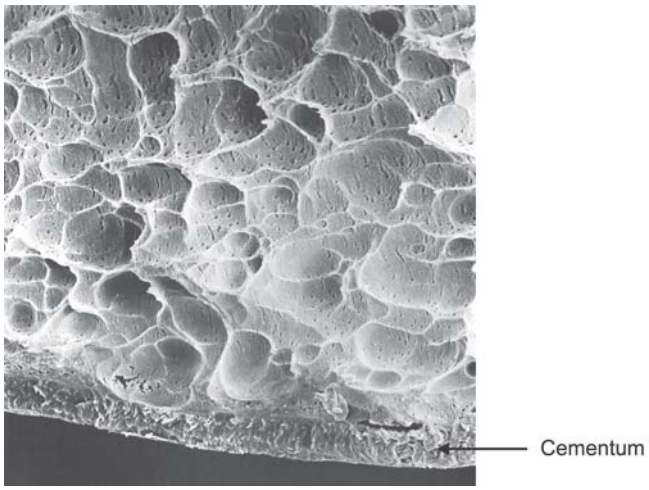


Fig. 4.20: Photomicrograph showing dentinal and cemental surfaces destroyed by the osteoclastic activity in the form of concave depressions, also showing the dentinal tubules along the dentinal surface

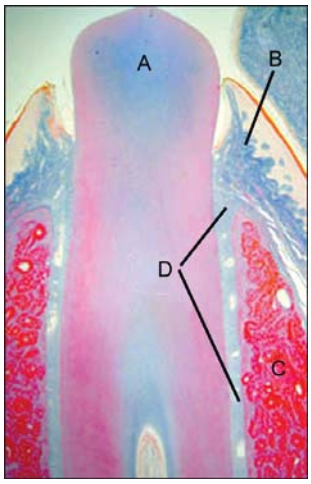


Fig. 4.21: Histologic slide of tooth erupting into the mouth (a) tooth (b) gingiva (c) bone (d) periodontal ligaments

Symptoms

The symptoms vary from child-to-child. Because of these differences, parents and physicians often disagree to the symptoms of teething and how painful it is. Some of the common symptoms include:

a. **Irritability:** As the tooth approaches closer to the surface, baby's gums become increasingly sore and painful leading to; baby being fussy and irritable

   Use of teething pads /teething toys, etc would help reducing the discomfort caused

b. **Drooling:** There may be excessive salivation in certain children during the period of teething.

c. **Coughing:** Excess salivation can occasionally trigger cough or gag reflexes in a child.

d. **Biting and gnawing**

e. **Diarrhea:** Many physicians disagree with this, however, scientific study has shown that due to soreness of gums, babies try to bite and gnaw at play toys or fingers which may not be clean or dirty and eventually leads to diarrhea.

f. **Low grade fever**

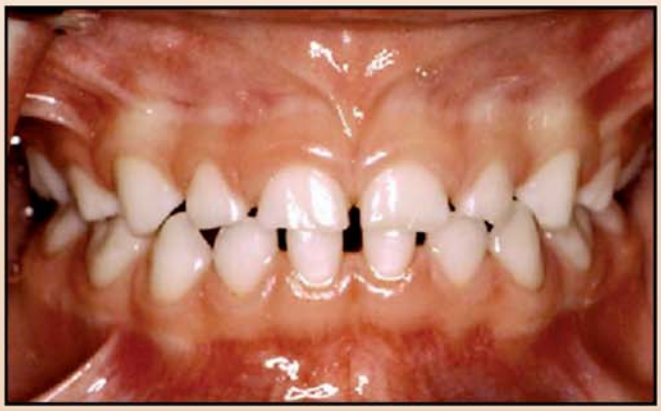
g. **Sleeplessness**

h. **Cold like symptoms.**

Management of Teething

- Advocate use of clean and chilled teething rings, rubber teething toys
- Offer cold bottle of water or cooled foods like yoghurt etc
- In case of low grade fever, a mild antipyretic on physician's advice is recommended.

5



Morphology of Primary Teeth

INTRODUCTION

It is very much essential to be familiar with morphology of primary teeth when we study the specialty of pediatric dentistry. The need for detailed information on morphology of primary teeth is due to a number of differences that exists between them and their successors (i.e.) permanent teeth the differences exist in:

- Morphology
- Histology thereby necessitating variations in procedures like:
  - Restorative procedures
  - Pulp therapy, etc.

When studying primary teeth morphology it is also very important to know about chronology.

LIFE CYCLE

In the deciduous teeth, the center of formation of each lobe is located at the cusp tip.

Deciduous teeth begin to erupt in children at age of 7-8 months and eruption is completed by age of 2½ to 3 years.

Their life span is for a short period and is gradually replaced by the permanent teeth starting from 6-7 years of age and the replacement in completed at about 12-13 years.

NOMENCLATURE

Deciduous teeth are also termed as "milk teeth" or "primary teeth". The word deciduous is a Latin term which means "to fall off".

There are 20 teeth in deciduous dentition, 10 in each jaw. The right and left halves of each jaw teeth have equal numbers of teeth (5 in each quadrant). The teeth are named from midline in each quadrant as (Figs 5.1 and 5.2).

- Central incisor
- Lateral incisor
- Canine
- First molar
- Second molar

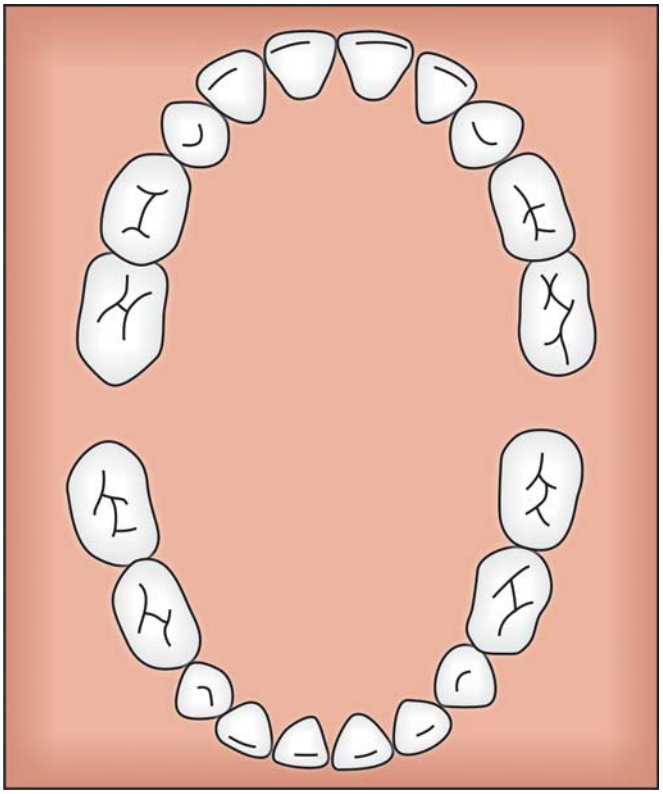


Fig. 5.1: Primary dentition—occlusal view

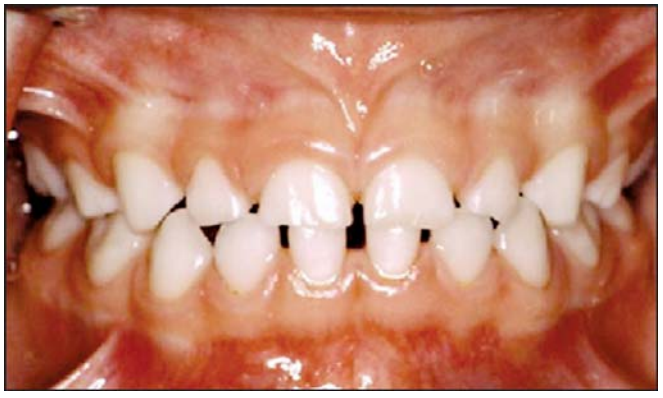


Fig. 5.2: Primary dentition—facial view

Numbering of Primary Teeth

According to "The Federation Dentaire International Special Committee on Uniform Dental Recording",

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

According to the palmer system; the deciduous teeth are named as follows:

Upper Right	Upper Left
E D C B A	A B C D E
E D C B A	A B C D E
Lower Right	**Lower Left**

Numbering of Permanent Teeth

Upper Right	Upper Left
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
Lower Right	**Lower Left**

First number indicates the quadrant and second number indicates the tooth number.

Dental formula primary dentition is as follows:

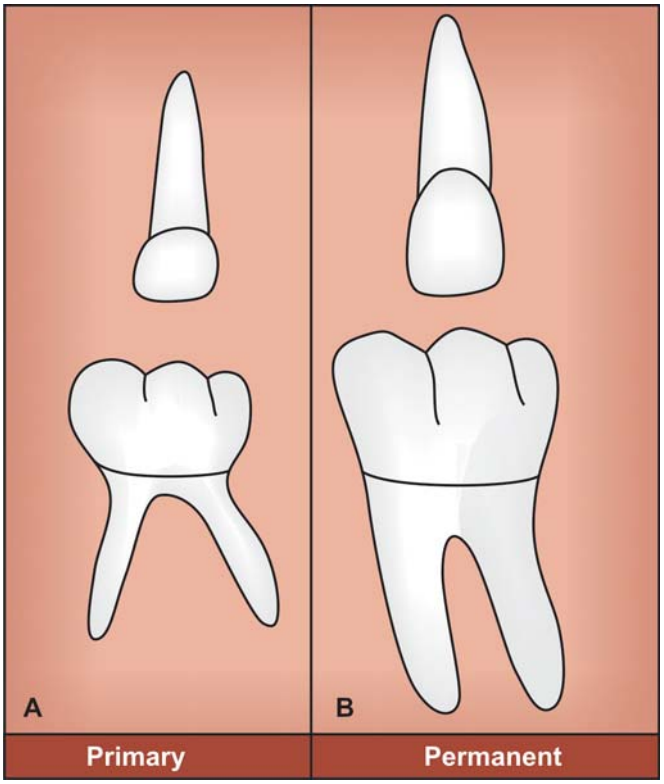
$$\text{Incisors } 4/4 \text{ Canines } 2/2 \text{ Molars } 4/4 = \frac{\text{10 maxillary teeth}}{\text{10 mandibular teeth}}$$

Important Functions of Sound Primary Teeth

1. Efficient mastication of food.
2. Maintenance of normal facial appearance.
3. Formulation of clear speech.
4. Maintenance of space and arch integrity and continuity.
5. An aid for normal growth and physical development.
6. Prevents malocclusion
7. Periapical infection in deciduous teeth can lead to dark hypoplastic lesion in permanent teeth (Turner's hypoplasia) developing beneath them.
8. Missing and badly decayed deciduous teeth are important reasons why children reject foods that are essential for their growth and development.

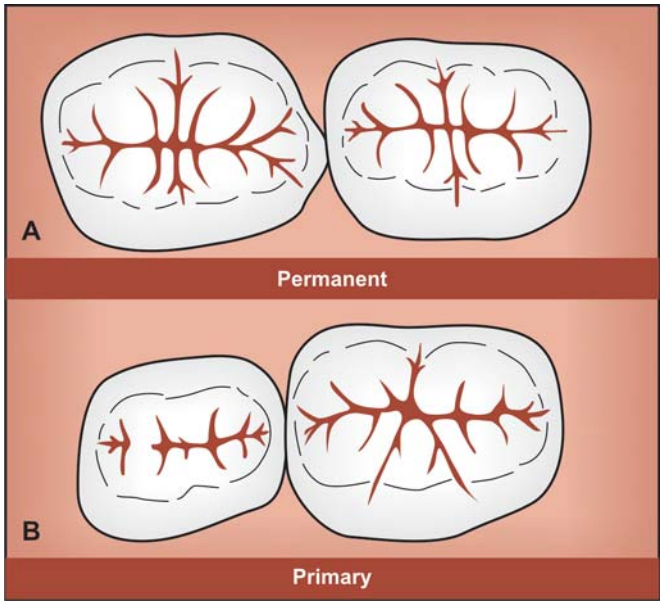
GENERAL CHARACTERISTICS OF PRIMARY TEETH CROWN MORPHOLOGY (FIG. 5.3)

1. Primary teeth are smaller in size than the analogous permanent teeth.
2. They are brighter and whiter in color.
3. They are less mineralized than the analogous permanent teeth.
4. They have shorter crowns with respect to roots.
5. Crowns have marked constriction at cervix .

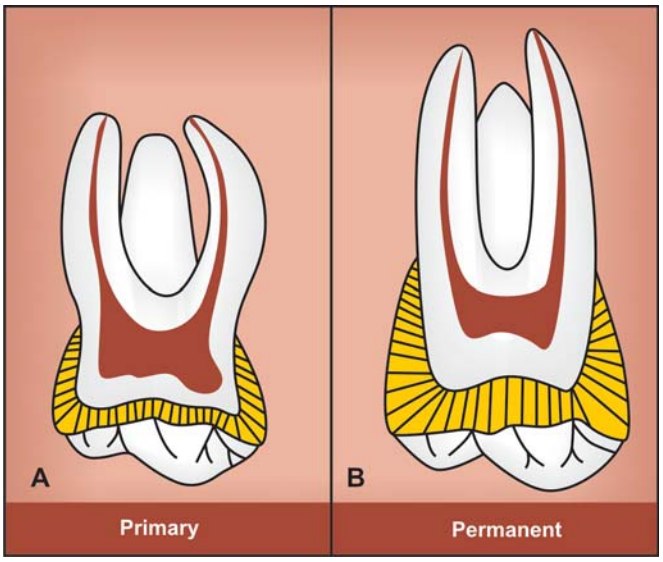


Figs 5.3 A and B: Differences between primary and permanent teeth—crown size

6. Enamel and dentin thickness is less than that of permanent teeth.
7. Crowns of deciduous anterior teeth are wider mesiodistally as compared to their length in comparison to the permanent teeth.
8. They are highly prone to attrition. A greater degree of attrition is present in primary dentition because of the jaw growth and the various positions of occlusion of teeth before their shedding.
9. Absence of mamelons in primary anterior teeth.
10. Labial surfaces of deciduous incisor are smooth and do not show depressions or perikymata.
11. They have prominent cingulum and occupy about 1/3 rd of the crown length.
12. The facial surfaces of anterior teeth show prominent cervical ridges running mesiodistally in cervical third.
13. In deciduous molars, the buccal and lingual surfaces are flatter above the cervical bulge in comparison with permanent molars.
14. Molars crowns have narrow occlusal table bucco-lingually due to occlusal convergence of buccal and lingual walls in primary teeth (Fig. 5.4).



Figs 5.4 A and B: Differences between primary and permanent teeth— occlusal table. A. Permanent, B. Primary



Figs 5.5 A and B: Differences between primary and permanent teeth—Pulpal morphology

15. Cusps are short, ridges are not pronounced and fossae are correspondingly not as deep.
16. Deciduous crowns have prominent mesial cervical ridge and rights can be differentiated from lefts easily.
17. The contact areas between primary molars are broader, flatter and situated more gingivally than between permanent molars.
18. Microscopically, the enamel rods in the gingival one-third of primary molars as they proceed towards enamel surface incline towards the occlusal, compared to permanent teeth where these rods incline cervically.
19. Comparatively in deciduous dentition the pulp horns are high and pulp chambers are relatively larger than in permanent dentition (Fig. 5.5).

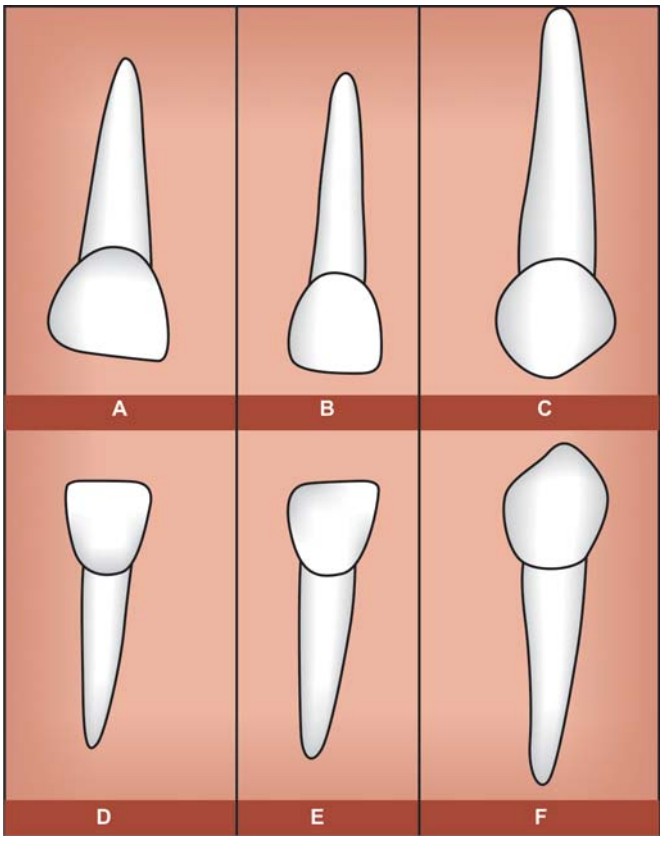
DIFFERENCES BETWEEN THE ROOTS OF DECIDUOUS AND PERMANENT DENTITION

1. The roots of deciduous teeth are fully formed about one year after eruption whereas the roots of permanent teeth take a longer duration about 3 years.
2. The resorption occurring in roots of primary teeth is physiologic resorption, whereas in permanent teeth only pathologic resorption occurs.
3. The roots of deciduous anterior teeth differ the roots permanent anterior teeth as follows:
   a. The roots of primary anterior teeth are narrower mesiodistally and proportionally longer as compared to crown size than the roots of permanent teeth.
   b. Usually roots of deciduous teeth incline labially in their apical third to one-half by 10 degrees.
4. The roots of deciduous posterior teeth differ from the roots of permanent posterior teeth as follows:
   a. The roots of deciduous molars are longer and more slender comparatively.
   b. The roots show furcation nearer to crown with very little or no root trunk.
   c. The roots flare out more extending beyond the mesiodistal and buccolingual outlines of the crowns.
   d. This flare provides more room for the development of permanent tooth crowns between the roots.

MORPHOLOGIC DESCRIPTION OF DECIDUOUS TEETH

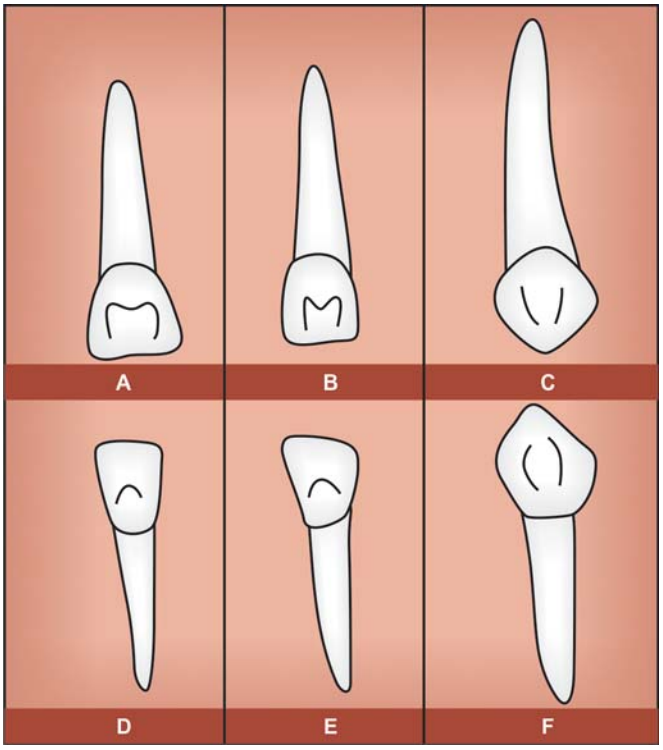
Maxillary Central Incisor (Figs 5.6 and 5.7)

1. Mesiodistal diameter of the crown is wider than the cervicoincisal length.



Figs 5.6 A to F: Facial surface. Maxillary—A. Primary incisors, B. Primary laterals, C. Primary canines. Mandibular—D. Primary incisors, E. Primary laterals, F. Primary canines

2. Labial surface appears smooth and absence of developmental lines.
3. Absence of mamelons.
4. Presence of slightly rounded distoincisal line angles.
5. Presence of flat incisal edges.
6. Contact areas are situated almost at same place as in permanent incisors.
7. Usually a cervical ridge is present on the labial surface which may be called as labial cingulum.
8. Presence of prominent marginal ridges on palatal aspect.
9. Cingulum is selectively incisors are slightly narrower on lingual side than on the labial side.
10. Convexity of cervical line towards the incisal is greater on mesial than on distal surface.
11. Root is almost straight with very slight bending lingually in the cervical third to half and labially in the apical half. The root in cone shaped with tapered slides.



Figs 5.7 A to F: Lingual surface. Maxillary—A. Primary incisors, B. Primary laterals, C. Primary canines. Mandibular—D. Primary incisors, E. Primary laterals, F. Primary canines

Maxillary Lateral Incisors (Figs 5.6 and 5.7)

1. The outline of maxillary lateral incisor is similar to that of central incisors, but the crown is smaller in dimensions.
2. The length of the crown cervicoincisally is greater than its mesiodistal width.
3. Distoincisal line angle is more rounded than the central incisor. Labial cervical ridge is slightly prominent.
4. The root outline is similar to that of the central incisor but is longer in proportion to the crown.

Maxillary Canine (Figs 5.6 and 5.7)

1. The crown of the maxillary canine is more constricted at cervical region than in the incisors.
2. Crown is slightly wider and longer.
3. Mesial and distal outlines are convex.
4. Labial cervical line is more curved than on mandibular canine.
5. It has a long, well developed and sharp cusp with two cusp ridges meeting at an acute angle.

6. The mesial cusp slopes are longer than the distal cusp slopes (which are the opposite of permanent canine)
7. Mesial and distal contact areas are almost at the center of crown cervico-incisally, the mesial contact is cervically located.
8. Mesial and distal cusp ridges are pronounced and bulky.
9. A prominent lingual ridge with adjacent mesial and distal fossa is located on the crown about 1mm distal to the middle of the crown.
10. Maxillary canine has longest root gradually tapering to a blunt apex root length is more than double the crown length.

Mandibular Central Incisors (Figs 5.6 and 5.7)

1. The mandibular central incisor is smaller than the maxillary central.
2. Its labiolingual width is usually 1mm less than that of maxillary centrals.
3. The labial surfaces are relatively smooth, but with slight depression in the incisal third.
4. In the lingual surface, marginal ridge is rudimentary in nature and has a small cingulum and small lingual fossa.
5. Root length is approximately twice to the length of the crown.

Mandibular Lateral Incisors (Figs 5.6 and 5.7)

1. Its outline is similar to that of the central incisor but is somewhat larger in all dimensions except labiolingually.
2. Lingual surface shows presence of greater concavity between the marginal ridges.
3. Incisal edge appears to slope towards the distal aspect of the tooth.

Mandibular Canine (Figs 5.6 and 5.7)

Mandibular canine is much similar to maxillary canine except for the following differences:

1. Crown is shorter and narrower labiolingually with shallow developmental grooves.
2. There is presence of a single lingual fossa.
3. The proximal surfaces resemble that of primary incisors.
4. The incisal ridge is straight and centered over the crown.

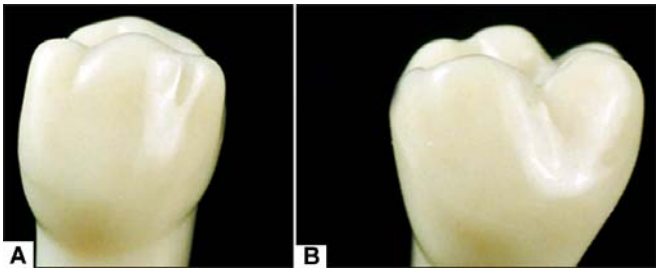
Maxillary First molar

Buccal Surface (Fig. 5.8)

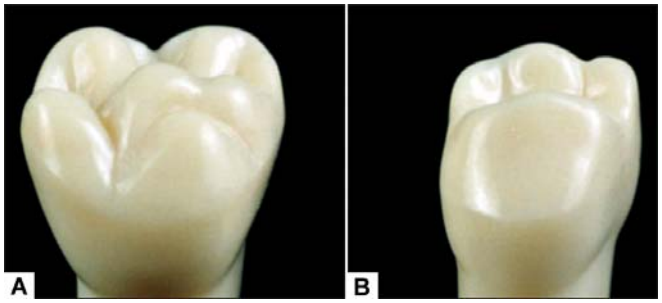
1. Greatest mesiodistal width at mesial and distal contact areas.
2. Absence of any development grooves.
3. Crown converges towards the cervical line from contact areas gradually with a mesiodistal width less by 2 min at the cervical region.
4. It is smaller in size than the second molar.
5. Presence of molar tubercle on the mesiobuccal corner of maxillary first deciduous molar called 'molar tubercle of Zuckerkandl'

Lingual Surface (Fig. 5.9)

1. Similar in outline like a buccal surface.
2. Mesiolingual cusp is the most prominent, longest and sharpest cusp of the tooth.
3. However distolingual cusp, if present is small and rudimentary.



Figs 5.8 A and B: Buccal view of maxillary primary molars. A. Primary 1st molar, B. Primary 2nd molar



Figs 5.9 A and B: Lingual view of maxillary primary molars. A. Primary 1st molar, B. Primary 2nd molar

4. All three roots are visible from lingual surface.
5. Crown converges in lingual direction.

Mesial Surface

1. Greater width at cervical third than on occlusal third.
2. Mesiolingual cusp longer than mesiobuccal cusp.
3. Cervical line may show slight curvature towards occlusal surface.

Distal Surface

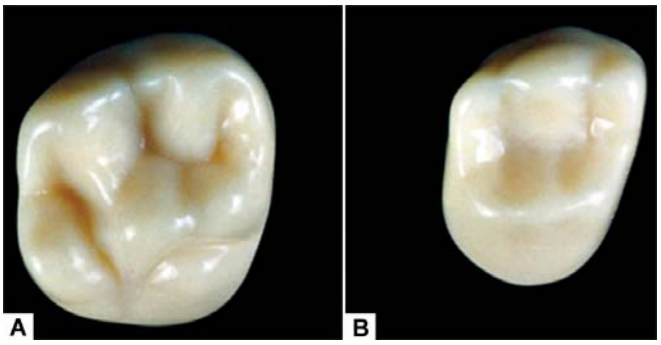
1. Crown tapers distally and hence it is narrower than mesial surface.
2. Distolingual cusp is small and distobuccal cusp is long.

Occlusal Surface (Fig. 5.10)

1. It resembles a maxillary premolar
2. Crown outline converges palatally and distally
3. Occlusal surface is nearly rectangular.
4. Presence of mesial and distal marginal ridges.
5. Shallow triangular and central fossa and grooves present.

Roots

1. Roots are slender and long spreading widely apart.
2. All three roots are visible from buccal aspect.
3. Distal root is relatively shorter than mesial root.
4. Trifurcation starts immediately from the cervical line.



Figs 5.10 A and B: Occlusal view of maxillary molars. A. Primary 1st molar, B. Primary 2nd molar

Maxillary Second Molar

Deciduous second maxillary molar resembles permanent maxillary molars, but are much smaller in size. Rarely there is one cusp of carabelli or a supplemental cusp.

Buccal Surface (Fig. 5.8)

1. Crown is narrow at cervix in comparison to its mesiodistal width at the contact area.
2. There are two well defined buccal cusps with a shallow buccal development groove.
3. Buccal cusps are almost equal in size.
4. Crown is larger than first deciduous molar.

Lingual Surface (Fig. 5.9)

1. Two lingual cusps present.
2. Mesiolingual cusp is large and well developed.
3. Distolingual cusp is well developed than in first deciduous molar.
4. Presence of well defined developmental groove separately the two lingual cusps.
5. Occasionally a third and rudimentary supplemental cusp is seen.

Proximal Surface

1. Mesial surface resembles that of permanent molars.
2. Dimension of distal surface is smaller than the mesial surface.
3. Cervical line shows very little curvature on mesial and distal aspects.

Occlusal Surface (Fig. 5.10)

1. Presence of a prominent oblique ridge that connects the mesiolingual cusp with the distobuccal cusp.
2. The occlusal outline is somewhat rhomboidal and has 4 well developed cusps and one supplemental cusp.
3. It has well defined mesial triangular fossa with a mesial pit at its center.
4. Both mesial and distal marginal ridges are equally well developed than in deciduous first molars.

Roots

1. Curvature of lingual root is similar to deciduous first molars.
2. Bifurcation point is between the mesiobuccal root and distobuccal root and the lingual root is 2-3 mm apical to the cervical line of the crown.

Mandibular First Molar

Unlike the other deciduous teeth, mandibular first deciduous molars do not resemble any other deciduous or permanent teeth.

Buccal Aspect (Fig. 5.11)

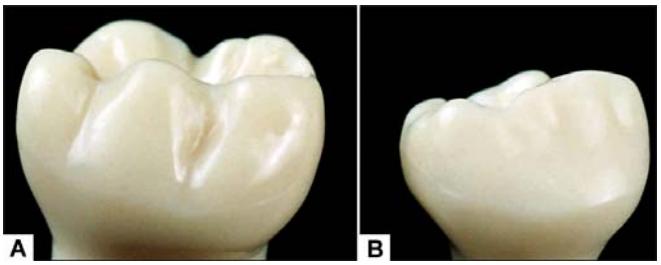
1. The constriction towards cervical line is very little.
2. Mesial portion of crown is longer than distal.
3. Mesial cusp is larger than distal cusp.
4. Mesial outline when viewed from buccal aspect is almost straight from the contact area to cervical line.
5. Absence of development groove.
6. Presence of a tubercle on the mesiobuccal corner

Lingual Aspect (Fig. 5.12)

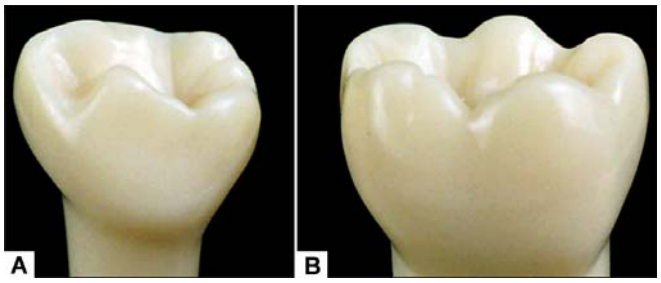
1. Presence of pronounced lingual convergence in the mesial aspect.
2. Mesiolingual cusp, being long and sharp at tip than distolingual cusp which is separated by a developmental groove.
3. Distolingual cusp is rounded and well developed.

Proximal Contours and Contacts (Figs 5.13 and 5.14)

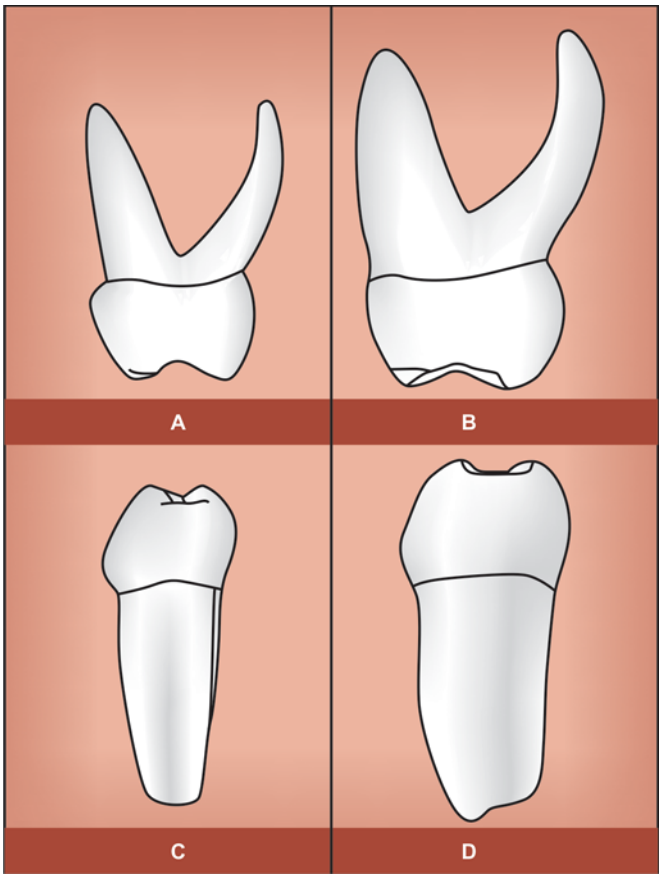
1. Mesial marginal ridge is well developed and more prominent and occlusally positioned than the distal marginal ridge.



Figs 5.11A and B: Buccal view of primary mandibular molars. A. Primary 1st molar, B. Primary 2nd molar



Figs 5.12A and B: Lingual view of primary mandibular molars. A. Primary 1st molar, B. Primary 2nd molar

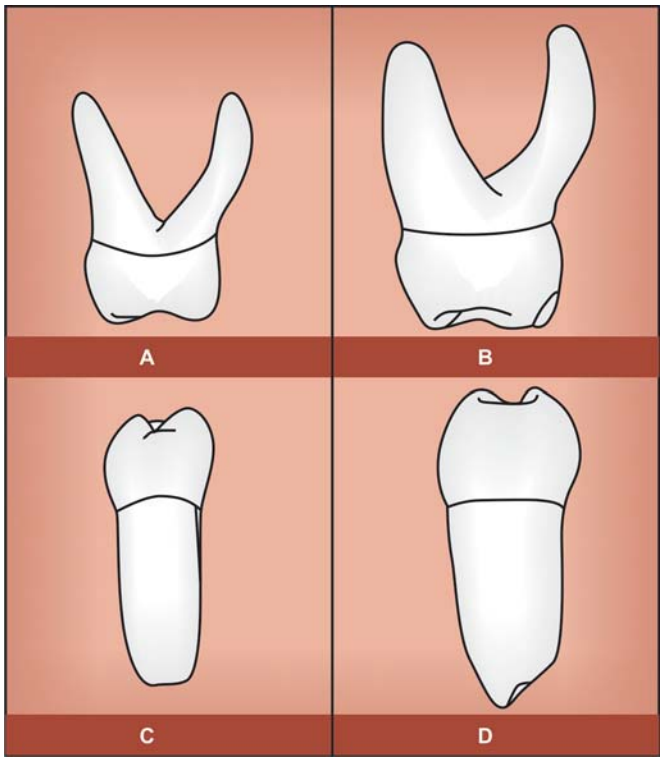


Figs 5.13A to D: Mesial aspect of primary molars. Maxillary—A. 1st molar, B. 2nd molar. Mandibular—C. 1st molar, D. 2nd molar

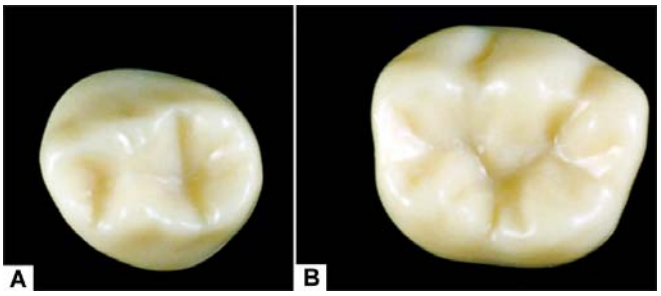
2. The cervical line on mesial side is convex towards occlusal; while on distal side it in relatively flat or horizontal.

Occlusal (Fig. 5.15)

1. Outline is rhomboidal (wider mesiodistally than buccolingually) in nature.



Figs 5.14A to D: Distal aspect of primary molars. Maxillary— A. 1st molar, B. 2nd molar. Mandibular— C. 1st molar D. 2nd molar



Figs 5.15 A and B: Occlusal view of primary mandibular molars. A. Primary 1st molar, B. Primary 2nd molar

2. The cusps in order of diminishing size are mesiobuccal, mesiolingual, distobuccal and the smallest distolingual cusp.
3. The characteristic feature of deciduous first mandibular molar is presence of transverse ridge which runs between mesiobuccal and mesiolingual cusps care should be taken not to undermine this ridge during cavity preparation.

Roots

1. There are two roots, mesial and distal.
2. Mesial being wider and longer than distal root.
3. Distal root is more rounded, less broad, thinner and shorter than mesial root.

Mandibular Second Molar

It resembles mandibular first permanent molar, except for its smaller dimension.

Buccal Aspect (Fig. 5.11)

1. Presence of cervical constriction
2. The mesiobuccal and distobuccal shallow developmental grooves divide the buccal surface into their cusps of almost equal size namely, mesiobuccal, distobuccal and distal cusp.
3. Distobuccal cusp is the widest.

Lingual Aspect (Fig. 5.12)

1. A short lingual groove divides the lingual surface into two cusps of almost equal size, but shorter than buccal cusps.
2. They are named as mesio and distolingual cusps.
3. When viewed from lingual aspect, the mesial portion of the crown seems to be little higher than the distal portion of the crown.

Proximal Contact and Contours (Figs 5.13 and 5.14)

1. Mesial marginal ridge is comparatively higher and is crossed by a thin and shallow groove that extends 2 mm on way down on mesial surface.
2. The contact area with first molar of the sharp of an inverted crescent just below the notch of marginal ridge.
3. Distal marginal ridge is more cervical than mesial marginal ridge.
4. The cervical line is regular having slight upward incline buccolingually on the distal and mesial sides.

Occlusal (Fig. 5.15)

1. Occlusal outline appears rectangular with slight distal convergence of the crown.
2. Mesial marginal ridge is well developed than distal marginal ridge.

3. Mesiodistal widths of three buccal cusps are more than the total width of two lingual cusps.
4. Presence of well defined triangular ridges; buccal, lingual and central developmental grooves.
5. The only difference between primary molar and the first permanent molar is the distobuccal cusp; the distal cusp of the permanent molar is smaller than the other two buccal cusps.

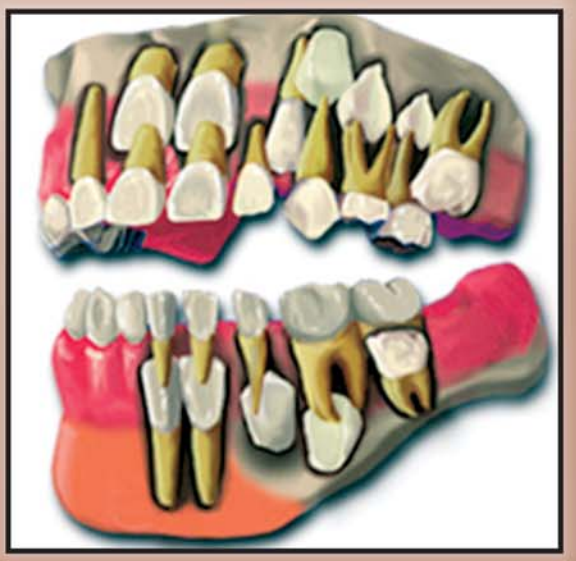
Roots

1. Roots are almost twice the length of the crown and are thin mesiodistally.
2. Mesial root is relatively broad and flat with a blunt apex and has a shallow longitudinal depression.
3. Distal root is less blunt at apex and is narrower than the mesial root.
4. Root furcation is very near to the cervical line with very little root trunk.

Pulps of Primary Teeth

1. Pulp chambers are relatively larger
2. Pulp horns are closer to the outer surface
3. Usually a pulp horn is located under each cusp.
4. Although there is great variation in size and location.
5. Pulp chambers are shallow.
6. Form of pulp canal/chamber follows the external anatomy.
7. More tortuous and irregular pulp canals are present.

6



Anomalies of Tooth Formation, Development and Eruption

INTRODUCTION

Development of tooth is a continuous process which undergoes several morphologic stages. Though the size and shape of individual teeth are different; they pass through the similar stages of development. Hereditary is believed to be the frequent cause for developmental disturbances.

Disturbances in size, shape, number, position, and structure of teeth may be due to disturbances in corresponding stages/physiologic phases of tooth development, being initiation, proliferation, histodifferentiation, morphodifferentiation, apposition phases.

Disturbances in initiation phase leads to alteration in number of teeth.

Disturbances in morphodifferentiation stage leads to alteration in size and shape.

Disturbance in apposition phase leads to alteration in structure of the teeth.

Commonly occurring developmental disturbances are discussed in Table 6.1.

SUPERNUMERARY TEETH

Etiology

May develop from third tooth bud arising from dental lamina near permanent tooth bud or possibly from splitting of permanent bud itself.

Supernumerary teeth are common in maxilla than mandible. Most common is 'Mesiodens', situated between maxillary central incisors. It is a cone shaped crown and short root.

Second most common is maxillary fourth molar, and other teeth with some frequency are maxillary paramolars, mandibular premolars, and maxillary lateral incisors.

Table 6.2 summarizes the disturbances in size of teeth.

DEVELOPMENTAL ALTERATIONS IN SHAPE

Gemination

1. It arises from an attempt at division of single tooth germ by invagination with resultant incomplete formation of two teeth.

Table 6.1: Commonly occurring disturbances in alterations in number of teeth

	Anodontia	*Hypodontia*	*Oligodontia*
Definition	Total lack of teeth of one or both dentition	Developmental absence of multiple teeth	Excess number of teeth
Etiology	Hereditary, frequently associated with hereditary ectodermal dysplasia	Autosomal dominant with incomplete penetrance, trauma, infections, radiation overdose, glandular dysfunction, systemic conditions like rickets, rubeola during pregnancy Absence of appropriate/sufficient segments of dental lamina	Recessive, autosomal or sex-linked, continued activity of dental lamina even after formation of normal number of tooth series or complete division of developing tooth buds
Clinical features	Total absence of teeth	Missing teeth: Primary, permanent and other abnormalities like fusion, ankylosis, short roots	Rudimentary or may be of normal size. Erupted or impacted as in case of mesiodens Supplemental teeth like paramolars, distomolars
Treatment	Rehabilitation with complete dentures or implant supported prostheses	Orthodontic space closure / rehabilitation with fixed or removable partial dentures/ implants	Surgical removal of supplemental teeth in case of crowding, impaction, infection, etc

Table 6.2: Developmental disturbances in size of the teeth

	Microdontia	*Macrodontia*
Definition	Teeth that are relatively smaller in size than normal	Teeth are relatively larger in size than normal
Classification	True generalized Relative generalized Involving a single tooth alone	True generalized Relative generalized Involving a single tooth alone
Description	*True generalized:* All teeth are smaller in size than normal *Relative generalized:* Normal or slightly smaller than the normal teeth that are present in the jaws that may seem to be larger than normal *Involving a single tooth:* Microdontia of lateral incisors commonly termed as "peg laterals" *Example:* X-linked hyphydrotic ectodermal dysplasia	*True generalized:* All the teeth are larger than in normal *Relative generalized:* Presence of normal or slightly larger than normal teeth in relatively smaller sized jaws *Involving single tooth alone:* It gives an illusion of fusion of two or more teeth *Example:* Associated with pituitary gigantism, unilateral facial hyperplasia and in hereditary gingival fibromatosis

2. It is seen in both the dentitions.
3. The structure is usually one with two completely or incompletely separated crowns that have a single root and root canal.

Fusion

1. Arises through union of two normally separated tooth germ.
2. Some physical force or pressure is thought to produce contact of developing teeth and their subsequent fusion.
3. If the contact occurs before calcification, two teeth may be completely united to form a single large tooth.
4. If the contact occurs later, there is union of roots only.
5. The tooth may have separate or fused root canals and is common in both dentitions.

Concrescence

1. This is a form of fusion which occurs after root formation.
2. The teeth are united by cementum only.
3. It is believed that it arises as a result of traumatic injury or crowding of teeth with resorption of interdental bone so that two roots are in approximate contact and become fused by deposition of cementum between them.

Dilaceration

1. It is an angulation or a sharp bend or curve, in the root or crown of formed tooth.
2. The condition is thought to be due to trauma during the period in which tooth is forming, with the result that position of calcified portion of tooth is changed and remainder of tooth is formed at an angle.
3. The curve or bend may occur anywhere along the length of tooth, sometimes at cervical portion, mid way along root apex of root.

Talon's Cusp

1. The Talon cusp, an anomalous structure, projecting lingually from cingulum areas of maxillary or mandibular permanent incisor.
2. The cusp blends smoothly with tooth except that there is a deep developmental groove where the cusp blends with sloping lingual tooth surface.
3. It is composed of normal enamel and dentin and contains a horn of pulp tissue.

Dens in Dente (Dens Invaginatus)

1. Presence of an invagination in the crown of tooth, forming an in folding lined by enamel within the crown of tooth, sometimes extending into the root.

2. Invagination of enamel epithelium into the dental papilla during development leads to formation of abnormality.
3. Maxillary lateral incisor is most commonly affected tooth. The condition is frequently bilateral.
4. Radiographically, it is pear shaped invagination of enamel and dentin with a narrow constriction at the opening on the surface of the tooth and closely approximating the pulp in its depth.
5. The problems which arise in invaginated teeth are develop of caries and pulpal pathology.

Dens Evaginatus

1. It is a developmental condition that appears clinically as an accessory cusp or a globule of enamel on the occlusal surface between buccal and lingual cusps of premolars.
2. It is thought to be the proliferation and evagination of an area of inner enamel epithelium and subjacent odontogenic mesenchyme into the dental organ during early tooth development.
3. The problems like incomplete eruption, pulp exposure following occlusal wear or fracture can occur.

Taurodontism

1. The body of the tooth is enlarged vertically at the expense of roots. The normal constriction of a tooth at the level of cementoenamel junction is reduced or absent.
2. Taurodont is caused by failure of Hertwig's epithelial sheath to invaginate at the proper horizontal level.
3. The furcation is displaced apically. The teeth involved are almost invariably molars.
4. The condition may be unilateral or bilateral.
5. Radiographically, the involved teeth may be rectangular in shape rather than taper towards the roots. The pulp chamber is extremely large.

DISTURBANCES IN STRUCTURE OF TEETH

Amelogenesis Imperfecta (Hereditary) (Figs 6.1 and 6.2)

1. It is a hereditary defects of enamel unassociated with any other generalized defects. It is an ectodermal disturbance, since mesodermal components are normal.
2. Depending on the stages of enamel development, the amelogenesis imperfecta can be;
   Hypoplastic type – Defective formation of matrix
   Hypocalcification type – Defective mineralization of matrix
   Hypomaturation type – Enamel crystallites remain immature.

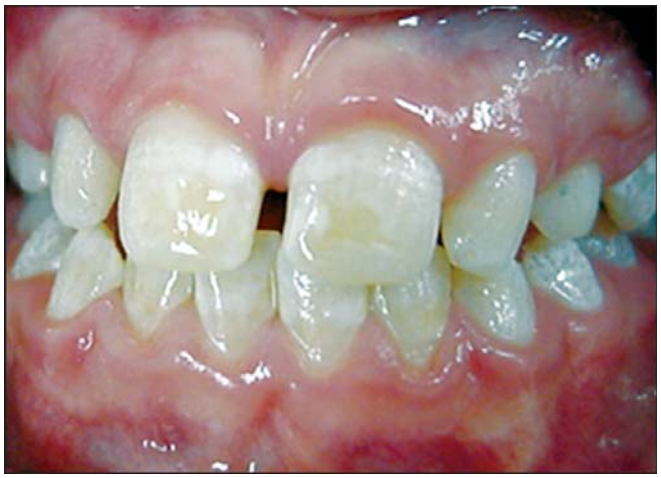


Fig. 6.1: Amelogenesis imperfecta—anterior teeth

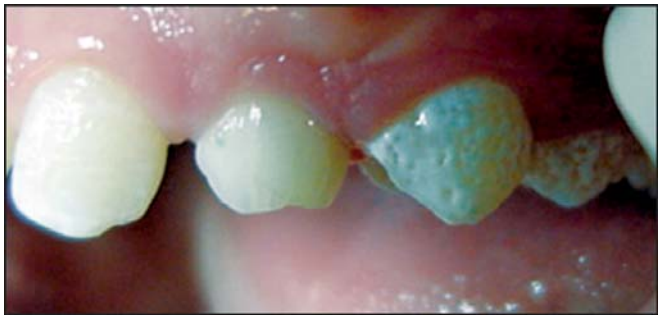


Fig. 6.2: Pitted appearance of enamel due to amelogenesis imperfecta

Etiology

Autosomal dominant, autosomal recessive and X-linked patterns of inheritance.

Clinical Features

1. *Hypoplastic type:* Enamel has not formed to full normal thickness on newly erupted developing teeth.

2. *Hypocalcified type:* Enamel is soft that it can be removed by prophylaxis instruments.
3. *Hypomaturation type:* Enamel can be pierced by explorer tip under firm pressure.

Environmental Enamel Hypoplasia (Fig. 6.3)

1. Local factors are capable of producing injury to ameloblasts and systemic disorders also play a role
2. Conditions like nutritional deficiency, exanthematous diseases (measles, chickenpox, and scarlet fever), congenital syphilis, and hypocalcemia, ingestion of chemicals, Rh hemolytic disease and idiopathic causes.
3. Dental fluorosis is considered to be one of the type of environmental enamel hypoplasia.

Fig. 6.3: Environmental hypoplasia—fluorosis

Dentinogenesis Imperfecta

1. It is an autosomal dominant inherited condition.
2. It occur in isolation or in association with osteogenesis imperfecta.

Table 6.3 summarizes the clinical characteristics of dentinogenesis imperfecta.

The color of teeth may range from grey to brownish violet or yellowish brown.

- Translucent or opalescent hue. Because of abnormal dentinoenamel junction, the enamel may be fractured.
- This scalloping of junction which tends to form an interlocking union between enamel and dentin is absent. So early loss of enamel, the dentin undergoes rapid attrition.

Dentin Dysplasia (Table 6.4)

It is an autosomal dominant trait with both dentitions being affected.

Regional Odontodysplasia

1. It is a localized disorder of tissues of dental origin resulting in characteristically bizarre clinical and radiographic appearances.
2. Somatic mutation affecting dental lamina, viruses, local circulatory disorders, pharmacotherapy, pregnancy neural disorders, Rh incompatibility, migration of neural crest cells can be few causes.
3. They exhibit delayed or total failure is eruption.
4. Evidence of defective mineralization.
5. Since there is reduction in radiodensity – teeth assume a ghost appearance radiographically.

DISTURBANCES OF ERUPTION

Eruption time of permanent teeth in female tends to be slightly ahead of eruption times in males.

Table 6.3: Dentinogenesis imperfecta

Type I	Occurs in families with osteogenesis imperfecta Autosomal dominant in nature	Deciduous dentitions are more commonly affected than the permanent dentition	Obliteration of pulp chambers, root canals. Roots are short and blunted
Type II	Dentinogenesis imperfecta not associated with osteogenesis imperfecta	Both primary and permanent dentitions are equally affected	
Type III	Brandy wine type Autosomal dominant in nature	Both primary and permanent dentition are affected	"Shell teeth" Enamel appears normal while dentin is extremely thin with large pulp chambers

Table 6.4: Dentin dysplasia			
Type I (Radicular)	Both primary and permanent dentitions are affected	Amber like translucency	*Primary teeth:* Obliteration of pulp chambers and root canals *Permanent teeth:* Crescent shaped Pulpal remnants
Type II (Coronal)	Both primary and permanent dentitions are affected	Yellow, brown, bluish grey opalescence	*Primary teeth:* Obliteration of pulp chambers *Permanent teeth:* Abnormally large pulp chambers in coronal portion with characteristic " thistle tube" appearance

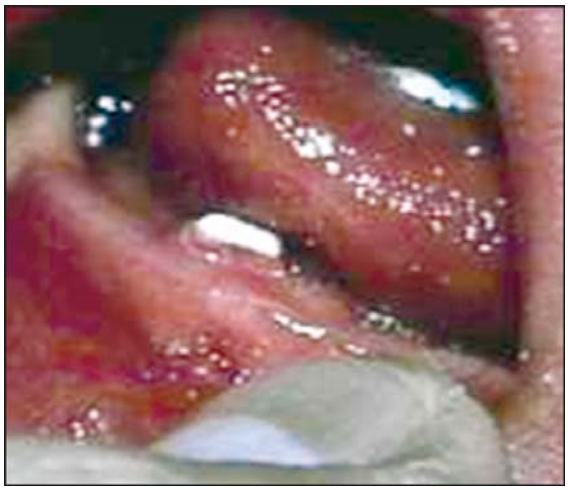


Fig. 6.4: Natal teeth

Premature Eruption

1. Children with high birth weight, with precocious puberty, endocrine abnormalities, growth or thyroid hormones show premature eruption.
2. Teeth present at birth are natal teeth and those that erupt within first month of life—Neonatal teeth (Fig. 6.4).

Delayed Eruption

1. Can be due to systemic or local factor.
2. Associated with prematurity or low birth weight.
3. Down's syndrome, Turner's syndrome, nutritional abnormalities, endocrine disorders.

Impacted and Embedded Teeth

Embedded teeth are individual teeth which are unerupted usually because of lack of eruptive force, while impacted teeth are those prevented from erupting by some physical barrier. The common impacted teeth are mandibular third molars followed by maxillary 3rd molars, maxillary canines.

Based on the position, they are classified into:

i. Mesioangular
ii. Distoangular
iii. Vertical
iv. Horizontal.

DISTURBANCES OF EXFOLIATION

The normally short life period of deciduous teeth belies their importance in maintaining the integrity of the dental arch in an individual. Disturbances in the normal shedding time of the primary teeth lead to a spectrum of changes in the dentofacial region causing little to serious malocclusion problems. The distrubance to normal shedding cycle may be due to an accident or disease, possible disturbances of shedding of primary teeth includes:

- Early or premature loss
- Retarded shedding

Early or Premature Loss of Deciduous Teeth

- The premature loss of primary teeth due to accident or disease can be the first step in a series that can end with severe malocclusion and shortened life for the entire dentition.
- Early loss of anterior teeth does not have any serious effects on the adjacent teeth and usually does not require space maintenance except for the aesthetic requirements of the patient has to be looked for.
- Premature loss of first deciduous molars also have a little effect on space and usually the space closure

in such cases results from distal drift of the crowded anterior which does not lead to net loss in arch length (it is mere redistribution of space)

- However premature loss of second deciduous molars in lower arch are of great concern due to the possible risk of permanent first molars drifting into this space and leading to locking off the premolars from erupting in its space and leading to serious malocclusion. These cases require space maintenance.

Ankylosed Primary Teeth (Submerged Teeth)

- Roots of the primary teeth usually resorb under stimulus from the erupting permanent teeth below. Retardation or failure of this physiologic process may be due to the displacement or absence of the permanent tooth or due to periodontal abnormalities in the deciduous tooth (periapical infections, failure of resorptive process and ankylosis) or genetic influence (debatable).
- Ankylosis is the result of a breakdown of the periodontal membrane that results in a direct bony union between the root of the tooth and the alveolar bone. This abnormal calcification across the flexible adjustment zone prevents the normal physiologic resorption.
- The first clinical symptom is usually an apparent submerged appearance of the affected tooth.
- Any primary tooth not in occlusal contact with its opposing tooth should be suspected for ankylosis and tested clinically by tapping lightly with an instrument handle; an Ankylosed tooth will produce a sharp, clear, ringing sound when compared to a tooth with the normal soft tissue support between tooth and alveolar bone.
- Comparison with adjoining teeth provides a convenient standard that is already adjusted for individual patient differences, but allowance must also be made for relative root area, which can also affect the sound.
- Dental radiographs may occasionally show ankylosis, but it is difficult to strictly diagnose based only on radiographs because the affected areas may not be in the profile perimeter that is seen on the film.
- Severity of the clinical problems due to ankylosis varies depending on the growth that takes place between the time of ankylosis and normal shedding time of the ankylosed tooth; thus it is largely dependant on the age of the patient/individual.
- Ankylosis can lead to various problems such as space loss, over/supra eruption of opposing tooth, disruption of the occlusal plane, etc.
- Most common submerged teeth–Mandibular 2nd molars.

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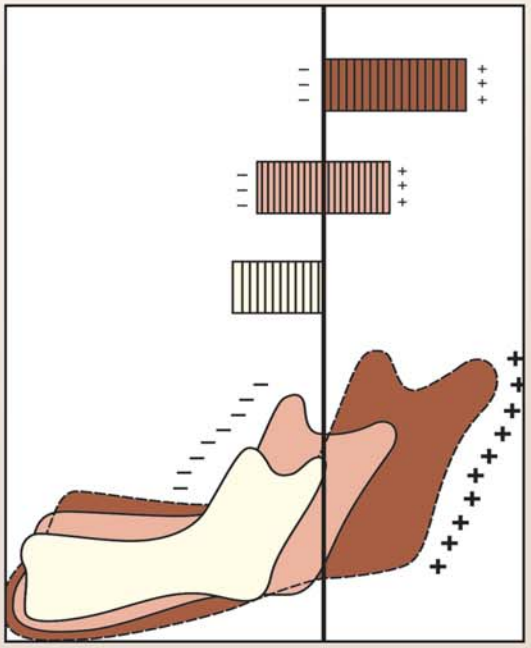

Growth and Development

INTRODUCTION

Growth is a dynamic process. The complex nature of growth and development fascinates the scientist's even till date. The very terms growth and development can cause difficulties in understanding; though closely related, are not synonymous.

Growth is defined as an increase in size (Todd).

Development is defined as progress towards maturity (Todd). Development may also be defined as all the natural sequential series of events between fertilization of ovum and the adult state.

Maturation is defined as the stabilization of the adult stage brought about by the growth and development.

A thorough background in craniofacial growth and development is necessary for every dentist.

VARIABLES INFLUENCING GROWTH

1. Genetic factors
2. Maternal factors
3. Environmental factors.

Genetic Factors

Hereditary/genetic factors are the basic influencing effects on growth of an individual. These factors make up for the basic inherent potentials for the individuals and it tunes the growth pattern and rhythm.

Maternal Factors

Maternal factors contain a number of variables that influences growth. They are:

a. Nutritional status of the pregnant mother
b. Health status of the mother
c. Endocrine factors
d. Emotional status
e. Uterine and placental condition
f. Family size and birth order
g. Socioeconomic factors.

Environmental Factors

Environmental factors may have stronger influence on growth at certain instances. Environmental factors include:

a. Socioeconomic factors
b. Endocrine factors
c. Nutrition
d. Climatic and seasonal effect
e. Habits
f. Health status.

NORMAL HUMAN GROWTH

From the time of conception to death, a differential dynamic process of growth occurs sequentially through different phases. These phases are briefly summarized as follows:

A. Prenatal growth
   a. The period of ovum
   b. The period of embryo
   c. The period of fetus.

B. Postnatal growth
   a. At birth
   b. 3-6 years
   c. 6-12 years
   d. > 12 years

Prenatal Growth

It is a phase where height is increased by 5,000 fold and weight is increased by 6.5 billion fold from stage of ovum to birth.

Period of Ovum (Fertilization–End of 14th Day)

This period primarily comprises of cleavage of the ovum and its process of implantation into uterine wall. At the end of this period the ovum is 1.5 mm length without any cephalad differentiation.

Period of Embryo/Embryonic Period (14th-56th Day/2-8 Weeks)

The period of embryo is characterized by a series of dynamic changes with growth at its highest phase. The following events occur during the period of embryo:

i. Formation of branchial arches
ii. Development of perioral region
iii. Formation of nasal pits
iv. Development of craniomaxillary complex, Comprising of cranium, palate, tongue, maxilla and mandible, etc.

By the end of embryonic period the embryo measures 1½-2 inches I length with significant cephalad differentiation.

In this stage sexual differentiation occurs; with greater growth proportionality seen in head region than the rest of the body.

Period of Fetus/Fetal Period (3rd-9th Month/57th-270th Day)

At this phase embryo develops into "fetus".

1. By third month face assumes a more human appearance. The head is erect and the bridge of the nose becomes prominent. Ears migrate from the lower corners of the face to a horizontal plane at the level of eyes.
2. From 12th-36th week head increases in length.
3. Cranium to face ratio is about 5:1.
4. Due to relatively greater increase in mandibular size, maxillomandibular relation approaches that of a newborn infant.
5. Breadth of the palate increases more rapidly than its length.

Postnatal Growth

At Birth

Skull at birth (Fig. 7.1):

a. Fontanelles are wide and not closed which are located at 6 sites:
   i. Anterior
   ii. Posterior
   iii. Right and left anterolateral
   iv. Right and left posterolateral.
b. Skull contains 45 separate bones.
c. *Facial features:*
   - Size of head is greater than the body
   - Lack of prominence of chin
   - Forehead is larger than face
   - Eyes are small
   - Tiny mouth.

By 1 year, the infant shows highest growth rate in his/her entire life. From 2-3 years growth is slow and steady until puberty.

3-6 Years

i. Fusion of separate bones occur gradually.
ii. Soft tissue prominences continues to increase in nose and mandible.
iii. Cartilaginous tissues get replaced by osseous tissue and bone calcification increases.
iv. Palatal descent occurs due to sutural growth and sequential apposition and deposition of bone on oral and nasal surfaces.
v. Facial growth and differentiation continues.

6-12 Years

Proportional body changes are the hallmark of this. Stage with growth of extremities significant Neural and cranial growth nears stage of completion. Endocrinal influences begin to establish at end of 11th-12th years.

>12 Years

This age is marked by influencing endocrinal factors on growth characterized by attainment of puberty with associated physical and sexual characteristics. There is increase in muscle mass, redistribution of fat and increased skeletal growth and development of secondary sexual characters and development of genital tissue. Puberty changes include:

i. Increase in body size including the attributes of height, weight and body proportion.
ii. Functioning of primary sexual characteristics.
iii. Development of secondary sexual characteristics like
   - Change in voice
   - Distribution of hair in facial, armpits, pubic and body

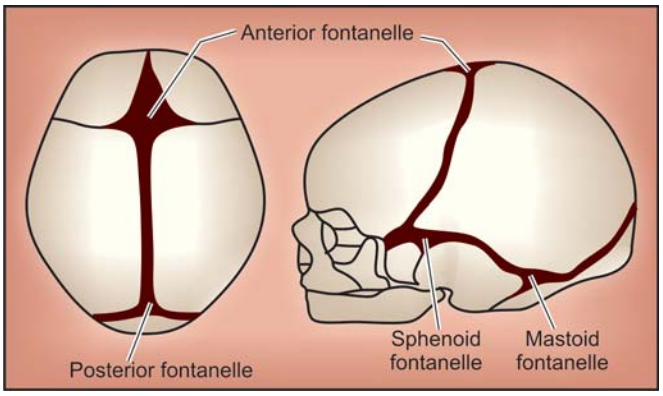

Fig. 7.1: The fontanelles of the new born skull

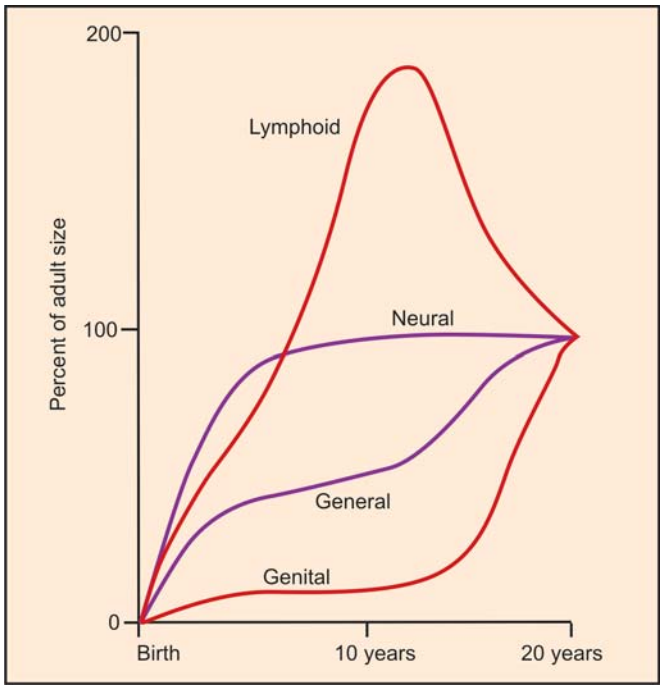

Fig. 7.2: Scammon's growth curve

- Development of genitals and breast development in females, etc.

GROWTH PATTERN

Pattern in growth is a complex factor. It is proportionality of growth over a period of time. A characteristic feature of normal growth pattern is that not all tissue systems of the body grow at the same rate. Which is best illustrated by Scammon's growth curve that explains the growth of the four major tissue systems of the body (Fig. 7.2).

Mechanisms/Theories of Growth

Mechanisms of craniofacial growth are briefly summarized as follows:

i. Genetic theory
ii. Sicher's sutural dominance theory
iii. Scott's hypothesis
iv. Enlow and Bang's "V" principle or area relocation theory
v. Functional matrix theory of Moss.

Genetic Theory

This theory suggests dominance of genetic influence in growth. According to this theory growth is genetically determined.

Sicher's Sutural Dominance Theory (1955)

Postulated by sicher, this theory states that sutures are responsible for growth of the cranial vault. It emphasizes the role of cartilaginous, osseous and periosteal tissues in growth. It states that the sutures calcify and force the bones apart thereby causing an expansile force or action.

According to Sicher, paired parallel sutures attaching nasomaxillary complex, grows forwards to pace its growth with that of mandible. This theory assigns more active role for sutures, hence called sutural dominance theory.

However, it is disregarded due to various experimental studies where growth continued even in the absence of sutures.

Scott's Hypothesis

This theory emphasizes the role of intrinsic growth factors that are cited in cartilage and periosteum are primary while the sutures being only secondary and dependent on extrasutural influence.

He states that cartilages are primary centers of growth. According to him, growth is in response to synchondrosis proliferation and local environmental factors.

There are experimental evidences to support the Scott's hypothesis. Which states sutural growth is secondary synchondrosis growth which occurs at the same time.

1. Atraumatic resection of nasal septum causes significant interference to mid face or maxillary growth.
2. Growth does not lease to occur in cleft palate cases where there is no suture.

Enlow and Bang's "V" Shaped Principle or Area Relocation Theory (Fig. 7.3)

This theory emphasizes that growth is a complex, multidirectional, dynamic process that continues, specific local areas come to occupy new actual positions in succession which involves continuous growth changes, sequential remodeling to maintain the shape, relative position and proportion of each individual area.

According to this theory, there is sequential resorption and apposition of bone on different surface of bone causing bone on different surface of bone causing bone remodelling. Facial and cranial bones posses a 'V' shaped configuration; thereby bone apposition occurs on the

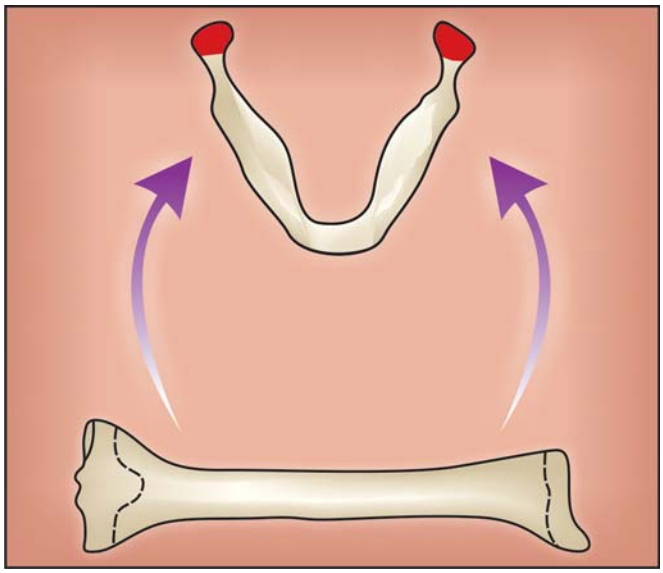

Fig. 7.3: Enlow and Bang's expanding 'V' principle

inner surface of 'V' while resorption occurs on the outer surface. Thus the 'V' moves away from tip and enlarges simultaneously with uniform growth and proportional increase in overall size.

Functional Matrix Theory of Moss

This theory is based on functional cranial component theory of Van der Klaauw. According to this theory there are nine different functional cranial components in craniofacial skeleton, each carrying out specific functions.

He states that each functional cranial component comprises of skeletal unit and functional matrices and in turn skeletal units are made up of micro-and macro-skeletal units while functional matrix is made up of periosteal and capsular matrix.

Moss claims that biomechanical role is to protect and or support its specific functional matrix.

For example mandible is a macroskeletal unit with coronoid process, condyle, angle of mandible and alveolus being macroskeletal units. Periosteal matrices like muscles and teeth act directly on microskeletal units in bringing about active growth; which alters the form of their respective skeletal units. Capsular matrices act on macroskeletal unit (mandible) and bring about translocation or passive growth.

POSTNATAL GROWTH

Postnatal Growth of Cranial Base (Fig. 7.4)

Cranial base grows at several cartilaginous sutures by synchondroses. There are four synchondroses at the cranial base namely:

1. Sphenooccipital
2. Spheno-ethmoidal
3. Intersphenoid
4. Intraoccipital

Sphenooccipital Synchondrosis (Fig. 7.5)

It is the most important growth site located between sphenoid and occipital bones in the cranial base which is active throughout the growing period and closes at late adolescence or early adulthood (14 years in girls and 16 years in boys).

The growth occurring at synchondrosis is directed upwards and forwards thereby carrying the upper part of face and anterior half of the cranial base bodily upwards and forwards. It is known primarily to accommodate the growing brain and upper respiratory tract.

Spheno-ethmoidal Synchondrosis

It is situated between sphenoid and ethmoidal bone. The growth is complemented by the growth of cartilage between the mesethmoid and frontal bone. The frontal bone subsequently allows increase in thickness by pneumatization and formation of frontal sinus.

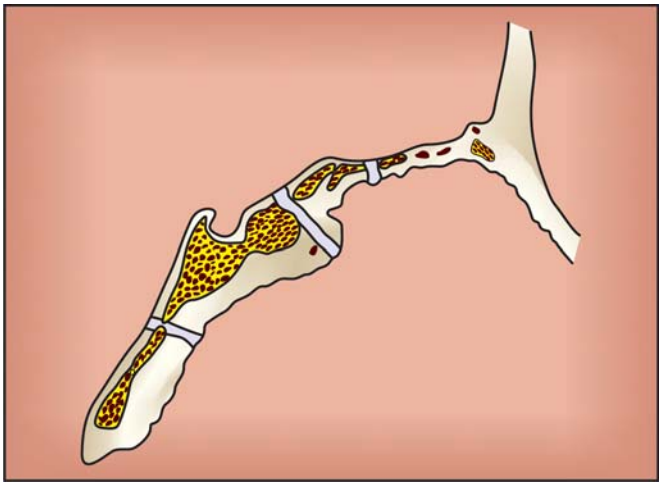

Fig. 7.4: Diagrammatic representation of synchondroses in the cranial base

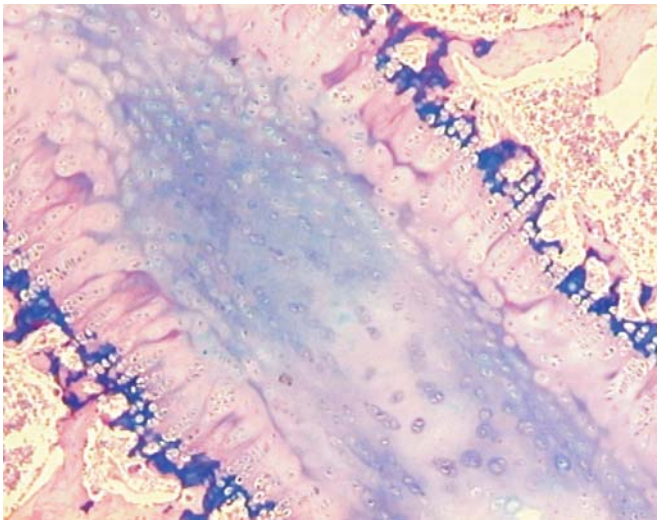

Fig. 7.5: Photomicrograph showing spheno-occipital synchondrosis

Spheno-ethmoidal synchondrosis ceases to grow by the time of eruption of first permanent molar.

Intersphenoid Synchondrosis (Fig. 7.6)

It is a cartilaginous tissue at the junction of two sphenoid bones. It ossifies at birth and ceases to grow beyond.

Intraoccipital Synchondrosis

Intraoccipital synchondrosis closes between 3rd and 5th years of life.

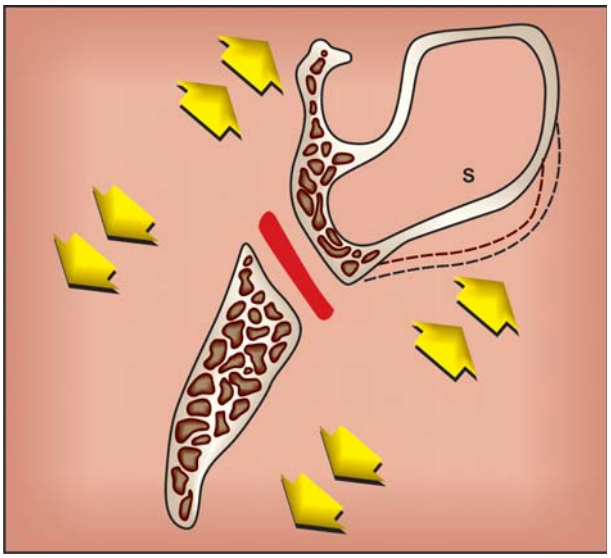

Fig. 7.6: Diagrammatic representation of intersphenoid synchondroses

Postnatal Growth of Cranial Vault

The cranial vault is made up of number of flat bones that are formed directly by intramembranous bone formation, without cartilaginous precursors. Cranium grows because the brain grows. This growth which is accelerated during infancy reaches to about 90% completion by the end of 5th year of life.

From the time that ossification begins at a number of centers that foreshadow the eventual anatomic bony units, the growth process is entirely the result of periosteal activity at bone surfaces. Remodeling and growth occur primarily at the periosteum-lined contact areas between adjacent skull bones, the skull sutures, but periosteal activity also changes both the inner and outer surfaces of these bones.

At birth, the flat bones are separated by the fontanelles which are open spaces that allow considerable amount of deformation of the skull at birth during descent through birth canal in delivery.

After birth, apposition of bone along the edges of the fontanelles eliminates these open spaces, but the bone remains separated by a thin periosteum lined sutures, which eventually fuses in adult life.

Apposition of new bone at these sutures is the major mechanism for growth in the cranial vault. Although growth in cranial vault occurs along the sutures; there is concomitant resorption along inner surface of the cranial vault along with apposition of new bone on the outer surface of the cranial vault.

This remodeling of inner and outer surfaces allows for changes in contour during growth.

Growth of Maxilla (Nasomaxillary Complex)

Maxilla at birth shows following characteristic features:

- Absence of Anterior nasal spine
- Very minimal height of the alveolar process, long enough to hold the deciduous teeth
- Presence of infra-orbital foramen close to the alveolar margin
- Presence of large frontal process due to relatively large orbit.

Maxilla develops postnatally entirely by intramembranous ossification. Growth occurs by two mechanisms.

i. By apposition of bone at sutures connecting maxilla to the cranium and cranial base
ii. By surface remodeling.

Sutural Growth (Fig. 7.7)

Sutures that connect maxilla to cranial vault and cranial base are:

a. Frontonasal
b. Frontomaxillary
c. Zygomaticotemporal
d. Zygomaticomaxillary
e. Pterygopalatine

The sutures attaching the maxilla posteriorly and superiorly are ideally situated to allow its downward and forward repositioning. The proliferation of bone along sutures moves maxilla in the downward and forward direction.

Surface Remodeling

Bone apposition occurs on both sides of the suture, so the bone to which the maxilla is attached also becomes larger. Part of the posterior border of the maxilla is a free surface in tuberosity region; bone apposition occurs along this surface to create space into which primary and later permanent molars erupt.

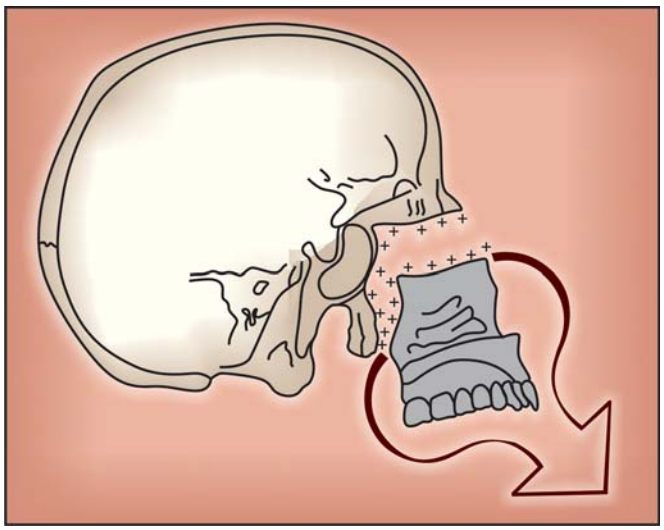

Fig. 7.7: Sutural growth of maxilla

As the maxilla grows downward and forward its facial and anterior surface gets remodeled by resorption along the anterior surface although the anterior surface is moving forward while new bone apposition occurs along superior and posterior sutures.

At the same time bone is removed on the nasal side and added on oral side, thus creating an additional downward and forward movement of the palate. Immediately adjacently, however, the anterior part of the alveolar process is a resorptive area, so removal of bone from the surface here tends to cancel some of the forward growth that otherwise would occur because of translation of the entire maxilla (Fig. 7.8).

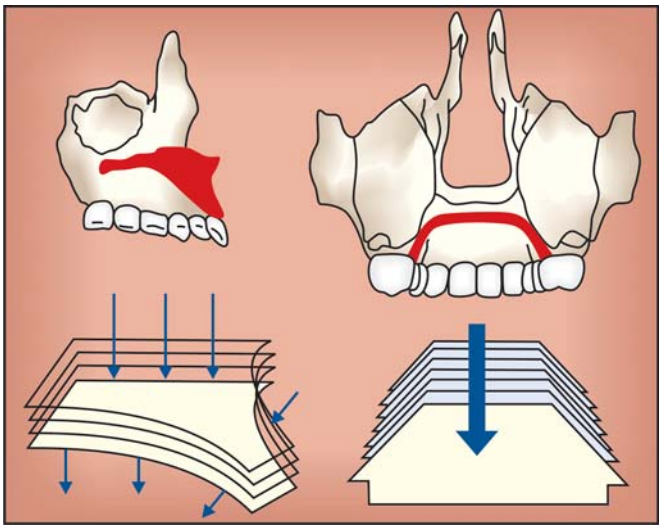

Fig. 7.8A: Surface remodeling of the palatal vault

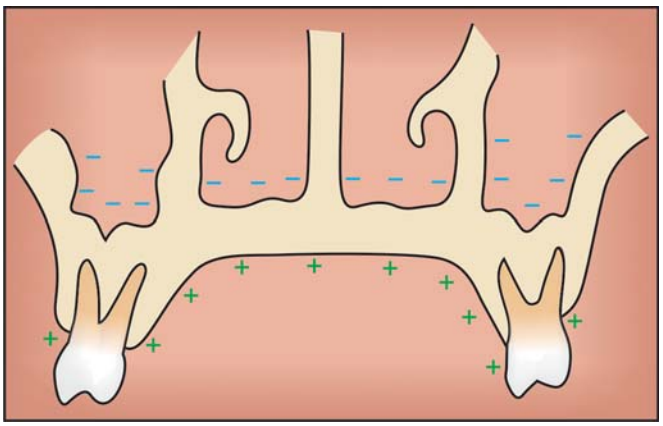

Fig. 7.8B: Surface remodeling in nasomaxillary complex a schematic representation

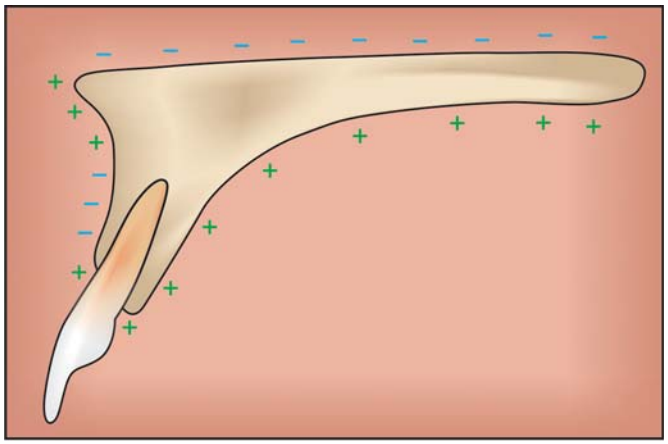

Fig. 7.8C: Surface remodeling in nasomaxillary complex a schematic representation indicates resorption and + indicates apposition of bone

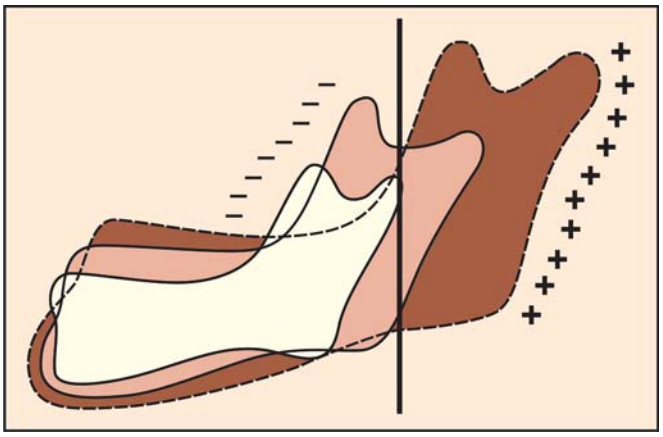

Fig. 7.10: Postnatal growth of mandible

Postnatal Growth of Mandible (Fig. 7.10)

Mandible at birth (Figure 7.9) is of the following dimensions:

- The anterior height of the mandible is approximately in 10 mm.
- The length of the mandible is approximately 35 mm.
- The ramus of the mandible measures 20 mm in length.
- The mental foramen is directed forwards.
- Presence of large mandibular angle (130° which changes to 120° in females and 115° in males in due course) and relatively large coronoid process.

At birth, the mandible is large enough to hold all the deciduous teeth and it is well-developed at the front but need to grow at the back to make space for the permanent molars. All the teeth lie in a trough, separated from each other by bone partitions and their crowns are just covered by mucosa, not by bone.

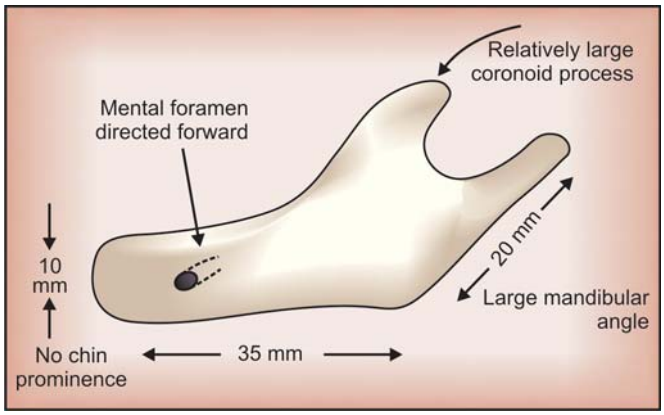

Fig. 7.9: Mandible at birth

Unlike maxilla, mandible grows both by endochondral and periosteal activity. Postnatal growth along the condyle at temporomandibular joint occurs by hyperplasia, hypertrophy and endochondral replacement within the cartilage; all the other areas of the mandible are formed and grow by direct surface apposition and remodeling.

Secondary cartilaginous fusion at the midline joint/symphysis occurs around 6 months after birth after which the mandible becomes a single bone; as a growth site, the chin is almost inactive.

It is translated downward and forward, as the actual growth occurs at the condyles and along the posterior surface of ramus of the mandible. The body of the mandible grows longer by periosteal apposition of bone on its posterior surface while ramus grows higher by endochondral replacement at the condyle accompanied by surface remodeling.

Conceptually mandible gets translated in downward and forward direction, while at the same time increases in size by growing upward and backward direction. Thereby bone apposition occurs along posterior surface of the ramus while resorption occurs along the anterior surface of ramus.

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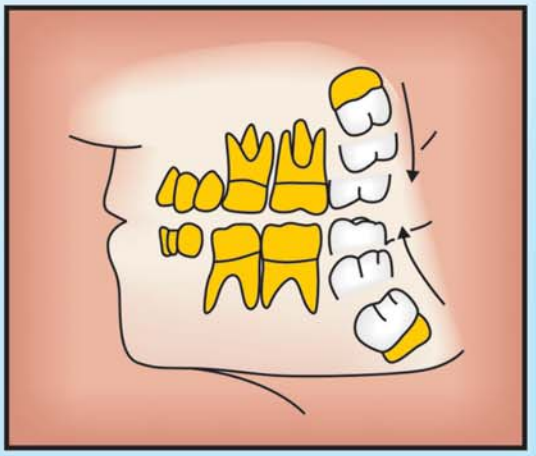

Development of Dental Arch and Occlusion

INTRODUCTION

As a general rule, the characteristic changes associated with growth and developments are continuous. But from the clinical point of view, there is a need to classify these continuous changes into several characteristic stages. In order to evaluate the growth of children, dental age is more clinically useful than chronological age, because it is based on the development of dentition. The Hellmann's classification (Table 8.1) is well known as the classical and traditional evaluation of dentitional development. Another such classical example in classification of stages of occlusion is by Barnett's classification which has greater clinical value (Table 8.2).

Table 8.1: Hellmann's stages of development of occlusion

Stage I	A	Before eruption of primary tooth
	C	Before completion of primary occlusion
Stage II	A	Completion of primary occlusion
	C	Eruptive phase of permanent first molar or incisors
Stage III	A	Eruption of first permanent molar or incisor completed
	B	Exchange phase of lateral teeth
	C	Eruptive phase of permanent second molar
Stage IV	A	Eruption of permanent second molar completed
	C	Eruptive phase of permanent third molar
Stage V	A	Eruption of permanent third molar completed

Table 8.2: Stages of occlusal development from the clinical point of view (Barnett, 1978)

Stages	*Age in years*	*Characteristics*
First stage	3	Primary dentition
Second stage	6	Eruption of first permanent molars
Third stage	6-9	Exchange of incisors
Fourth stage	9-12	Exchange of the lateral teeth
Fifth stage	>12	Eruption of the second molar

Another common clinically applicable classification is as follows:

I. Pre-dentate phase.
II. Deciduous dentition phase.
III. Mixed dentition phase.
  i. First transitional period
  ii. Intertransitional period
  iii. Second transitional period.
IV. Permanent dentition phase.

PREDENTATE PERIOD

This is the period of edentulism starting from birth until eruption of first primary tooth in the oral cavity. The alveolar ridge at this stage is termed to as "gum pads" which is briefly summarized as follows.

Features of Gum Pads (Fig. 8.1)

The alveolar ridge from birth until the eruption of first primary teeth is referred to as 'gum pads'. Gum pads are horseshoe shaped; pink, firm structures seen along maxilla and mandible.

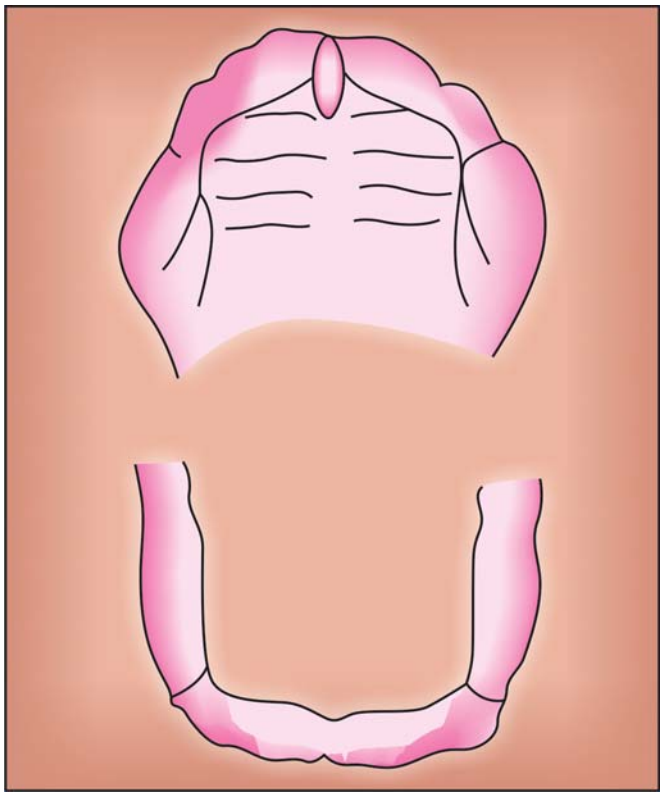

Fig. 8.1: Gum pads

The gum pad develops into two parts the lingual and labiobuccal portions which are separated by a dental groove. The upper gum pad is wider and larger than the lower gum pad. The gum pads are divided into ten segments by ten grooves called as transverse grooves. Each of these ten segments corresponds to space for on developing deciduous tooth.

The transverse groove situated in the canine region; i.e. between canine and first deciduous molar segment

in deep and is referred to as lateral sulcus which predicts the interarch relationship at an early stage. Lateral sulci are more distally located in the mandibular arch than in the maxillary arch. The gum pad is separated from the rest of the palate and floor of the mouth in upper and lower arch by a groove called as gingival groove.

Relation of Gum Pads (Fig. 8.2)

At rest the upper and lower gum pads are separated from each other by the positioning of the tongue. On occlusion/approximation; there is contact between upper and lower gum pads in the first primary molar region. The gum pads do not have a definite relationship when occluded in most of the times.

When occluded there is a complete overjet all around and open bite in the anterior region; this infantile open bite is considered to be normal and it helps in suckling/feeding, tongue will be occupied in between the two gum pads. This physiological phenomenon helps the infant during breastfeeding.

At birth, the gum pads are not wide enough to accommodate the developing incisors which are crowded and rotated in their crypts; later during the first year of life they increase in width sufficient enough to accommodate developing incisors. Labiolingual width and length of the gum pads increases considerably as growth progresses.

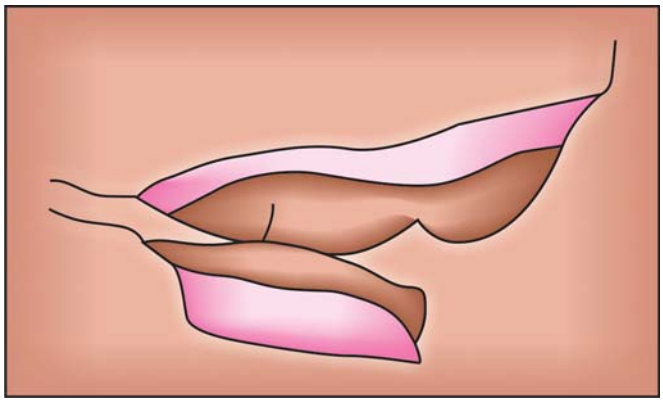

Fig. 8.2: Relation of gum pads

DECIDUOUS DENTITION PHASE

Chronology and sequence of eruption of primary teeth are already explained in detail in earlier chapters; however, it is summarized as follows:

Initiation of primary tooth buds occur during the first 6 weeks of intrauterine life. The eruption of the first primary tooth begins at about 6 months after birth, and all primary teeth are usually erupted by 2½ years of age, when second primary molar comes into occlusion. However, at their age, the rots of the second primary molars are usually not yet complete. Therefore, the establishment of the primary dentition is usually around 3 years of age when the roots of second primary molars complete their root development, and to last until about 6 years of age when the first permanent tooth begins to erupt.

From 3-4 years of age; the dental arch is relatively stable and changes very slightly. From 5-6 years of age, the size of the dental arch begins to change due to eruptive force of the first permanent molar.

Characteristic Features

The characteristic features of deciduous dentition period are as follows:

Spaces in the Primary Dentition (Figs 8.3 and 8.4)

It is very common to find physiological spaces in the primary dentition, with the most prevalent spaces mesial to the primary canine in maxilla and distal to the primary canine in mandible. These spaces are called the primate spaces on anthropoid spaces or simian spaces which are helpful in canine positioning and relationship with opposing arches.

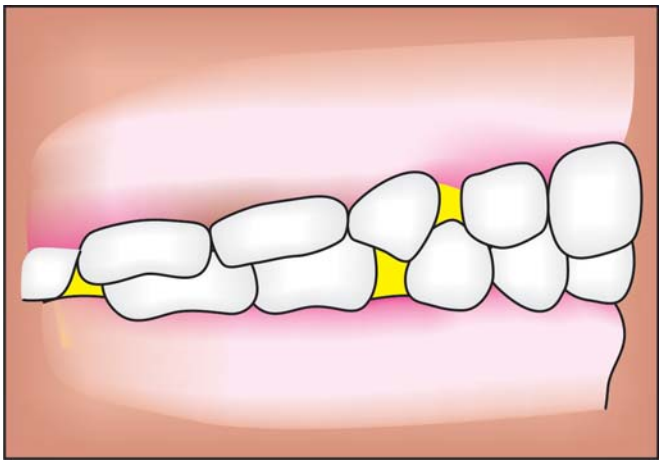

Fig. 8.3: Spaces in primary dentition

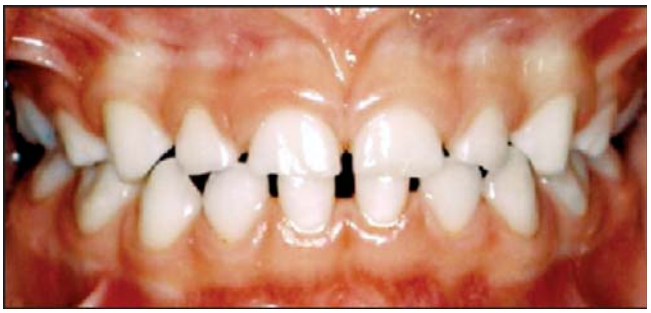

Fig. 8.4: Spaced dentition

The other spaces in primary dentition are called the developmental spaces which play an important role in the development of permanent dentition.

Some children do not have such physiological spaces thereby referred to as closed space or non-spaced dentition. Such dentition is highly prone to malocclusion during the development of permanent dentition.

Occlusal Relationship of Second Primary Molars

The primary dentition is complete after the eruption of the second primary molars. The dental arch circumference that connects the most distal surface of the right and left second primary molars should be preserved for the permanent dentition after the exchange of dentitions, and the space behind the primary molars is adequate for the permanent molar space including the first permanent molars.

The distal surface relation of maxillary and mandibular second primary molar is therefore one of the most important factor, that influences the future occlusion of permanent dentition.

The mesiodistal relation between the distal surface of the upper and lower second primary molars is called the terminal plane. When the primary teeth contact in Central occlusion, the terminal plane can be classified into three types:

Flush or vertical plane: The distal surfaces of upper and lower second primary molar are situated in a same vertical plane.

Mesial step: The distal surface of lower second primary molar is more mesial to that of upper.

Distal step: The distal surface of the lower second primary molar is more distal to that of the upper.

Though these relationships are functionally unimportant at this time; they greatly influence the position of the first permanent molars later. Because the eruption path of the first permanent molars is guided by the distal surface of the distal roots and crowns of second primary molars, the terminal plane decides the interocclusal relationship of the first permanent molars when the upper and lower molars first meet.

Size of the Dental Arch

The size of the primary dental arch is measured by the dental arch width between primary canines and between the second primary molars. The dental arch length is measured from the most labial surface of primary central incisors to the canines and to second primary molars.

Shape of Dental Arch

In deciduous dentition, the dental arch are wider and almost in wide 'U' shape with spaces between teeth visible.

Other Features

Other characteristic features of deciduous dentition are:

i. Deep bite
ii. Relatively flat curve of spee
iii. Shallow cuspal interdigitation, etc.

MIXED DENTITION PHASE

Mixed dentition period can be classified into following three periods for detailed study.

i. First transitional period

a. Eruption of first molar.
   1. Pathway of eruption of first molar
   2. Establishment of occlusion.
b. Exchange of incisors.
   1. Interdental spacing in primary incisor region
   2. Increase in intercanine width
   3. Increase of anterior length in dental arch
   4. Change of tooth axis of incisors.

ii. Intertransitional period.

iii. Second transitional period.

a. Exchange of canines and premolar.
   1. Leeway space
   2. "Ugly duckling" stage of Broadbent

b. Eruption of second permanent molar.

First Transitional Period

Eruption of First Molar (Fig. 8.5)

The first permanent molar is the key to the permanent occlusion of the teeth. It plays a vital role in the establishment and function of the occlusion of permanent dentition.

Pathway of eruption:

1. Maxillary first permanent molar tooth germ develops in the maxillary tuberosity region oriented with its occlusal surface downwards and backwards.
2. The tooth germ of the mandibular first permanent molar is located at mandibular gonion with its occlusal surface facing upwards and forwards.
3. Terminal plane of primary second molar is very important in determining the interocclusal relationship of the first permanent molars. As soon as the first permanent molars erupt into oral cavity, it comes in contact with the distal surface of second primary molar.
4. During this process, any unusual spaces created by the carious/traumatic destruction of the tooth crown and/or premature loss of the primary teeth will result in the mesial shift of the first molar in various ways due to presence of physiological spaces in primary dentition.

Establishment of occlusion of first permanent molars: The relationship between the types of terminal plane and the early occlusion of the first molars when they have just are as follows.

a. *Vertical plane type:* If dental spaces exited in primary dentition; the first molars will erupt into class I occlusion. It not, they will erupt into cusp-to-cusp occlusion.
b. *Mesial step type:* The first molars erupt directly into Angle's class I occlusion.
c. *Distal step type:* The first molars erupt directly and definitively into Angle's class II occlusion.

The establishment of the first molar's occlusion is dependent upon several factors including the second primary molar's occlusion; the spaces in the primary dentition and the growth of maxilla and mandible.

Exchange of Incisors

Before and after the eruption of the first permanent molars, the primary incisors begin to exchange with the

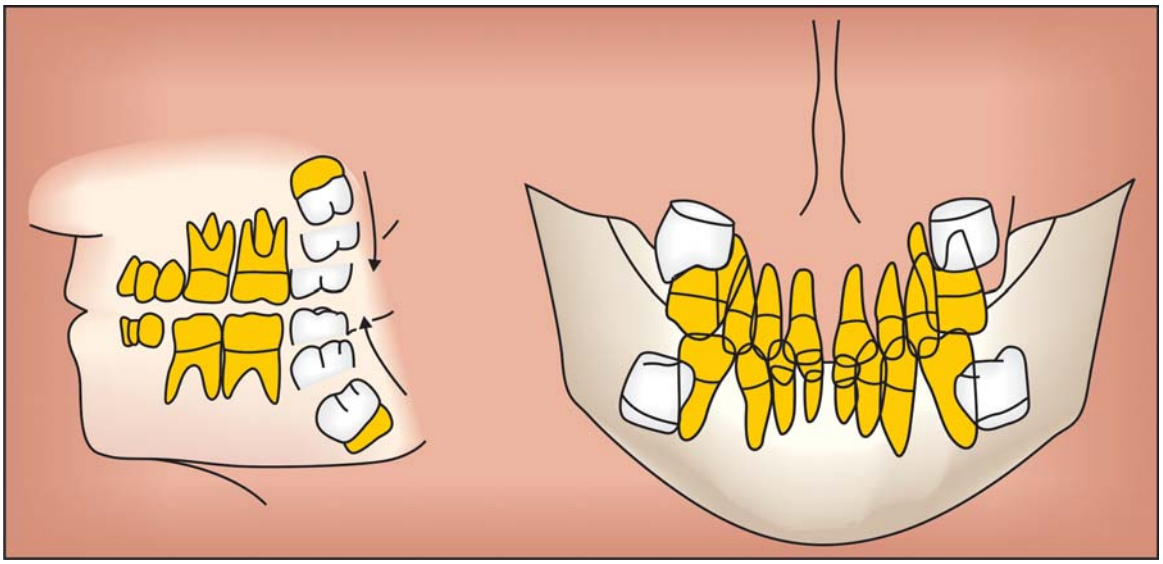

Fig. 8.5: Pathway of eruption of first permanent molar

permanent incisors, starting from lower central incisors.The total sum of mesiodistal width of four permanent incisors is larger than that of primary incisors by about 7 mm in maxilla, and by about 5 mm in the mandible which is termed as incisal liability.

The regulatory factors controlling the arrangement of the four permanent incisors is as follows:

i. Utilization of the physiologic spaces that exists in the primary dentition.
ii. Increase in the intercanine width.
iii. Increase of anterior length in the dental arch will provide space to accommodate the larger permanent incisors.

The permanent incisors erupt labially about 2-3 mm from the location of primary incisors.

iv. Both maxillary and mandibular permanent incisors show labial inclination much more than primary incisors making the permanent dental arch circumference wider for the arrangement of larger permanent incisor.

Intertransitional Period

It is a relatively lag phase with no active tooth movements to occur. The dental arches are comprised of both primary and permanent teeth.

Second Transitional Period

Exchange of the Lateral Teeth (Canines and Premolars)

The space available for the canine and premolars is limited as the mesial surface of the first permanent molar forms its distal limit and the distal surface of the permanent lateral incisor forms its mesial limit. To allow easy and noncomplicated exchange of lateral teeth following factors are necessary.

Leeway space (Fig. 8.6): The sum of the mesiodistal widths of the upper buccal deciduous teeth according to Black is 22.5 mm. The total for permanent teeth is 21.6 mm; leaving a difference of 0.9 mm that is available for mesial molar movement on each side. In lower arch the corresponding deciduous total is 22.6 mm; whereas the total mesiodistal width of permanent teeth is only 20.9 mm, leaving a difference of 1.7 mm. Generally Mesiodistal width of permanent teeth is smaller than that of the primary teeth by about 1.8 mm (both the sides) in maxilla and 3.4 mm (both the sides) in mandible. This extra space that is available in the final transition from primary to secondary dentitions is termed as **'Leeway space'**.

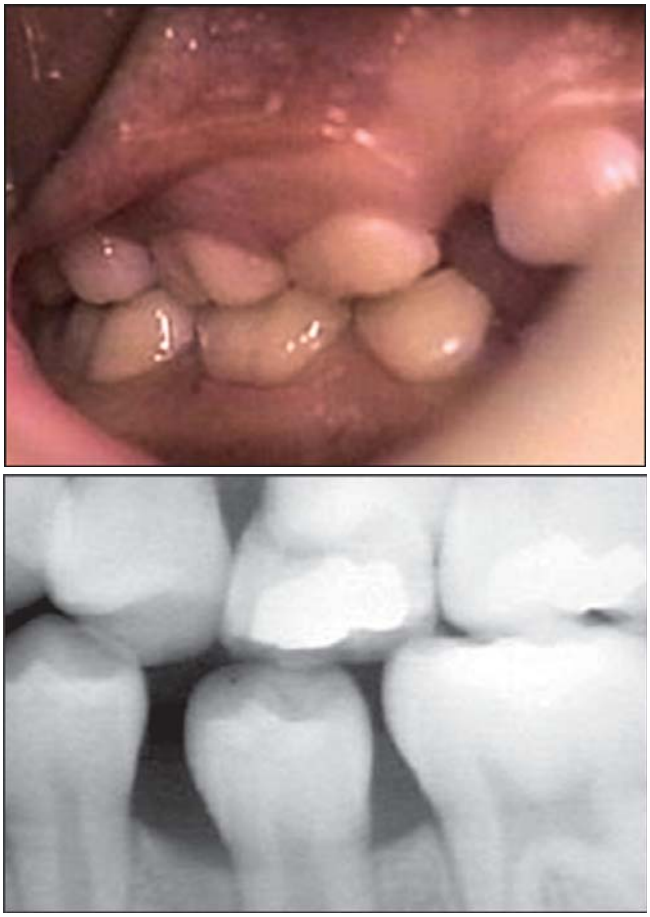

Fig. 8.6: Leeway space

When one observes the size of each lateral teeth, the permanent canine is larger than the primary canine, the first premolar is as large as the primary first molar and second premolar is smaller than second primary molar. Therfore, although an exchange of the lateral teeth may be carried out smoothly, there is crowding as each tooth is exchanged which is transient.

Ugly duckling stage of Broadbent (Fig. 8.7): The ugly duckling stage is also termed as broadbent phenomenon; named after the scientist who explained this stage.

It is a transient stage; referred to as a self correcting anomaly that occurs in children of 8-9 years age in the

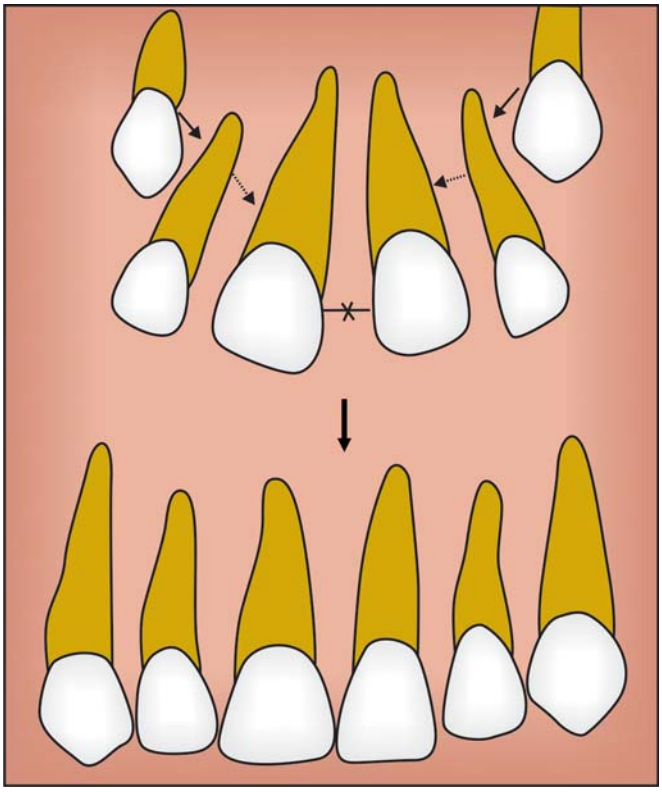

Fig. 8.7: Broadbent phenomenon—Ugly duckling stage

maxillary incisor region; during the eruption of permanent maxillary canines.

Initially there is midline diastema between the permanent incisors and spacing between the laterals; as the developing permanent canines erupt, they displace the roots of the laterals mesially resulting in transmitting of the force onto tooth of the central incisors which also show mesial displacement thereby closing the diastema between maxillary permanent incisors.

Eruption of the Second Permanent Molar

After the exchange of lateral teeth is completed and the dental arch upto the first molar is established, the second permanent molars begin to erupt. With the eruption of second permanent molar, the arch circumference may become shorter than that of the primary arch by utilization of the Leeway space.

LOCAL FACTORS AFFECTING THE EXCHANGE OF TEETH

The conditions required for smooth exchange of teeth can be subdivided into:

a. General factors.
b. Local factors.

Local factors are clinically related and are as follows:

Dental Caries of the Primary Teeth

1. Proximal carious lesions reduce the mesiodistal width of the crown.
2. Retained primary tooth roots.
3. Premature exfoliation/extraction of primary teeth.
4. Pathologic root resorption of primary teeth.
5. Presence of periapical lesions that may offend the eruption of permanent teeth.
6. Loss of vertical dimension, due to multiple, gross carious destruction.

Dental Abnormalities

1. Supernumerary teeth.
2. Dilacerations.
3. Odontomas, etc.

Permanent Tooth Related Factors

1. Impacted teeth.
2. Ectopic eruptions.
3. Congenital missing teeth.
4. Premature loss of permanent teeth, etc.

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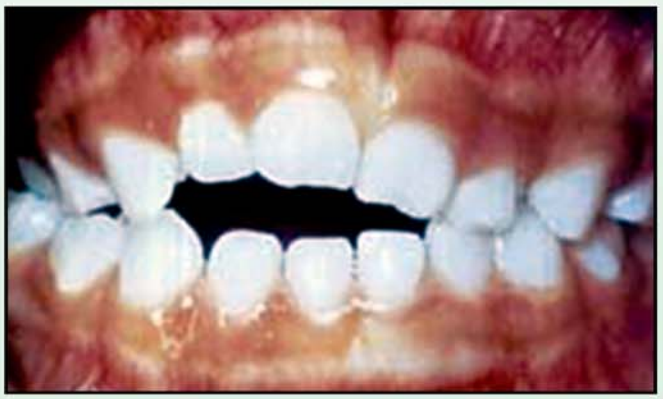

Pernicious Oral Habits in Children

DEFINITION OF HABIT

Habits are defined as "**Learned patterns of muscle contraction, which are complex in nature**" (Moyer's). Dorland defines habit as **"Fixed or constant practice established by frequent repetition"**. Maslow (1949) defines habit "**as a formed reaction that is resistant to change, whether useful or harmful, depending on the degree to which it interferes with the child's physical, emotional and social functions".**

Such oral habits in children are of great concern of worry to the parent. Treating oral habits in children is best managed by confluence of specialist care comprising of Dentist, Pedodontist, and Orthodontist and in certain cases even psychologist may be needed.

An infant's mouth is considered to be an erogenous zone through which the infant derives pleasure by stimulating it by means of suckling, light sucking, tongue movements, etc. These habits help in development of motor skills and other effects. Occasionally a retained infantile pattern can lead to what is known as **"Habit".**

CLASSIFICATION OF HABITS

Various authors have expressed their views regarding the classification of habits in different ways. They are as follows:

William James Classification (1923)

Useful Habits

This includes habits of normal function such as correct tongue position, proper respiration and deglutition and normal use of lips in speaking.

Harmful Habits

This includes all habits which exert perverted stress against teeth and dental arches as well as those habits such as open mouth habit, up biting, lip sucking, thumb sucking, etc.

Kingsley's Classification (1956)

Kingsley classified oral habits based on the criterion on the nature of the habits as:

i. Functional oral habit, e.g. mouth breathing
ii. Muscular habits, e.g. tongue thrusting, lip/cheek biting
iii. Combined muscular habits, e.g. thumb and finger sucking
iv. Postural habits.

Earnest Klien Classification (1971)

Klien classifies oral habits as:

Intentional/Meaningful Habits

These habits are those caused by an underlying psychological disturbance.

For example: Children of working mothers may develop habit of thumb sucking in childhood.

Unintentional/Empty Habits

A meaningless habit that has no underlying reason and such habits are best treated by reminder appliances.

Graber Classification

Graber classified all habits under extrinsic factors responsible for malocclusion as:

- Thumb/digit sucking
- Tongue thrusting
- Lip/nail biting
- Mouth breathing
- Abnormal swallow pattern
- Postural defects
- Speech defects
- Psychogenic habits – clenching, bruxism
- Defective occlusal habits.

Finn and Sim's Classification (1975)

Finn and Sim classified oral habits into two categories as:

- Compulsive habits
- Non-compulsive habits.

Compulsive Oral Habits

An oral habit is said to be compulsive when it has acquired a fixation in the child to such an extent that

the child retreats to the practice of the same at time of insecurity, stress, threat or fear. They have deep seated emotional need and attempts to correct such habits may cause increased anxiety in the child. Such habits have multifactorial causes as:

- Lack of parental love/attention
- Too little/improper feeding
- Bottle feeding.

Non-compulsive Oral Habits

Non-compulsive oral habits are those that care forgotten/dropped by the child as age and maturity develops. It is easy to treat such habits and requires continuous behavior modification to drop undesired habits.

THUMB/FINGER SUCKING

Gellin (1978) defines thumb/finger sucking as placement of thumb or one or more fingers in varying depths into the mouth.

Sucking Reflex

Thumb sucking reflex is observed even in the intrauterine life. At birth, it is established as a neuromuscular reflex pattern. The habit of sucking occurs during normal growth between 1-3½ years of age and gradually disappears; but in certain individuals due to any psychological; emotional or physiological disturbance it leads to fixation of the habit.

Theories of Thumb Sucking

Oral Theory of Sears and Wise (1950)

It suggests that strength of oral drive is in part a function of how long a child continues to feed by sucking. Thus it is not frustration of weaning that produces thumb sucking, but oral drive, which has been strengthened by the prolongation of nursing.

Benjamin's Theory

This theory states that thumb sucking is an expression of need to such which arises because of association of sucking with the primary reinforcing aspects of feeding.

Psychosexual Theory Sigmund Freud

Thumb sucking arises from an inherent psychosexual drive. It is a pleasurable structuralization of lips and mouth. Morality in the infant is related to pre-genital organization thus, the object of thumb sucking is nursing.

Oral Gratification Theory – Sheldon (1932)

If a child is not satisfied with sucking during the feeding period, it will persist as a symptom of an emotional disturbance by digit sucking.

Classification of Thumb Sucking

Normal Thumb Sucking

The thumb sucking habit until the age 3½ years is considered to be normal which usually disappears as the child matures. This does not have any deleterious effect causing malocclusion.

Abnormal Thumb Sucking

When thumb sucking habit persists beyond the prescribed age, it is then thought to be abnormal. If not intercepted at right time, it may have deleterious effect on dentofacial complex.

It is further classified into:

- Psychological
- Habitual

Other Classification

Sucking can also be classified as:

- Nutritive sucking habits (e.g. Breast/bottle feeding)
- Non-nutritive sucking habits.

Subtelny (1973) has graded thumb sucking into 4 types:

Type A: Such type is seen in almost 50% of children, where the whole digit is placed inside the mouth with the pad of the thumb pressing over the palate while at same time maxillary and mandibular anteriors contact is present.

Type B: It is seen in 13-24% of cases in which thumb is placed in mouth without touching the palatal vault

while the contact is maintained between maxillary and mandibular anterior.

Type C: It is seen in 18% of case where thumb is placed in mouth just beyond the first joint and contacts the hard palate and only maxillary incisors; but there is no contact with the mandibular incisors.

Type D: It is seen in 6% of cases where only a little portion of thumb is placed into the mouth.

Johnson (1993) Classified

Non-nutritive sucking habits based on the factors that influence the severity of the habit (Table 9.1).

Causative factors for thumb sucking:

- Parental factors
- Sibling factors
- Social factors
- Nutritive/feeding practices, etc.

Table 9.1: Classification of non-nutritive sucking habits (Johnson, 1993)

Level	*Description*
Level I(+/–)	Boys/girls of any chronological age with a habit that occurs during sleep
Level II(+/–)	Boys below age 8 with a habit that occurs at one setting during waking hours
Level III(+/–)	Boys under age 8 years with a habit that occurs at multiple settings during waking hours
Level IV(+/–)	Girls below age 8 or a boy over 8 years with a habit that occurs at one setting during waking hours
Level V(+/–)	Girls under age 8 years or a boy over age of 8 years with a habit that occurs across multiple settings during waking hours
Level VI(+/–)	Girls over age 8 years with a habit that occurs during waking hours

Clinical Features of Thumb Sucking (Fig. 9.1)

- Anterior open bite
- Proclination of maxillary anteriors
- Retroclination of mandibular anteriors
- Narrowing/constriction of maxillary arch
- Narrow nasal floor and high palatal vault
- Posterior cross bite
- Increased unilateral and bilateral
- Increased lip incompetence

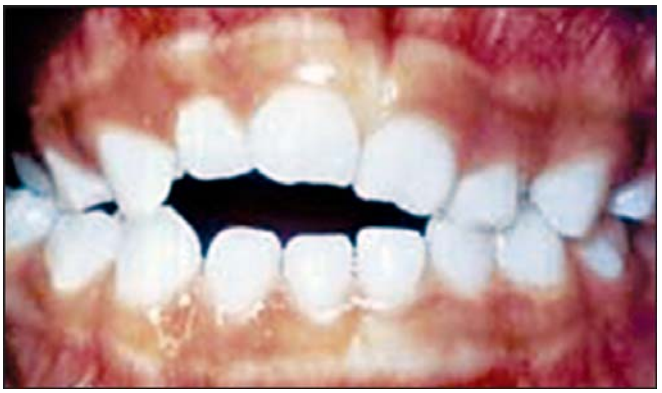

Fig. 9.1: Pretreatment photograph of patient with thumb sucking habit

- Hypotonicity of upper lip
- Hyperactive lower lip
- Compensatory tongue thrusting, etc.

The type of malocclusion produced by digit sucking is dependent on a number of variables such as:

- Position of the digit
- Associated orofacial muscle contraction
- Mandibular position during sucking
- Facial skeletal pattern
- Intensity, frequency and duration of force applied.

Management of Thumb Sucking Habit

Discussion with Child

Establish a friendly rapport with the child and learn the child's attitude towards the habit and his/her ability to perceive things. Be supportive and let the child know that you want to help him. Explain the ill-effects of the habits and the methods to help him prevent it.

Discussion with Parents

Explain to the parents not to ridicule the child by discussing about the habit at home and reinforce them that this child would loose the habit by end of this phase.

Measures of Correcting Thumb Sucking Habit

1. Apply non-palatable substance to thumb/finger.
2. Apply a bandage to thumb or finger.
3. "Do it yourself kit," as advocated by Whitman.

   Apply a reminder lotion on the thumb/finger and a magic pill to be taken at bed time. The child is

told the pill goes into the stomach, up into your shoulder, down the arm into your thumb, and then automatically pops out of your mouth.

4. *Dunlop's beta hypothesis:* Forced purposeful repetition of a habit eventually associates it with unpleasant reactions and the habit is abandoned. The child should be asked to sit in front of a mirror and asked to such his/her thumb, observing himself/herself as he/she indulges in the habit.

Habit Breaking Appliances

Requirements of an ideal appliance:

1. Should offer no restraint whatsoever to normal muscular activity
2. Should not require remembering to be used
3. Should have no shame attached to its use
4. Should not involve the parents.

Removable appliances:

- Reminder appliance – Simple Acrylic plate
- Appliance with tongue spikes
- Appliance with tongue guard
- Appliances with rakes, palatal crib, palatal arch, lingual spurs.

Mechanotherapy (Figs 9.2 to 9.4)

Fixed intraoral anti-thumb sucking appliance:

- Intraoral appliance attached to maxillary teeth by means of bands fitted to primary second/permanent first molars.
- A lingual arch forms the base of appliance to which interlacing wires in the anterior area of hard palate is added.
- This appliance prevents the patient from putting the palmar thumb surface in contact with palatal gingival, depriving the pleasure of sucking.

Quad helix: It prevents insertion of thumb and also corrects the malocclusion by expanding the arch.

Fig. 9.2: Thumb sucking appliance

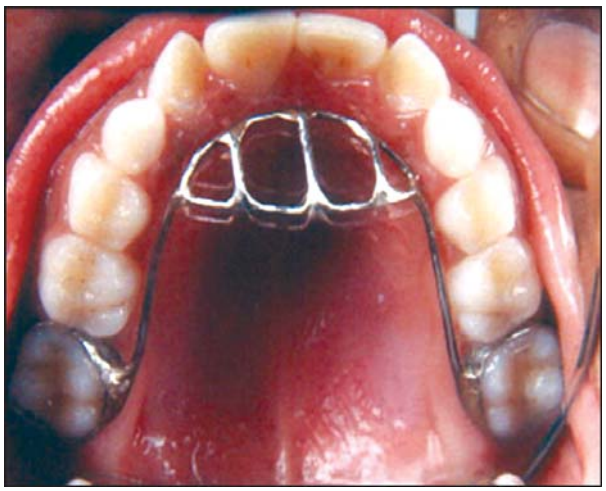

Fig. 9.3: Thumb sucking appliance in position intraoral photograph

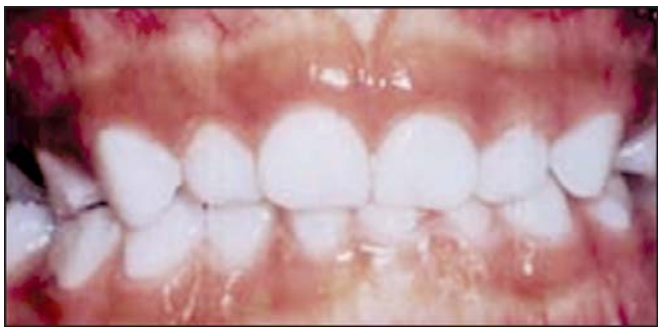

Fig. 9.4: Post-treatment photograph after appliance therapy

TONGUE THRUSTING

Brauer (1965) states "A tongue thrust was said to be present if the tongue was observed thrusting between, and the teeth did not close in centric occlusion during deglutition".

Barber (1975) defines "Tongue thrust as an oral habit pattern related to the persistence of an infantile swallow pattern during childhood and adolescent and thereby produces an open bite and protrusion of the anterior tooth segments."

Etiology

- Genetic factors
- Retained infantile swallow
- Upper respiratory tract infections
- Neurological disturbances
- Feeding practices
- Compensatory induction by oral habits
- Tongue size.

Classification of Tongue Thrust

Moyer's Classification

a. Normal infantile swallow
b. Normal mature swallow
c. Simple tongue thrust swallow
d. Complex tongue thrust swallow
e. Retained infantile swallow.

Braner and Holt Classification

Type I	Non-deformity tongue thrust
Type II	Deformity tongue thrust: Subgroup I – Anterior open bite Subgroup II – Associated with procumbency of incisors. Subgroup III – Associated with posterior cross bite
Type III	Deformity lateral tongue thrust: Subgroup I – Posterior open bite Subgroup II – Posterior cross bite Subgroup III – Deep over bite
Type IV	Deformity anterior and lateral tongue thrust Subgroup I – Anterior and posterior open bite Subgroup II – Associated procumbency of anterior teeth Subgroup III – Associated posterior cross bite

Simple Tongue Thrust (Fig. 9.5)

It is defined as tongue thrust with teeth together swallow. Open bite in the anterior region is the characteristic feature. It is associated with abnormal functioning of the lips, mentalis and other circumoral muscles. As the patient swallows, anterior lip seat is made partly with the teeth and partly with the lips. The inferior orbicularis cannot elevate itself, it must be elevated with the help of mentalis.

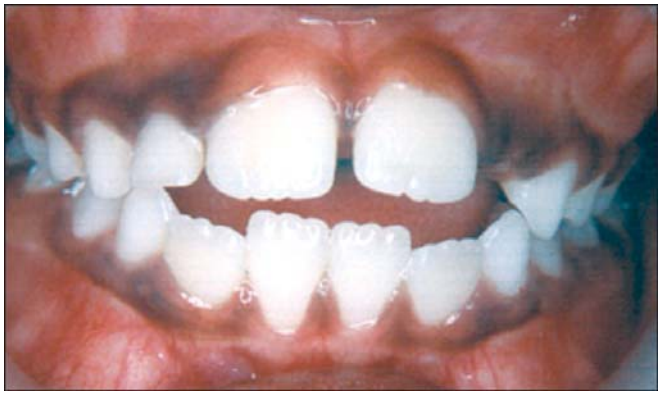

Fig. 9.5: Simple tongue thrust

Complex Tongue Thrust

This is defined as tongue thrust with teeth apart swallow. The malocclusion associated with it has following characteristics:

- Generalized anterior open bite
- Absence of contraction of lip and musculature during swallowing
- Abnormal occlusal reflex
- Abnormal swallow reflex.

Retained Infantile Swallow

It is persistence of the infantile swallow pattern even after eruption of permanent teeth where the tongue does not drop back as it should and continues to thrust forward and maintains the forward position even at rest.

Patients exhibit expressionless face since facial muscles are used to stabilize of seventh cranial nerve musculature during swallowing.

Treatment

Training of Correct Swallow and Tongue Posture

Myofunctional exercises:

- Ask the child the place the tongue tip in the rugae area for 5 minutes and then asked to swallow.

- Ask the patient to hold orthodontic elastic band at the tip of the tongue against the palate on the rugae area and practice.
- *4s exercise:* Use the pressure point on papilla to show where the 'Spot' is. The tongue tip is against this spot at rest position. The child is then asked to 'squeeze' the tongue again the spot with the teeth closed, followed by relaxing and swallowing.

Appliance Therapy

- Use of Nance palatal arch appliance to guide the posture and positioning of the tongue.
- Habit breaking appliance with tongue cribs.
- Oral screen.

Other Measures

- Speech therapy
- Correction of malocclusion
- Surgical treatment.

MOUTH BREATHING

For normal dentofacial growth to occur, there should be normal breathing. Increased resistance to the flow of air through the nasal passages may be considered to be the primary cause of mouth breathing. Sassouni (1971) defines mouth breathing as **habitual respiration through the mouth instead of the nose.**

Classification

Finn (1987) has classified mouth breathing into:

Anatomic Type

The anatomic mouth breather is one whose upper lip is short and thereby does not permit complete closure of without undue effort.

Obstructive Type

Children who have increased resistance to/or complete obstruction of the normal flow of air through the nasal passages.

Habitual Type

Those who continue to mouth breathe even after removal of abnormal obstruction, are referred to as habitual mouth breathers.

Etiology

The most common cause being some degree of nasal insufficiency in children. Allergies, physical obstructions, chronic nasal infections, deviated nasal septum, nasal polyps, enlarged adenoids or tonsils abnormally short upper lips, obstructive sleep apnea syndromes, etc. can cause mouth breathing.

Clinical Features (Fig. 9.6)

Facial Form

- Have increased vertical growth pattern
- Patients have increased mandibular plane angle
- Retrognathic maxilla and mandible
- Mouth breathers show a characteristic facial configuration, commonly referred to as '**adenoid facies**' characterized by:
  - Long narrow face
  - Narrow nose and nasal passages
  - Flaccid lips with short upper lip
  - Doligofacial skeletal patterns
  - 'V' shaped and high palatal vault.

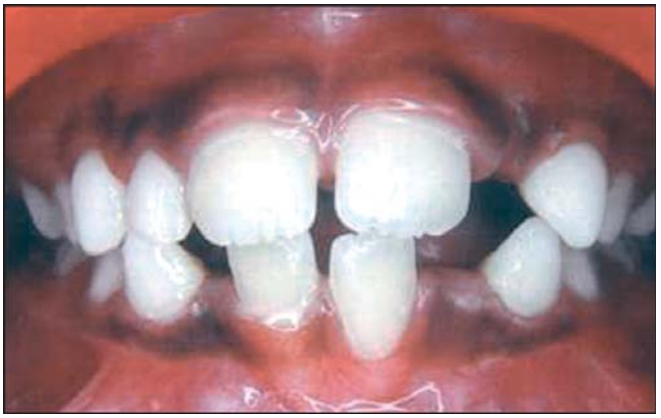

Fig. 9.6: Intraoral photograph of a boy with mouth breathing habit

Dental Effects

- Retroclination of upper and lower incisors
- Posterior cross bite
- Tendency towards open bite
- Constricted maxillary arch
- Exposed upper incisors due to short upper lip.

Speech Defects

Nasal twang in the voice is present.

Lip (Fig. 9.7)

- Lip incompetence
- Short upper lip causes excessive exposure of gums causing 'gummy smile'.
- Curling of lower lip.

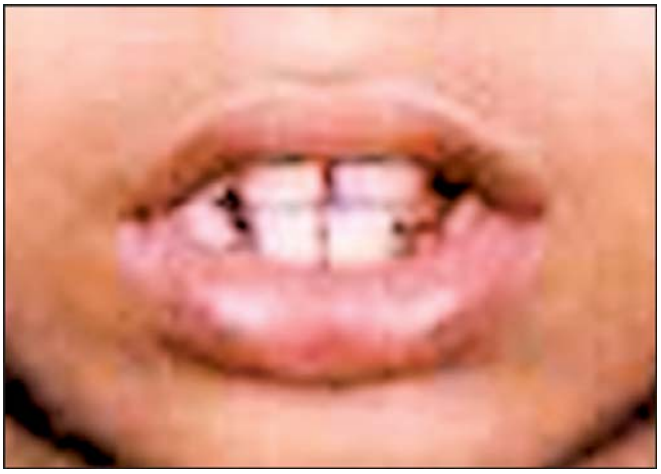

Fig. 9.7: Lip incompetence in a child with mouth breathing habit

Gingiva

- Inflamed and irritated in the relation to anteriors.
- Hyperplastic nature of gingiva due to mouth breathing

Clinical Tests for Diagnosis

1. Mirror test
2. Butterfly test
3. Water holding test
4. Inductive plethysmography
5. Cephalometrics.

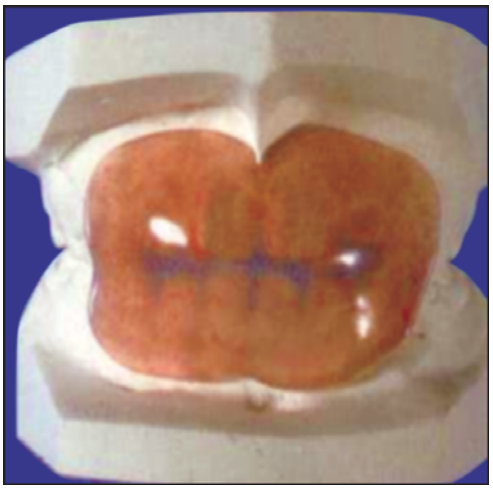

Fig. 9.8: Oral screen

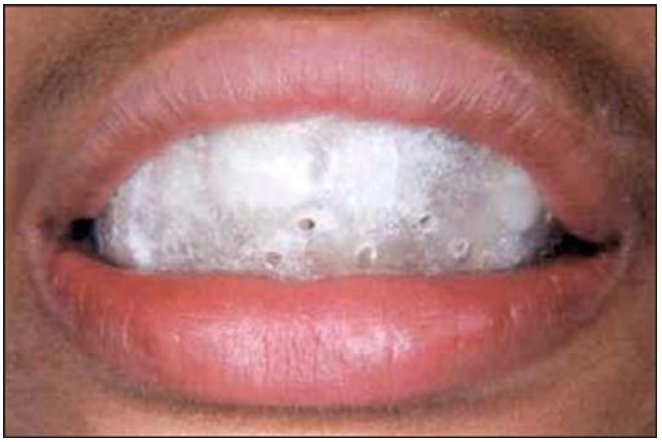

Fig. 9.9: Oral screen intraoral

Treatment Considerations

1. Elimination of underlying pathology.
2. Correction of mouth breathing by appliance therapy like oral screen (Fig. 9.8).
3. Interception of the habit through physical exercises like deep breathing exercise; lip exercises; maxillo-thorax myotherapy.
4. Correction of resultant malocclusion (Fig. 9.9).

BRUXISM

Ramfjord (1966) defines bruxism 'As the habitual grinding of teeth when the individual is not chewing or swallowing'.

Vanderas (1995) defines bruxism as 'Non-functional movement of the mandible with or without an audible sound occurring during day or night.

Types

1. Day time/diurnal bruxism
2. Night time/Nocturnal bruxism

Nocturnal bruxism is the most common in occurrence that can occur during any stage of sleep and is reported to be the most damaging.

Etiology

1. CNS
2. Psychological factors and stress
3. Occlusal discrepancies
4. Genetics
5. Systematic factors like magnesium deficiency, gastrointestinal disturbances.
6. Occupational factors.

Clinical Manifestation

Depends upon the frequency, intensity, age of the patient and duration.

The symptoms of bruxism are:

1. Occlusal trauma
   - Tooth mobility
   - Periodontitis.
2. Tooth wear
   - Attrition
   - Abfraction defects
   - Sensitivity
   - Fractures of tooth.
3. Muscular tenderness of masticatory muscles.
4. Temporomandibular joint disorders.
5. Headache.
6. Accidental injury to soft tissue causes buccal mucosal/lip ulcerations.

Treatment

1. Occlusal adjustment.
2. Occlusal splints.
3. Restorative treatment of affected teeth.
4. Psychotherapy.
5. Relaxation techniques.
6. Oral physiotherapy.
7. Pharmacotherapy – muscle relaxants, anti anxiety drugs.
8. Biofeedback.
9. Alternative medicines:
   - Acupuncture
   - Yoga
   - Meditation.
10. Orthodontic correction.

LIP BITING

Normal lip anatomy and function are important for speaking, eating and maintaining balanced occlusion. A lip sucking habit is a compensatory activity that results from excessive overjet and relative difficulty of closing the lips properly during deglutition.

Habits that involve manipulation of the lips and perioral structures are termed as lip habit.

Etiology

- Malocclusion – Class II Division. I, deep bite malocclusion

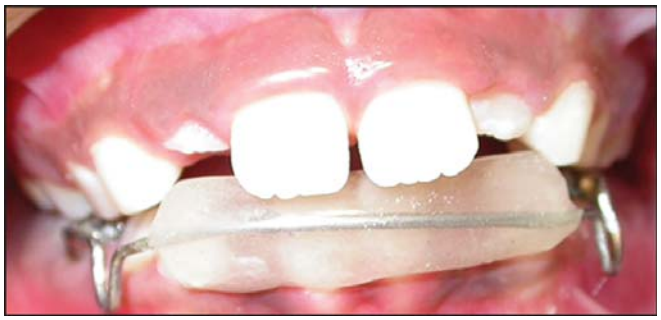

Fig. 9.10: Lip bumper in position

- Habits – Thumb/digit sucking
- Emotional stress.

Clinical Manifestations

- Protrusion of maxillary incisors
- Retrusion of mandibular incisors
- Accentuated mentolabial sulcus
- Resultant/existent malocclusion
- Reddened irritated and chapped vermilion border of the lips
- Increased probability of infection of traumatized up due to constant lip biting
- Scarring on the lips.

Treatment

- Correction of malocclusion
- Treatment of the primary habit
- Appliance therapy by use of appliances like oral shield
- Lip bumper (Fig. 9.10).

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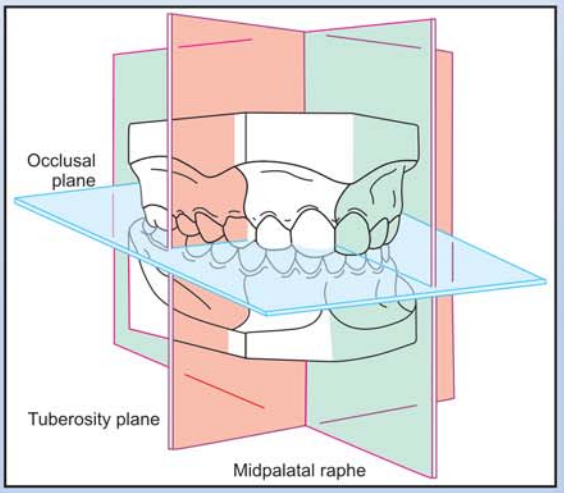


Preventive and Interceptive Orthodontics

PREVENTIVE ORTHODONTICS

Graber (1996) defines preventive orthodontics **as the action taken to preserve the integrity of what appears to be normal at a specific time** which includes:

- Early correction of carious lesions, particularly proximal caries.
- Early recognition and elimination of oral habits.
- Using space maintainers in case of early loss of deciduous teeth.

There are various procedures involved in preventive orthodontics. They are as follows:

- Pre-dental procedures.
- Care of deciduous dentition.
- Patient and parent education.
- Management of dental anomalies such as
  - Supernumerary teeth.
  - Congenital missing teeth.
  - Ankylosis
  - Ectopic eruption.
  - Early loss of deciduous teeth.
- Early management of carious lesions.
  - Especially proximal lesions.
- Occlusal equilibration.
- Management of Frenal attachments
  - Tongue tie
  - High attached labial frenum.
- Prevention of oral habits
- Space maintenance.

Predental Procedures

Predental procedures include educating the mothers about nursing care of the child; oral hygiene measures for the infant; nutritional status of mother and child. Appropriate nursing and care by mothers reduces the risk of fixation to habits and provides sufficient nutrition to aid in growth of the infant.

Proper oral hygiene measures at this early stage, prepares the mothers for their extra care in future during developing dentition and primarily maintenance of oral health.

In appropriate bottle feeding; use of pacifiers; can lead to early childhood caries; hence bottling and feeding/weaning techniques are to be taught to the mothers.

Care for Deciduous Dentition

Motivate parents to take their children for regular dental check ups. Dietary restriction of cariogenic agents, regular maintenance of oral hygiene which are essential for care of deciduous dentition.

Patient and Parent Education Program

Parent and patient education is an ultimate need to motivate them to know the value of oral health in maintenance of general health. Topics for parent and patient education may be various dental, oral/general health related topics using audiovisual aids and counseling. Some of the topics include:

- Need for the care of deciduous dentition
- Oral hygiene
- Correct brushing techniques
- Preventive services available, etc.

These programmes should be helpful in dispelling myths among the groups and educate, motivate them to march towards '**Dentally Healthy State**'.

Management of Dental Anomalies

Supernumerary Teeth

Though the presence of supernumerary teeth may/may not cause malocclusion in deciduous dentition; but it is generally a risk during mixed dentition period which may interface with the eruption of permanent teeth and at times may cause ectopic eruptions too. Early recognition of presence of such teeth and their timely extract prevents development of malocclusion.

Congenital Missing Teeth

They may cause spaced dentition, which causes drifting of adjacent teeth into the space leading to malocclusion. Early recognition of such conditions helps to prevent malocclusion by appropriate space maintenance.

Ankylosed Deciduous Teeth

Such teeth will interfere with the eruption of their permanent successor. Such teeth should be surgically removed at appropriate time to allow unhindered eruption of permanent teeth into normal position.

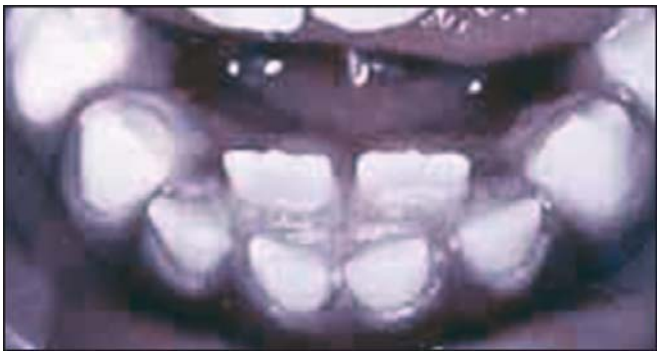


Fig. 10.1: Ectopic eruption

Ectopic Eruption (Fig. 10.1)

Ectopic eruption of permanent teeth, is generally due to interferences in the pathway of eruption of developing teeth causes for ectopic eruption include:

- Periapical pathologies
- Supernumerary teeth
- Trauma.

Ectopic eruption can be prevented timely elimination of such interferences.

Early Loss of Deciduous Teeth

Early loss of deciduous tooth is attributed due to causes like:

- Caries
- Unplanned extractions
- Trauma
- Resorption
- Syndromes like Papillon Lefevre syndrome.

Such early loss of deciduous teeth may cause collapse of the dental arch, drifting of teeth into spaces; malocclusion; psychological problems in young children, etc.

To prevent such features, following care should be taken:

1. Prevent carious lesions by controlling diet factor, fluorides, sealants, oral hygiene procedures.
2. Dentist should have the knowledge about life expectancy of each primary tooth, and plan extractions accordingly.

If extraction of the tooth is inevitable and it has longer life expectancy, then the space of the lost tooth should be maintained by suitable space maintainer until the eruption of permanent successor into the space.

Prolonged Retention of Deciduous Teeth

Prolonged retention of deciduous teeth due to failure of resorption of roots; ankylosis will interfere with the eruption of its permanent successor and may also cause its ectopic eruption resulting in crowding and malocclusion.

Such teeth should be identified by clinical and radiological investigations and extracted in appropriate time to facilitate uninterfered eruption of the developing permanent teeth.

Early Management of Carious Lesions

This requires frequent dental visits by a child, so that caries is arrested at the earliest stage by restorative/regenerative procedures. However large carious lesions especially proximal destruction of tooth structure also contributes to some amount of space less in the arch and this best managed by restoring the proximal caries/gross carious teeth by stainless steel crowns, restorative material, etc.

Occlusal Equilibration

Presence of Cuspal interferences can cause deviated path of closure of mandible leading to attrition of occlusal facts and other TMJ problems.

They prevent deviated path of closure, the occlusal prematurities should be carefully identified and corrected by selective grinding of cusps.

Abnormal Frenal Attachments (Figs 10.2 and 10.3)

Abnormal frenal attachments, as in lingual frenum and labial frenum cause partial/complete ankyloglossia and midline diastema and other malocclusion respectively. It can be prevented by procedures like frenectomy at appropriate time.

Oral Habits

Oral habits when identified earlier, they are treated more easily than in cases of established dependence to habits and ultimately preventing development of severe malocclusion.

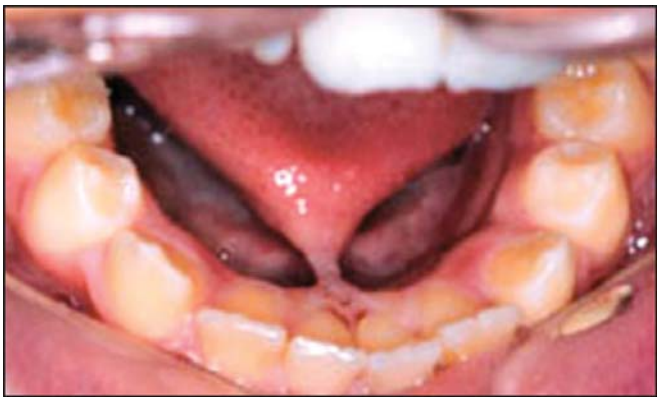


Fig. 10.2: Ankyloglossia

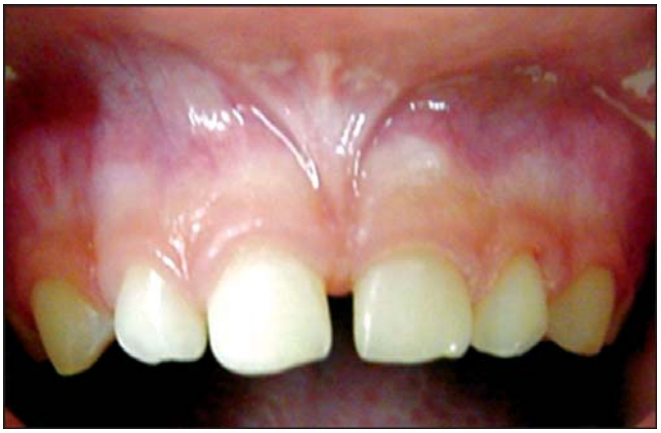


Fig. 10.3: High frenal attachment

Space Maintenance

Space maintenance is defined as "the measures or procedures that are brought into use due to premature loss of deciduous teeth/tooth, to prevent loss of arch development".

Space maintainers are defined as "the appliances that prevent loss of arch length and which in turn guide the permanent tooth into a correct position, in the dental arch.

Factors to be considered for space maintenance:

1. **Time elapsed since loss of tooth:** Maximum space loss occurs within 2 weeks to 6 months of the premature loss of the deciduous tooth. Therefore, it is highly advisable to fabricate the space maintainer before extraction and to be inserted at the time of extraction.
2. **Dental age of the patient:** Dental age is a more reliable parameter that the chronological age. The dentist should know the dental age of every patient to anticipate the exfoliation/eruption of teeth and their timings.

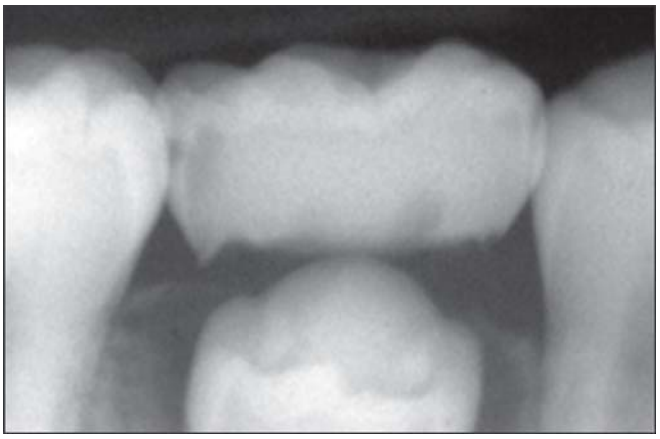


Fig. 10.4: Radiograph showing amount of bone covering the developing permanent tooth bud

3. **Amount of bone coverage over developing succedaneous tooth (Fig. 10.4):** This gives the time period for the eruption of tooth into oral cavity and thereby needs for space maintenance and type can be decided.
4. **Stage of root formation:** Developing tooth buds begins to erupt actively if the root is three-fourth formed.
5. **Sequence of teeth eruption.**
6. **Congenitally missing teeth.**
7. **Eruption of the permanent teeth in the opposite dental arch.**

Ideal Requisites of Space Maintainer

- Should maintain desired distal dimensions of the space.
- Should not interfere with the eruption of permanent teeth.
- Maintenance of physiological functional movement of teeth.
- Should allow for space regainence when required.

Classification of Space Maintainer (Fig. 10.5)

I. **Raymond C. Throw Classification**
   a. Removable.

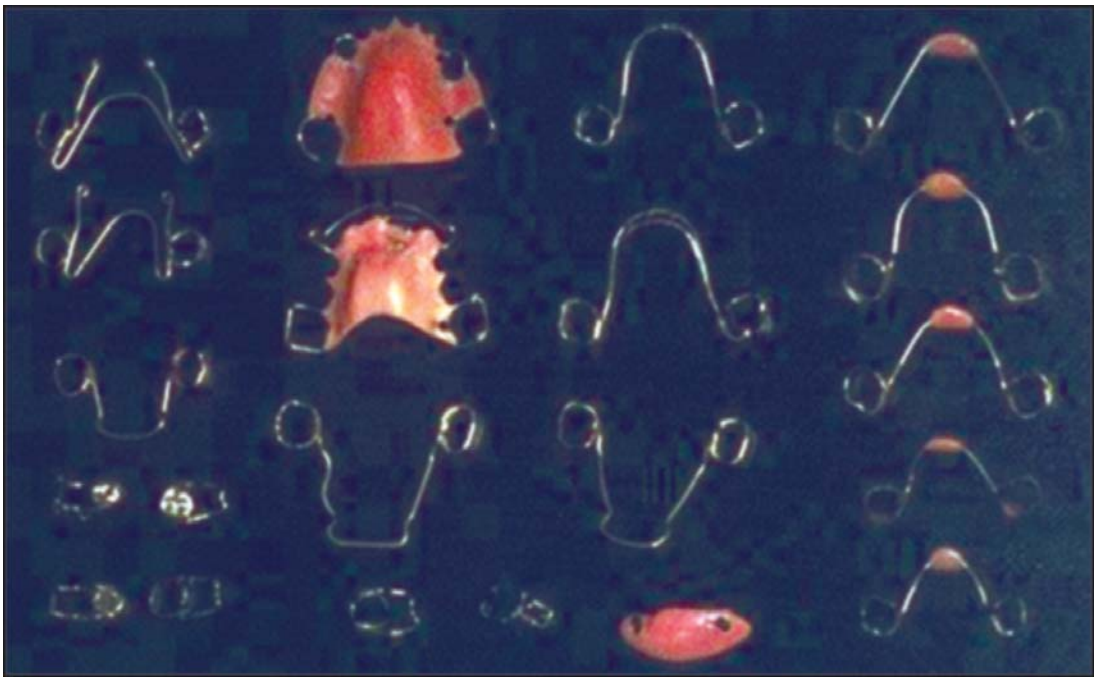


Fig. 10.5: Types of space maintainers

b. Complete arch
   - Lingual arch.
   - Extra Oral anchorage

c. Individual tooth space maintainer

II. According to Heinrichsen

a. Fixed space maintainers

Class I: I. Non–functional:
- i. Bar type – no longer in use
- ii. Loop type.

II. Functional type:
- i. Pontic Type
- ii. Lingual arch type.

Class II: Cantilever type:
- Distal shoe
- Band and loop.

b. Removable space maintainers

III. According to Hitchcock:

a. Removable, fixed or semi-fixed.
b. With or without bands.
c. Functional or non-functional.
d. Active or Passive.
e. Certain combinations of the above.

Table 10.1 provides information on space maintenance in different clinical situations.

Space Maintenance for Premature Loss of Deciduous First Molar

Sequence:

1. If primary first molar is lost during active eruption of permanent first molar- the primary second molar may tilt mesially leading to decreased space for eruption of first premolar.
2. If primary 1st molar is lost during eruption of lateral incisor, it leads to distal ditching of deciduous canine and it may lead to midline shift in the affected side.
3. It may cause blocking out of permanent canine.

In case of unilateral loss:

1. Band and loop space maintainer with deciduous second molar is the abutment.
   Modifications:
   Crown and loop space maintainer.
   Band and loop space maintainer with occlusal stop.

Table 10.1: Different situations of space maintenance

Primary dentition			
Maxilla		***Mandible***	
Missing tooth	***Treatment***	***Missing tooth***	***Treatment***
Unilateral loss of primary 1st molar	Band/crown and loop	Unilateral loss of primary 1st molar	Band/crown and loop
Unilateral loss of primary 2nd molar	No treatment until eruption of 1st permanent molar, then distal crown and loop until both 1st permanent molars are completely erupted and a transpalatal arch can be placed	Unilateral loss of primary 2nd molar	Distal shoe until eruption of 1st permanent molars and permanent incisors, then lower lingual holding arch
Bilateral loss of primary 1st molars	Bilateral bands/crowns and loops	Bilateral loss of primary 1st molars	Bilateral bands/crowns and loops
Bilateral loss of primary 2nd molars	No treatment until eruption of 1st permanent molar, then distal crown and loop until both 1st permanent molars are completely erupted and a transpalatal arch can be placed	Bilateral loss of primary 2nd molars	Distal shoe until eruption of 1st permanent molars and permanent incisors, then lower lingual holding arch
Multiple bilateral primary molar loss	Saddle appliance until 1st permanent molars are completely erupted and a Nance can be placed	Multiple bilateral primary molar loss	Saddle appliance until 1st permanent molars and permanent incisors are erupted and a lower lingual holding arch can be placed
Early mixed dentition (permanent 1st molars erupted, but all permanent incisors are not erupted)			
Unilateral loss of primary 1st molar	No treatment unless leeway space is to be preserved	Unilateral loss of primary 1st molar	No treatment unless leeway space is to be preserved
Unilateral loss of primary 2nd molar	Transpalatal	Unilateral loss of primary 2nd molar	Band and loop until eruption of permanent incisors, then lower lingual holding arch
Bilateral loss of primary 1st molars	No treatment unless leeway space is to be preserved	Bilateral loss of primary 1st molars	No treatment unless leeway space is to be preserved
Bilateral loss of primary 2nd molars	Nance	Bilateral loss of primary 2nd molars	Bilateral band and loop until eruption of permanent incisors, then lower lingual holding arch
Multiple bilateral primary molar loss	Nance	Multiple bilateral primary molar loss	Saddle appliance until eruption of permanent incisors, then lower lingual holding arch

Contd...

Table 10.1: Contd...

Late mixed dentition (permanent 1st molars and permanent incisors erupted)			
Maxilla		***Mandible***	
Missing tooth	***Treatment***	***Missing tooth***	***Treatment***
Unilateral loss of primary 1st molar	No treatment unless leeway space is to be preserved	Unilateral loss of primary 1st molar	No treatment unless leeway space is to be preserved
Unilateral loss of primary 2nd molar	Transpalatal	Unilateral loss of primary 2nd molar	Lower lingual holding arch
Bilateral loss of primary 1st molars	No treatment unless leeway space is to be preserved	Bilateral loss of primary 1st molars	No treatment unless leeway space is to be preserved
Bilateral loss of primary 2nd molars	Nance	Bilateral loss of primary 2nd molars	Lower lingual holding arch
Multiple bilateral primary molar loss	Nance	Multiple bilateral primary molar loss	Lower lingual holding arch

In case of bilateral loss: Permanent first molars are more preferred than deciduous second molars for abutments.

A. For Maxilla
   1. Nance palatal holding arch.
   2. Transpalatal arch.
   3. Bilaterally placed band and loop space maintainer.
B. For Mandible
   1. Lingual arch.
   2. Bilaterally placed band and loop space maintainers.

Space Maintenance for Premature Loss of Deciduous Second Molars

Sequence:

1. Causes mesial topping of permanent first molar; which in turn blocks out the space for second premolar.
2. Irregularity of molar relationships.

In case of unilateral loss:

1. Band and loop space maintainer.

In case of bilateral loss:

A. For Maxilla
   1. Nance palatal holding arch.
   2. Bilateral band loop.
B. For mandible
   1. Lingual arch

Space Maintenance of Premature Loss of Deciduous Second Molar Prior to Eruption of First Molar

It may lead to mesial tilting or migration of permanent first molar into the space of deciduous second molar.

Space maintainer of choice: Distal shoe space maintainer

Space Maintenance for Premature Loss of Deciduous Incisors

Removable partial dentures

Space Maintenance for Premature Loss of Deciduous Canines

In case of unilateral loss: Band and loop.

In case of bilateral loss

A. For Maxilla
   - Nance Palatal holding arch.
B. For Mandible
   - Lingual arch.

Nance Palatal Holding Arch (Fig. 10.6)

Both the first permanent molars are banded and impression is made with band in position and poured using dental store. Palatal arch is fabricated along the palatal surface and an acrylic button is placed along the

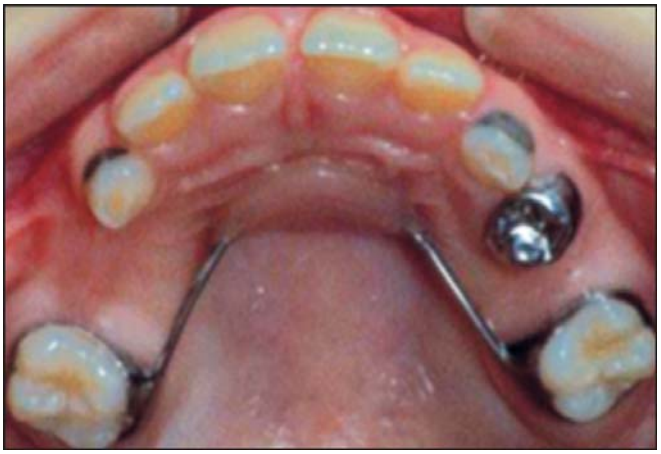


Fig. 10.6: Nance palatal arch

palatal arch wise and soldered to molar bands on either sides.

Transpalatal Arch

The molars are banded and cast is poured as usual and the trans palatal arch wire is fabricated along the palate from one molar to other molar on other side an soldered.

Lingual Arch (Figs 10.7 and 10.8)

Similar to Nance palatal arch except that it lacks acrylic button and is fabricated for mandibular teeth.

Distal Shoe Space Maintainer (Fig. 10.9)

Synonyms: Intra alveolar/Eruption guidance space maintainer.

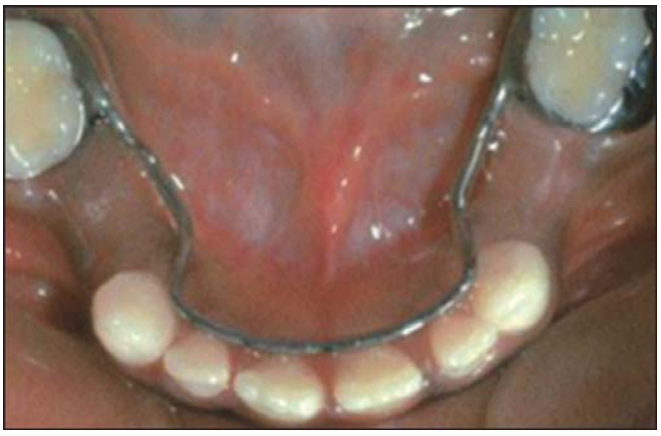


Fig. 10.7: Fixed lingual arch

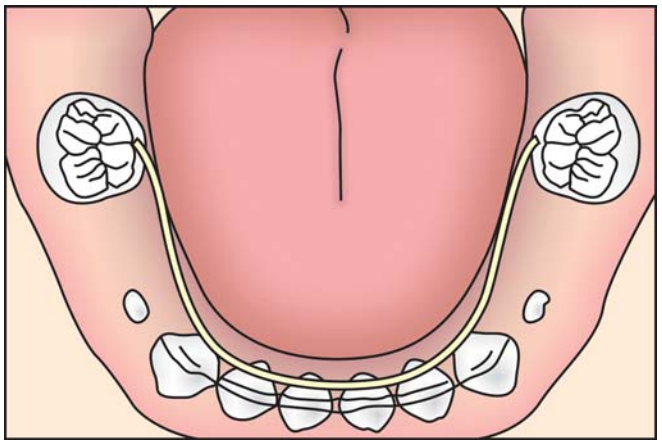


Fig. 10.8: Fixed lingual arch space maintainer

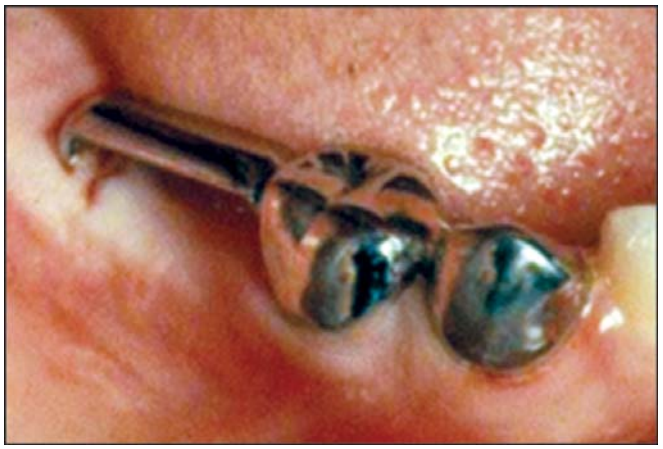


Fig. 10.9: Distal shoe space maintainer

Types:

a. Willets – Bar type gingival extension.
b. Roche's – 'V' shaped gingival extension.

Components:

1. A horizontal component bridging the mesiodistal divide left over by the premature loss of deciduous second molar.
2. Vertical component that extends 1 to 1.5 mm below the mesial marginal ridge of the unerupted permanent first molar.
3. Molar band, banded around deciduous first molar.

Contraindications:

1. If several teeth are missing in same quadrant as there is lack of abutment.
2. Medically compromised patients.

3. Poor oral hygiene.
4. Lack of patient or parental cooperation.

Removable Partial Denture (Fig. 10.10)

- Functional space maintainer.
- Helps in mastication in case of posteriors.
- Esthetics restored in case of anteriors.
- Phonetics is maintained.

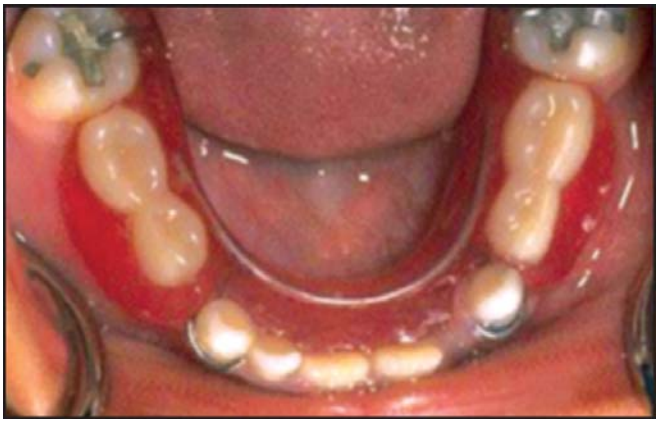


Fig. 10.10: Functional space maintainer

Disadvantages

- Easily lost/broken by patient
- Patient cooperation is must to wear the appliance.

Band and Loop Space Maintainer (Fig. 10.11)

Can be used for unilateral tooth loss and bilateral tooth loss on both sides.

Band and Bar Space Maintainer

Instead of loop a horizontal bar is soldered to the band on the abutment tooth.

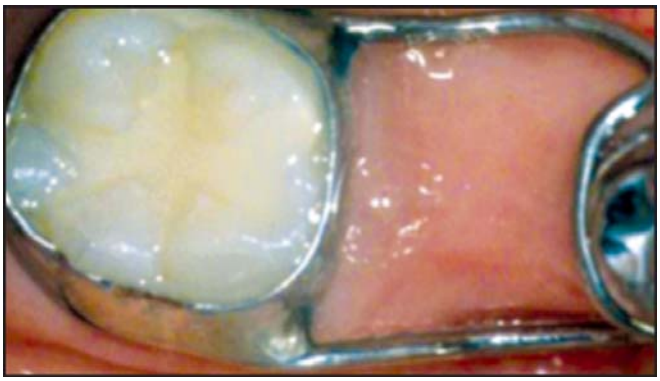


Fig. 10.11: Band and loop space maintainer

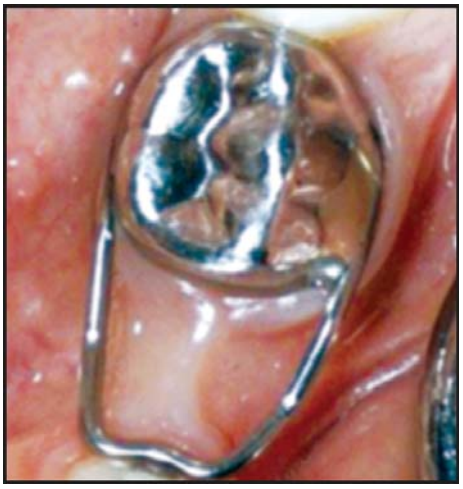


Fig. 10.12: Crown and loop space maintainer

Crown and Loop/Bar Space Maintainer (Fig. 10.12)

In cases of pulpally treated abutment tooth, a crown is adapted and either a loop/bar is soldered to the crown adjacent to the edentulous space.

INTERCEPTIVE ORTHODONTICS

The American Association of Orthodontists (1969) defined interceptive orthodontics as that phase of science and art of orthodontics employed to recognize and eliminate the potential irregularities and malpositions in the developing dentofacial complex.

The basic interceptive procedures that are undertaken by the interceptive pedodontist are:

1. Serial extraction
2. Space regaining
3. Correction of anterior and posterior crossbites
4. Oral habits elimination
5. Muscle exercises
6. Removal of soft or hard tissue interferences to the pathway of eruption
7. Resolution of crowding
8. Interception of developing skeletal malocclusion.

Space Regainence

If space maintenance is not carried out after premature loss of deciduous teeth; the space gets lost or closed by

shifting/drifting of teeth adjacent to the space resulting in the loss of arch length. The loss of arch length may impede on the space required for the eruption of succedaneous permanent teeth, thereby resulting in crowding, malocclusion, ectopic eruptions, etc.

The space lost can be regained by appliance therapy before the complete eruption of the permanent tooth. Such appliances are called **space regainers**. The methods of space regaining are divided into two broad groups.

1. Fixed appliances.
   - Open coiled space regainer
   - Herbst space regainer
   - Jack screw space regainer
   - Gerber space regainer.
2. Removable appliances.
   - Upper or lower Hawley's appliances with helical spring.
   - Hawley's appliance with palatal spring/slingshot elastic/expansion screws.

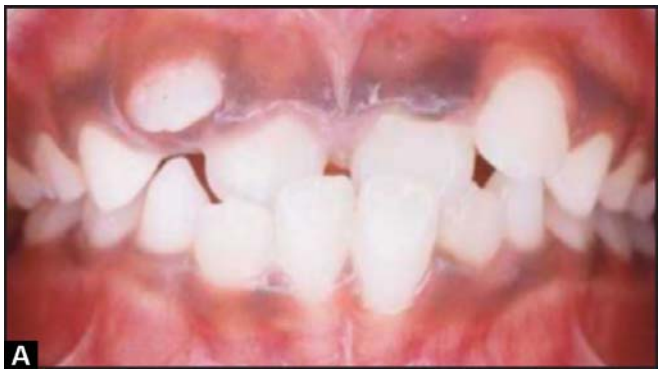
Correction of Anterior and Posterior Cross bites (Figs 10.13A to C)

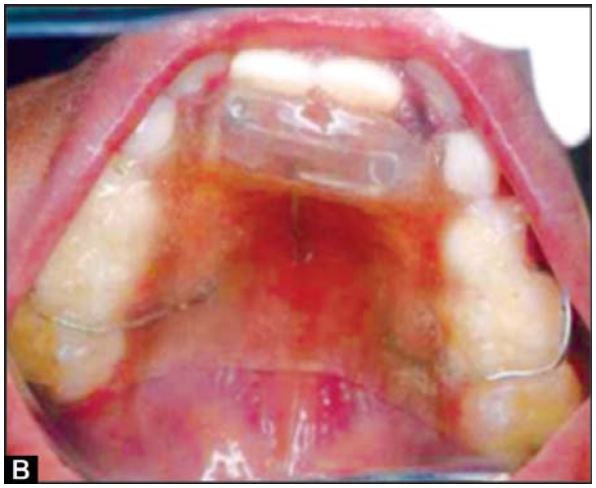
Cross bites should be corrected as soon as they are detected. The cross bites can be unilateral or bilateral, which can be true or functional in nature or a combination of the two. If the cross bite is left untreated; it will lead to skeletal malocclusion that requires corrective orthodontic treatment.

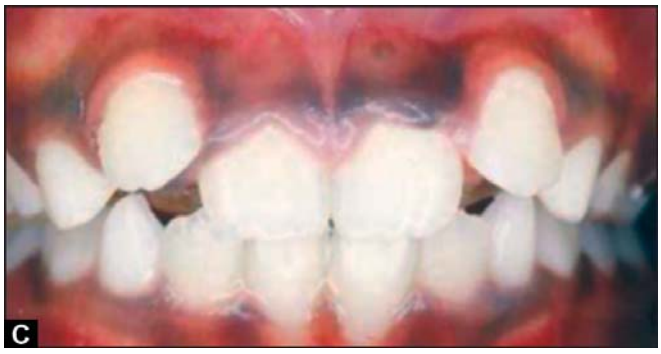
Commonly used appliances for correction of cross bites are:

- Tongue blade therapy
- Inclined planes
- Composite inclines
- Hawley's appliances with 'Z' spring
- Quad helix appliance.

The most easy and common method to intercept developing anterior cross bite is tongue blade therapy in which the wooden tongue blade is places behind the tooth which is erupting in cross bite at angle of about 60° to the occlusal plane and the patient should exert force by biting on it by using the lower anterior teeth as fulcrum for short period of 5-10 minute each time.







Figs 10.13 A to C: A. Developing anterior cross bite. B. Appliance therapy. C. Post treatment photograph

Elimination of Oral Habits

Correction of pernicious oral habits such as thumb sucking, tongue thrusting, lip biting, mouth breathing should be undertaken as a part of interceptive orthodontic procedure otherwise leading to development of dental malocclusions and over a long period becomes skeletal malocclusions.

Muscle Exercises

The purpose of muscle exercises is to create a normal health and functions of oro-facial musculature to aid in growth and development of normal occlusion.

Myotherapy is an adjunct to general mechanotherapy and has a synergistic effect in elimination of the habit/problem.

Muscle exercises are a various types targeted at different groups of orofacial musculature. That helps to guide the development of occlusion; allows optimal growth patterns and provides retention and stability in treated orthodontic cases.

The only disadvantages of muscle exercises is being its dependence on patient compliance and need to learn the correct technique, otherwise it may cause deleterious/undesirable effects.

Removal of Soft or Hard Tissue Impediments to Pathway of Eruption

Some of the common soft/hard tissue impediments to pathway of eruption are:

- Ankylosed
- Retained deciduous teeth
- Supernumerary teeth should be extracted by surgical intervention.

Resolution of Crowding

Crowding is one of the common problems present at different stages of development of dentition. Usually the crowding resolves but it tends to remain in case of:

- Lack of interdental spacing in primary dentition
- No change in intercanine width
- Early loss of deciduous teeth causing drifting/shifts of teeth into space
- Loss of dental arch space
- Deficient growth of the jaws etc.

Clinical Analysis of Crowding

Hereditary Crowding

The signs of a true hereditary tooth-size jaw-size discrepancy are :

1. Maxillary mandibular alveolodental protrusion without inter proximal spacing.
2. Crowded mandibular incisor teeth.
3. A midline displacement at the permanent mandibular incisors, resulting in the premature exfoliation of the primary canine on the crowded side.
4. A midline displacement of the permanent mandibular incisors, resulting in the premature exfoliation of the primary canine on the crowded side.
5. A crescent area of external resorption on the mesial aspect of the roots of the primary canines, caused by crowded permanent lateral incisors.
6. Bilateral primary mandibular canine exfoliation, resulting in an up righting of the permanent mandibular incisors; this, in turn, increases the overjet and/or the overbite.
7. A splaying out of the permanent maxillary or mandibular incisor teeth due to the crowded position of the unerupted canines.
8. Gingival recession on the labial surface of the prominent mandibular incisor.
9. A prominent bulging in the maxilla or mandible due to the crowding of the canines in the unerupted position.
10. A discrepancy in the size of the primary and permanent teeth, reducing the Leeway space.
11. Ectopic eruption of the permanent maxillary first molars, resulting in the premature exfoliation of the primary second molars; this indicates a lack of development in the tuberosity area.
12. A vertical palisading of the permanent maxillary first, second, and third molars in the tuberosity area, again indicating a lack of jaw development.
13. Impaction of the permanent mandibular second molar in the absence of treatment.

True hereditary tooth-size jaw-size discrepancies must be differentiated from crowded dentitions resulting from factors that are more environmental in nature. It is quite likely that true hereditary crowding will be treated with the aid of extractions and, if discovered early, with serial extraction. On the other hand, crowding resulting from environmental factors may be treated without extractions.

Environmental Crowding

Environmental crowding may result under the following conditions:

1. Trauma

2. Iatrogenic treatment
3. A discrepancy between mandibular tooth size and maxillary tooth size
4. A discrepancy between mandibular tooth size and maxillary tooth size
5. An aberration in the shape of teeth.
6. An aberration in the eruptive pattern of the permanent teeth
7. Transposition of teeth
8. Uneven resorption of primary teeth
9. Rotation of teeth
10. Premature loss of primary teeth resulting in the reduction of arch length due to subsequent drifting of permanent teeth
11. A reduction of arch length due to inter proximal caries in the primary teeth
12. Prolonged retention of primary teeth.

Management of Crowding

The stepwise management involves:

1. Observation of the dentition for timing of eruption and exfoliation of teeth space analysis, etc.
2. Disking/Slicing of primary teeth.
   It is an effective method to relieve crowding in case of minor space discrepancy disking is done along mesial surfaces of canine or deciduous first molars to relieve crowding of anteriors or posterior teeth.
3. Extractions and serial extraction.
4. Corrective orthodontic referral.

Serial Extraction

Tweed defines it as "the planned and sequential removal of the primary and permanent teeth to intercept and reduce dental crowding problems".

The term "serial extraction" was coined by Kjellgren in 1929 and popularized by Nance (1940) who has been called the '**father of serial extraction**'.

Indications

1. Class I malocclusions with anterior crowding space discrepancy of 10 mm more.
2. Lingual eruption of lateral incisors.
3. Midline shift of mandibular incisors due to displaced lateral incisors.
4. Premature loss of deciduous canine.
5. Abnormal canine root resorption.
6. Severe proclination of mandibular and maxillary interiors with associated crowding.
7. Ectopic eruption.
8. Ankylosis.

Contraindications

1. Mild to moderate crowding (about 8 mm or less)
2. Congenital absence of teeth providing space
3. Class II div 2 and Class III malocclusion
4. Spaced dentition
5. Extensive caries involving permanent first molars which cannot be conserved.
6. Open bite and deep bite
7. Cleft up and palate cases, etc.

Diagnosis and Treatments Planning

The primary step is assess that a malocclusion exists on clinical examination and the need for investigations and collection of diagnostic records. The recommended investigations are:

a. Study models
b. Radiographs
   — Periapical view
   — Orthopantamograph
   — Cephalograms with Cephalometric tracings
c. Mixed dentition analysis
d. Photographs.

Types of Serial Extraction Methods

- Tweed's method
- Dewel's method
- Nance method
- Grewe's method

It is important that before each step of extraction the sequence should be re-evaluated.

Tweed's Method (1966)

1. Proposed extraction sequence DC4.
2. Deciduous first molars are extracted at 8 yrs of age.
3. The deciduous canines are maintained to slow down the eruption of permanent canines.

4. As soon as the first premolars are in advanced stage of eruption, the deciduous canines along with first premolars are extracted.

Dewel's Method (1978)

1. Proposed extraction sequence CD4.
2. At 81/2 yrs of age deciduous canine are first extracted to provide space for alignment crowded anteriors.
3. At 91/2 yrs of age, as the incisor crowding resolves, the first premolar would have had their one third or three fourth of root completed; the deciduous first molar is extracted to allow first premolar to erupt prematurely.
4. Then the first premolars are extracted to allow the permanent canines to erupt in their place and in alignment.

Nance's Method

1. Proposed extraction sequence of D4C.
2. First the deciduous first molars are extracted at about 8 years of age, which is followed by extraction of first premolars and deciduous canines.

11

Child Psychology and Behavior Management

DEFINITION

It is the science or study of child's mind and how it functions. It is also defined as the science that deals with the mental power or interactions between the conscious and subconscious element in a child.

INTRODUCTION

Guidance of child's behavior in the dental office is the prerequisite to complete dental care. The concept of treating the patient and not just the tooth should be operative with all the patients, but is essential with the child patient. Mistakes made by the dentist are often hidden by the stoic nature of the adult patient or by subsequent failure of the patient to return. The effects of errors made with the child not only have to be faced immediately, but often have to be dealt with subsequently when the child returns unwillingly.

Another important aspect of treating the child patient, which is different from treating adult, is the intimate involvement of the parent in the relationship, which completes the '**Pedodontic treatment triangle**' with *Dentist, Child Patient* and the *Parent.* Since many of the child's attitudes towards society, likewise towards dental care are learned from parents, it is necessary to educate the parents in making the dental experience for the child a favorable one.

The importance of child psychology is to:

- Know the child better
- Understand the problem psychologically
- Deliver dental services in a meaningfully and effective manner
- Establish effective communication and gain confidence of the child and the parent
- Produce a comfortable environment for the dental team to work on the patient
- Teach the parent and the child, the importance of primary and preventive care.

For treating the child successfully, first we should have knowledge on the psychological and personality development of the child.

There are different theories of psychology (Table 11.1).

Table 11.1: Theories of psychology

I. Emotional development
1. Psychoanalytic theory or psychosexual theory by Sigmund Freud
2. Psychosocial theory or Eriksson's model of personality development
II. Cognitive development
1. Cognitive development theory by Jean Piaget
III. Learning and development of behaviour
1. Classical conditioning by Ivan Pavlov
2. Operant conditioning by BF Skinner
3. Observational learning (modeling) by Albert Bandura

FREUD'S PSYCHODYNAMIC THEORY

According to Freud, the personality is largely determined during the first few years of life during which the person develops characteristic ways of dealing with internal drives and conflicts.

Structure of Personality

It is composed of three mental structures, the **id**, the **ego** and the **superego.** What a person thinks, feels and does is a function of the actions and interactions of these three hypothetical structures called ***psychic triad***, each of these structures developing at a different time.

The Id

The id is that portion of the mind that contains the unconscious drives for pleasure and destruction. The id strives for immediate satisfaction of its drives. The two ways by which the id tries to achieve reduction of tension is:

a. Reflex action
b. Primary process thinking.

Reflex action: Simple response reflexively to stimulus in the environment, e.g. child's sucking on a nipple which reduces tension and quiets the aroused and unhappy child.

Primary process thinking: Creation of a fantasy about the object or behavior to reduce tension and satisfy the id's drives, e.g. the child might fantasize about nursing in order to partially reduce tension.

The Ego

Primary process thinking alone cannot ensure the survival of the child. Imagination of feeding is pleasurable, but

it does not satisfy nutritional requirements. This is the time when this second mental structure develops. The ego is *conscious and reality oriented* portion of the mind. It is conscious of what happens in the child's would and by perceiving the difference between the child and the external environment. It is also responsible for the child's capacity for self-awareness. It helps the id to obtain real rather than imaginary satisfaction. It follows the *reality principle* rather than the *pleasure principle*.

To follow the reality principle the ego uses the secondary process thinking or *realistic thinking* rather than simple *fantasy or primary process thinking*. Although it is more realistic than the id, the ego derives all its energy from the unconscious drives of the id and exists only to find the effective ways of satisfying those drives.

The Superego

It develops during the phallic period. It contains *moral principle* and *values* that have been acquired from the child's parents and society. It contains two sub-parts.

a. Conscience
b. Ego ideal

Conscience contains *moral prohibitions* against certain behaviors, especially those expressing the sexual and aggressive drives of the id.

Ego ideal is the image of what one ideally can be and how one ought to behave.

Libido: The energy the id produces to obtain pleasure.

Erogenous zones: Freud believed that at different ages, human beings experience tension most intensely in different areas of body. These areas require pleasure – producing stimulation to reduce or eliminate the tension. These areas are known as erogenous zones.

Development of Personality

Freud divided this into five stages.

1. The oral stage (first 18 months of life)
2. The anal stage (18 months to 3 or 4 years)
3. The phallic stage (3 or 4 to 6 years)
4. The latency period (6 to 11 years)
5. The genital stage (11 onwards)

Oral Stage

During the first 18 months of life, mouth is the most sensitive zone. The mouth experiences the most tension and requires the most tension reducing stimulation. Sucking, even on thumb or pacifier or keeping objects in the mouth is the means by which the tension in and around the mouth is reduced. Care related to feeding from parents is important in this stage.

Anal Stage

The child enters the anal stage around 18 months of age, when the locus of gratification changes to the anal region, either from retention or elimination of the feces. Parents want to control when and where to eliminate the feces as they begin toilet training at this stage. A child's pleasure in retaining or eliminating feces at his/her choice of time and place can lead to conflict with parents.

Phallic Stage

At about the age of 3 to 4 years, the child enters phallic stage during which the genitals become the most sensitive area, and the child derives pleasure by manipulating them. Again develop conflicts with parents as they try to curb this behavior.

Oedipus or *Electra complexes* mark this stage. Oedipus complex is for boys and Electra complex for girls. Freud proposed that children experience a desire to have sexual relationship with their parent of the opposite sex. Second aspect of this complex is a related wish, to eliminate the parent of the opposite sex, who stands as arrival for the affection of the opposite sex parent. The ego fears the consequences of expressing them and realizes that these drives cannot be satisfied directly. The ego obtains partial satisfaction by identifying with the parents of the same sex. Boys try to imitate their father; girls try to imitate the mothers. This process is known as *identification*.

The specific aspect of identification with parents is *accepting* their *values* and *morals*. This forms the third part of the personality, the superego. When the oedipal conflict is resolved and superego is formed, the phallic stage is over.

Latency Stage

This period extends through late childhood to puberty. Children may learn a good deal about the world around them, other people and their own skills, capacities and interests. However, there is little pressure from the id and little internal conflict.

Genital Stage

When the young person reaches puberty or sexual maturation, he or she enters the genital stage during which the person feels strong and adult sexual desires for the first time. This may also be a reawakening of old oedipal sexual and aggressive feelings.

From this point on the ego has to work hard to balance the demands of the id for sexual gratification with the conscience and ego ideal of superego. The adult's personality reflects how well the ego manages to do this.

Concept of Fixation

The desires of the id changes from age-to-age. However, some individuals never lose their desire of a particular kind of gratification, such as oral stimulation. This is called fixation. *Fixation* thus is a *failure of the development* in which the *individual continues to seek a particular kind of gratification* even after he/she has passed through the stage in which that kind of pleasure is normally sought.

Cause of Fixation

When a child is extremely frustrated in the pursuit of pleasure or over gratified, the person becomes continuously concerned with obtaining that pleasure, e.g. person getting fixated at oral stage might engage in behaviors that give direct oral stimulation, such as thumb/finger sucking habits, smoking or chewing habits. A person fixated on pleasure of expelling feces in the anal stage might be extremely sloppy, late and disorganized as an adult. A person might also be miserly, withholding money just as he or she withheld feces as a child if fixated on the pleasure of retaining feces.

ERIKSON'S PSYCHOSOCIAL THEORY OR THEORY OF PERSONALITY DEVELOPMENT

According to Erik Erikson there are eight stages in the human life cycle, of which the first five stages extend through adolescence. These stages are:

1. Development of Basic Trust (birth to 18 months) *Basic Trust vs. Mistrust.*
2. Development of Autonomy (18 months to 3 years) *Autonomy vs. Shame and Doubt.*
3. Development of Initiative (3 to 6 years) *Initiative vs. Guilt.*
4. Mastery of Skills (6 to 11 years) *Industry vs. Inferiority.*
5. Development of Personal Identity (12 to 17 years) *Identity vs. Role confusion.*

Basic Trust vs. Mistrust

This stage corresponds to the oral stage of Freud. The social interaction in this period involves basic trust at one extreme and mistrust at the other.

If the infant's needs are met as and when they arise, whose discomforts are quickly removed, cuddled, fondled and played with and talked to develop a sense of trust towards the world as a safe place and towards people as helpful and dependable. However if the care is inconsistent, inadequate and rejecting, a basic mistrust is fostered. Fear and suspicion develops towards the world in general and people in particular which carries through later stages of development.

The basic fear of this stage is ***"Fear of Abandonment***". The tight bond between parent and the child at this early stage of emotional development is reflected in a strong sense of "*Separation anxiety*" in the child when separated from the parent.

Dental treatment at an early age is preferable to do with the parent present, and if possible, while the child is being held by one of the parents. At later ages, a child who never developed a sense of basic trust is likely to be an extremely frightened and uncooperative patient who needs special effort to establish rapport and trust with the dentist and staff.

Autonomy vs. Shame or Doubt

It corresponds to anal stage of Freud. The emerging autonomy dimension builds on the child's motor and mental abilities. The child takes pride in new accomplishments and wants to do everything. If the parents recognize the young child's need to fulfill the capabilities at child's own pace, then a sense is developed of self control of muscles, impulses and the environment which will contribute to autonomy.

However, when the caretakers are impatient and do everything for the child, they reinforce a sense of shame and doubt. Now and then rarely rushing the child is not a problem. Only when caretaking is continuously overprotective and criticism of accidents (wetting, soiling, spilling or breaking) is harsh, the child develops an excessive sense of doubt in abilities to control the world and the self. The child who passes through this stage with a sense of autonomy is well prepared to be autonomous in later stages of life.

Principal anxiety or fear of this period is *Fear of loss of Love* and *Fear of Separation*.

To obtain cooperation from the patient is to have the child to think that whatever the dentist wants to do was his or her own choice. For a 2-year-old seeking autonomy, it is all right to open your mouth if you want to, but almost unacceptable to do it if someone tells you to. It is better to offer the child reasonable choices whenever possible. A child who is afraid of the situation will be unwilling for separation from parent. Simple procedures also should be done in presence of the parent. Complex procedures may require sedation or general anesthesia.

Initiative vs. Guilt

It corresponds to phallic stage of Freud. The child can initiate motor activities and no longer merely responds to or imitates the other children. It holds true for language and motor activities. Whether the child leaves this stage with a sense of initiative or guilt depends on how the parents respond to self initiated activities. Initiative is also reinforced when parents answer their questions (intellectual initiative) and do not deride or inhibit fantasy or play.

On the other hand, if the child is made to feel that certain activity is bad, that the questions are a nuisance, and that the play is silly and stupid, then the child may develop a sense of guilt over self initiated activities in general. The primary fear of this stage is *Fear of Bodily Injury.*

For most children, the first visit to the dentist comes during this stage of initiative. Going to the dentist should be constructed as an adventure in which the child can experience success. Success in coping with the anxiety of visiting the dentist can help develop greater sense of accomplishment. If poorly managed, the dental visit can result in a sense of guilt that accompanies failure. A child at this stage is curious about dental office and wants to learn about things found there. Initial appointment with parental presence and little treatment is important in getting the dental experience a good start. After the initial experience, the children at this stage can usually tolerate being separated from the parent for the treatment and likely to behave better, so that the independence rather than the dependence is reinforced.

Industry vs. Inferiority

It corresponds to the latency period of Freud or Elementary school years (6-11 years). Capabilities in this period are deductive reasoning, playing take-turn games, obedience to rules that are required for the above games.

When the children are encouraged and allowed to finish the efforts to make, do or build practical things and are praised and rewarded for the results, a sense of industry is enhanced. But parents who see their children's efforts at making and doing a 'mischief or making a mess' help to encourage their children a sense of inferiority. Also constant failure at academic level leads to inferiority. Principal anxiety of this stage is *Superego anxiety* created by child's own internal standards.

Behavioral guidance is done in this stage by clearly outlining the child, what to do or how to behave and then reinforcing it positively. Because of the child's drive for a sense of industry and accomplishment, cooperation with treatment can be obtained. They like to get attention of the peer groups and motivated by the facts that can

be noticed by the peers, e.g. improved appearance by orthodontic treatment rather than occlusion.

Identity vs. Role Confusion

It corresponds to genital stage of Freud, i.e. adolescence or 12-18 years. Maturation is seen mentally as well as physiologically. They experience new ways of looking and thinking about the world. Adolescents become capable of constructing theories and philosophies designed to bring all the varied and conflicting aspects of society into a working, harmonious and peaceful whole.

In a single word the adolescent is an impatient idealist who believes that it is as easy to realize an ideal, as it is to imagine it. Erickson believes that new interpersonal dimension that emerges during this period has to do with a sense of ego identity at the positive end and a sense of role confusion at the negative end.

The authority of the parents and others is usually rejected in this stage for establishing one's own identity and members of the peer group become important role models. At the same time, some separation from the peer group is necessary to establish one's own uniqueness and value. The inability to separate can lead to a poor sense of direction for the future, confusion regarding one's place in society, and low self-esteem. Atypical adolescent feels that the health problems are concerns of somebody else and neglects his own health concerns.

JEANS PIAGET'S COGNITIVE THEORY (FIG. 11.1)

Cognition refers to knowing and understanding. This involves three functional variants.

- **Assimilation:** This is concerned with observing, recognizing, taking up an object and relating it with earlier experiences or categories.
- **Accommodation** accounts for changing concepts and strategies as a result of new assimilated information. Piaget calls the strategies and mental categories as 'schemes'.
- **Equilibrium** refers to changing basic assumptions following adjustments in assimilated knowledge so that the facts fit better.

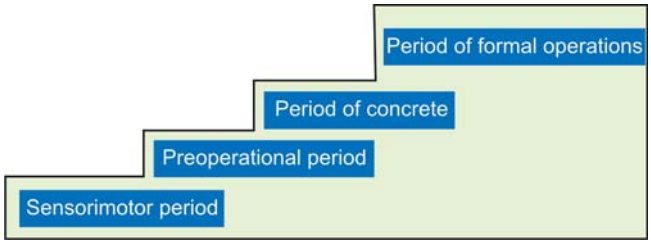


Fig. 11.1: Cognitive development

Periods of Cognitive Development

According to Piaget there are four distinctive periods of cognitive development under two broad categories.

1. Pre-verbal sensorimotor period, 18-24 months of life
2. Period of conceptual intelligence
   a. Preoperational period, 2 to 7 years
      i. Preconceptual stage 2 to 4 years
      ii. Intuitive stage 4 to 7 years
   b. Period of concrete operations, 7 to 11 years
   c. Period of formal operations, 11 years and beyond

Sensorimotor Period

It lasts from birth through approximately 2 years of age. During this stage infants understand their world by physically manipulating the world around them. They learn quiet literally by pushing, banging and poking. Infant begins to separate self from others. Piaget suggested that this physical exploration of the environment is cognitively productive and not just aimless play. Frequent interaction with objects leads eventually to the development of object permanence, the notion that an object continuous to exist even after it disappears from view. During second year children are capable of discovering a hidden abject even after it has been removed from one hiding place to another.

Symbolic thought is developed which is the capacity to construct a mental representation of an object – a symbol, pretending that an object is another, e.g. a spoon for a racing car. Using language, i.e. designating words for objects is another.

Preoperational Stage

By the end of sensorimotor period, the 2 years old is not surprised by any of the events around him. Most children can remember past events, engage in pretend

play and begin to use language effectively. But young children still demonstrate thought processes that are dramatically different those of the adult. A five-year-old child may think that a sandwich cut into four pieces contains more to eat than the same sandwich cut into two pieces. There are definite limitations to preoperational child's reasoning skills.

When a child is shown equal amounts of water in two glasses, of which one is short and wide, the other is narrow and longer the preoperational child thinks that the taller glass contains more water even if he is shown that the same quantity is being poured in both. They are influenced by what they see. Because the water level is higher in the tall glass, it must contain more water.

In the early stages of conceptual intelligence (2-7 years), the child's world is ego centered. His view of the world is animistic or artificialistic and he cannot distinguish what is real from what is not real. The descriptions of pain made by the child during these stages reflect this developmental level. He may, for example, feel that some one is the cause of the pain because that person was present at the time of painful experience. If he hurts himself by bumping against a door, he may hit the door so that hit gets hurt also.

The 2 to 7 years old child's thought is also centered only on one salient aspect of the problem or one feature of a multifaceted experience is emphasized in the child's perception. For instance, a 3-year-old may identify a series of pictures as cow, if each of the animals has horns like cow and, in fact, that is what the child has centered on in his recognition of a cow. A child can center on a salient aspect of a dental appointment. Obviously, centering on 'counting the teeth' or 'cleansing the teeth' is better than centering on tooth extractions, needles, etc.

The Concrete Operational Period

At age 7, child becomes capable of logical reasoning; the period during which the child begins to understand logical concepts. The child no longer makes judgments solely on the basis of how things appear. But there are limitations in child's reasoning in concrete operational period. Children reason logically when the problem is displayed before them. In the presence of objects that can be viewed and sometimes manipulated, they demonstrate a practical, concrete form of intelligence.

The child's thinking is more stable and he can understand many cause and effect relationships. With his knowledge he can understand that specific procedures cause pain. At the end of this stage and the beginning of the formal operational period, a child is able to express his pain like an adult would.

Formal Operational Period

People acquire new reasoning skills or formal operational thinking. The essence of thought is to reason hypothetically, to consider before hand all the possible solutions to a problem. Unlike concrete operational child, formal operational child can reason about what could be as well as what actually is.

LEARNING AND DEVELOPMENT OF BEHAVIOR

Classical Conditioning

It was first described by the Russian physiologist Ivan Pavlov, who discovered in the 19th century during his studies of reflexes that apparently unassociated stimuli could produce reflexive behavior.

Classical conditioning occurs readily with young children and can have a considerable impact on a young child's behavior on the first visit to a dental office. By the time a child is brought for the first visit to a dentist, even that visit is at an early age, it is highly likely that he or she will have had many experiences with pediatricians and medical personnel. When a child experiences pain, the reflex reaction is crying and withdrawal. In pavlovian terms, the infliction of pain is an unconditioned stimulus, but a number of aspects of the setting in which the pain occurs can come to be associated with this unconditioned stimulus.

For instance, it is unusual for a child to encounter people who are dressed entirely in white uniform or long white coats. If the unconditioned stimulus of painful treatment comes to be associated with the conditioned stimulus of white coats, a child may cry and withdraw immediately at first sight of a white coated dentist or

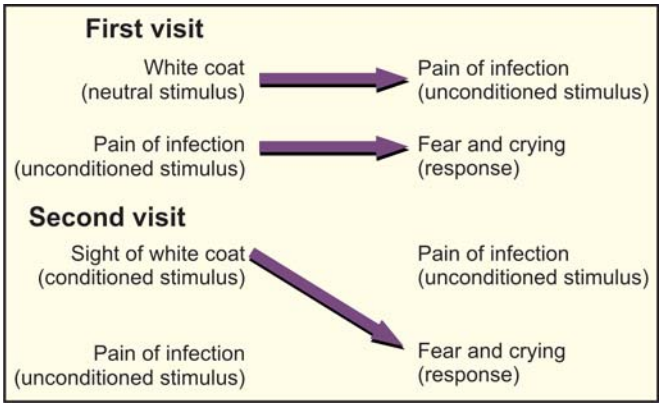


Fig. 11.2: Classical conditioning

(Fig. 11.2) dental assistant. In this case, the child has learned to associate the conditioned stimulus of pain and the unconditioned stimulus of a white coated adult and the mere sight of white coat is enough to produce the reflex behavior initially associated with pain.

Associations of this type tend to be generalized. Painful and unpleasant experiences can become generalized to the atmosphere of a physician/dentist's office.

Because of this association, behavior management in the dentist's office is easier if the dental office looks as little like the typical pediatrician's office hospital/clinic as possible. In practices where the dentist and auxiliaries work with young children they have found that it is helpful in reducing children's anxiety if their appearance is different from that associated with the physician. It also helps if they can make the child's first visit as different as possible from the precious visits to the physician. Treatments that might produce pain should be avoided if at all possible on the first dental visit.

The association between a conditioned and unconditioned stimulus is strengthened or reinforced every time they occur together (Fig. 11.3). Every time a child is taken to a hospital where something painful

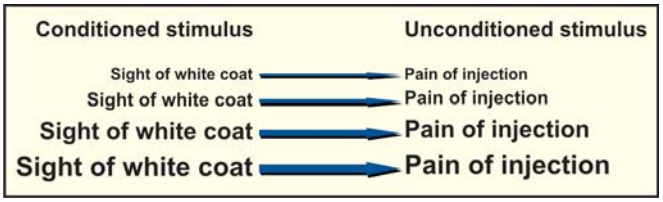


Fig. 11.3: Reinforcement

is done; the association between pain and the general atmosphere of that clinic becomes stronger, as the child becomes surer of his conclusion that bad things happen in such a place. Conversely, if the association between a conditioned stimulus and an unconditioned stimulus is not reinforced; the association between them will become less strong, and eventually the conditioned response will no longer occur.

This phenomenon is referred to as '*extinction of conditioned behavior*'. Once a conditioned response has been established, it is necessary to reinforce it only occasionally to maintain it. If the conditioned association of pain with doctor's office is strong, it can take many visits without unpleasant experiences and pain to extinguish the associated crying and avoidance.

Operant Conditioning

Operant conditioning which can be viewed conceptually as a significant extension of classical conditioning has been emphasized by the pre-eminent behavioral theorist BF Skinner. The basic principle of operant conditioning is that the consequence of behavior is in itself a stimulus that can affect future behavior (Fig. 11.4). In other words, the consequence that follows a response will alter the probability of that response occurring again in a similar situation.

In classical conditioning, a stimulus leads to a response; in operant conditioning, a response becomes future a stimulus. The general rule is that if the consequence of a certain response is pleasant or desirable, that response is more likely to be used again in the future; but if a particular response produces an

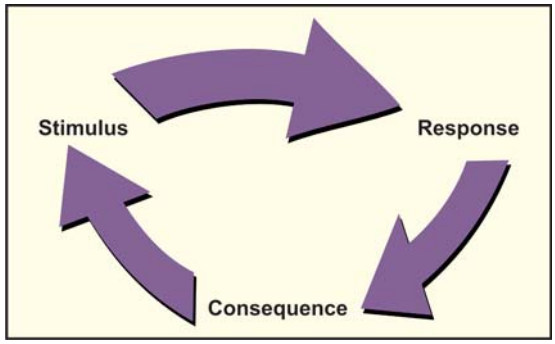


Fig. 11.4: Differences in operant conditioning and classical conditioning

Table 11.2: Four basic types of operant conditioning

	Probability of response increases	*Probability of response decreases*
Pleasant stimulus (S1)	S1 presented Positive reinforcement or reward	S1 withdrawn Omission or time-out
Unpleasant stimulus (S2)	S2 withdrawn Negative reinforcement	S2 presented Punishment

unpleasant consequence, the probability of that response being used in the future is diminished.

Skinner describes four basic types of operant conditioning, distinguished by the nature of consequence (Table 11.2).

Positive Reinforcement

If a pleasant consequence follows a response, the response has been positively reinforced and the behavior that led to this pleasant consequence becomes more likely in the future. For example, if a child is given a reward such as a toy or good remarks for behaving well during his/her dental visit; he/she is more like to behave well during future dental visits; her behavior was positively reinforced.

Negative Reinforcement

It involves withdrawal of an unpleasant stimulus after a response. Note that negative reinforcement is not a synonym for their term punishment.

For example a child, who views a visit to the hospital/clinic, as an unpleasant experience may throw a temper tantrum at a prospect of having to go there. If this behavior (response) succeeds in allowing the child to escape the visit to the clinic, the behavior has been negatively reinforced and is more likely to occur next time a visit to the clinic is proposed. The same can be true, of course in the dental clinic. If behavior considered unacceptable by the dentist and his staff nevertheless succeeds in allowing the child to escape from dental treatment that behavior has been negatively reinforced and is more likely to occur next time the child is in the dental office.

In dental practice, it is important to reinforce only desired behavior, and it is equally important to avoid reinforcing behavior that is not desired.

Omission

Also called as 'timed-out', involves removal of pleasant stimulus after a particular response. For example if a child who throws temper tantrum has his favorite toy taken away for a short time as a consequence of this behavior, the probability of similar behavior is decreased. Because children are likely to regard attention by others as a very pleasant stimulus, withholding attention following undesirable behavior is use of omission that is likely to reduce the unwanted behavior.

Punishment

Punishment occurs when an unpleasant stimulus is presented after a response. This also decreases the probability that the behavior that promoted punishment will occur in the future.

Omission and punishment should be used sparingly and with caution. Since a positive stimulus is removed in omission, the child may react with anger or frustration. When punishment is used both fear and anger sometime result. In fact punishment can lead to a classically conditioned fear response; hence these two types of operant conditioning should be used very cautiously.

One mild form of punishment that can be used with children is "voice control".

Observational Learning (Modeling)

Another potent way that behavior is acquired is through imitation of behavior observed in a social context. There are two distinct stages in observational learning.

1. Acquisition of the behavior by observing it.
2. Actual performance of that behavior.

A child can observe many behaviors and thereby acquire the potential to perform them without immediately demonstrating or performing that behavior. Children are capable of acquiring almost any behavior

that they observe closely and that it is not too complex for them to perform at their level of physical development.

A child is exposed to tremendous range of possible behaviors, most of which he acquires even though the behavior may not be expressed immediately or ever. The characteristics of the role model are important. If the model is liked or respected, the child is more likely to imitate him/her. For this reason, a parent or older sibling is often the object of imitation by the child. For adolescents the peer group is the major source of role models.

Observational learning can be an important tool in management of dental treatment. If a young child observes an older sibling undergoing dental treatment without complaint or uncooperative behavior, he/she is likely to imitate this behavior.

As stated by its profounder Albert Bandura (1969) Learning occurs only as a result of a direct experience which can be vicarious.

Modeling can be done by:

a. Live models – siblings, parents, friends, etc.
b. Filmed models
c. Posters
d. Audio visual aids, etc.

Stone and Church Classification of Child Development

Stone and Church (1975) classified the child development into five different stages.

1. Infant – 15 months
2. Toddler - 15 months to 2 years
3. Preschooler – 2 to 6 years
4. Middle year's child – 6 to 11 years
5. Adolescent – 11 to 18 years.

Infant

The 15 months period of infancy corresponds to the oral stage of Freud, stage of Trust vs. Mistrust of Erikson. This is the period in which the baby learns about the world it is placed, whether it is a good and acceptable place or a locus of hurt. The baby advances from relative helplessness to a positive, ambulatory toddler.

At about 6 months of age the baby begins to teeth and chew on anything in close proximity to the mouth and minor illness begins to appear as maternal antibody protection is disappearing. The first form of fear, referred to as *stranger anxiety* (to persons outside the accustomed environment) is seen. Another source of anxiety for the infant is due to the concept of trust vs. mistrust. The mistrust when the infant's needs are not adequately met, leads to fear and *distrust*.

Infant and parent each wield an influence over each other. Training the infant in regularity will give the infant their first lessons in character building. Parents who abuse their child because of personal problems lead to retarded and intellectually substandard children.

Toddler (15 Months to 2 Years of Age)

He or she is still very much a baby, taking one or two naps daily, and yet is developing unbaby like skills. The toddler is rapidly developing in cognitive and verbal skills and self-awareness is moving to the forefront.

Cooperative behavior and reasoning power are not observed. The child is capable of perceiving why dental measures need to be accomplished or realizing the cooperative behavior in the dental office.

Toddler is a child developing and growing in knowledge and motor skills but still an immature individual.

Preschooler (Child of 2 to 6 Years of Age)

The preschooler *"wears"* his or her *"personality"* whose behavior pattern is easily observed. Their behavior is influenced and shaped to a considerable degree by his or her immediate environment. He or she is more skilled in the use of words and symbols and more effective in the use of words and symbols and more effective in interpersonal communication. They will be expanding their circle to people outside the family. Aggression and sympathy co-exist with them. They adapt the behavior of familiar individuals like facial expressions, verbal mannerisms and gestures. *Fantasy,* pretension and strong telling by older people are accepted as facts. They create imaginary companions and situations. Emotional conditions are often made apparent by drawings.

Self-awareness is observed and fears about real and unreal things as a result of this are seen.

Time frames are not workable and meaningless for preschooler up to 5 years. They will not have logic on space, usually exaggerated in drawings. Causal relations change sequentially with the increase in overall knowledge, intelligence and thinking. At first they are *animistic*, assuming that all things have life, next *artificialism*, that all circumstances and things are human creations. Then they are more realistic in thinking with time.

Middle Years Child (Child of 6-12 Years Age)

This is rather peaceful period of time compared with the hustle and bustle nature of preschooler.

This is the time of "*loose tooth*" and the time for moderately rapid physical growth. This is the time for reaching out of independent identity, peer groups and sexual growth latency. This is the time for scapegoating when disobedient and time for imaginary and realistic fears. Thinking becomes logical and reversible, Learns conversations. He or she can now reflect, reason and understand logical relationships.

Adolescent (from Age 12 to Maturity)

It is a pause in the cycle of life. He or she is no longer a child but not yet an adult. Early development is marked by pubescence. Preadolescent period is marked by physical growth spurt, maturation of secondary sexual characteristics and changes in body proportions. Girls are initially ahead of boys but boys later catching up. Sexual maturation is marked by first menses in girls and presence of live spermatozoa in the urine for boys. Main point of issue is the identification of him or her for this new role.

A change in morphological and emotional characteristics is seen. Self-awareness becomes intensified and results in a new push for independence. They direct increasingly towards peer groups with less family supervision and restraint and more privileges. They concern about peer status and independent self but still welcome intervention into problems over which they have questionable control. They want to be popular and selective about friends and things.

Separation-Individuation Theory by Margaret s Mahler

Margaret believed that the "**psychological birth is not simultaneous to the biological birth**." She learned that disturbed children can work with a therapist and succeeded in a group setting, but that psychotic children need one on one help. Her main interest was in normal childhood development, but spent much of her time with psychiatric children and how they arrive at the "self".

Her theory for the developmental phase is as follows:

Normal Autistic Phase: Birth to 4 Weeks

a. The infant is oblivious to everything but himself
b. State of half –asleep, half-awake
c. Major task to phase is to achieve homeostatic equilibrium with the environment.

Normal Symbiotic Phase: 4 Weeks to 5 Months

a. He begins to recognize others in his universe, not separate beings, but as extensions to himself
b. Dim awareness of caretaker, but infant still functions as if he and caretaker are in state of undifferentiating or fusion
c. Social smile characteristic (two to fourth months).

Separation-Individuation Phase

This consists of four sub-phases that overlap. The next 5 to 36 months. It is during the critical sub-phases that physic is shaped and lays the foundation how the individual interprets and responds to information in his reality. The first three years of life are critical in determining personality and mental health. The sub-phases are:

First sub-phase: *Differentiation 5 to 10 months*

a. Process of hatching from autistic shell, i.e. developing more alert sensorium that reflects cognitive and neurological maturation.
b. Beginning of comparative scanning i.e. comparing what is and what is not mother.
c. *Characteristic anxiety:* Stranger anxiety, which involves curiosity and fear (most prevalent around 8 months).

Second sub-phase: *Practicing 10 to 16 months*

a. Beginning of this marked by upright locomotion- child has new perspective and also mood of elation.
b. Mother used as home base.
c. *Characteristic anxiety:* Separation anxiety

Third sub-phase: *Rapprochement 16 to 24 months*

a. Infant now a toddler – more aware of physical separateness, which dampens mood of elation.
b. Child tries to bridge gap between himself and mother– concretely seen as bringing objects to mother.
c. Mother's efforts to help toddler often not perceived as helpful temper tantrums typical.
d. *Characteristic event:* Rapprochement crisis. Wanting to be soothed by mother and yet not be able to accept her help.
e. *Symbol of rapprochement:* Child standing on threshold of door not knowing which way to turn in helpless frustration.
f. Resolution of crisis occurs as child's skills improve and child able to get gratification from doing things himself.

Fourth sub-phase: *Object constancy 34 to 36 months*

a. Child better able to cope with mother's absence and engage substitutes.
b. Child can begin to feel comfortable with mother's absences by knowing she will return.
c. Gradual internalization of image of mother as reliable and stable.
d. Through increasing verbal skills and better sense of time, child can tolerate delay and endure separations.

Theory of Hierarchy of Needs by Abraham Maslow (Fig. 11.5)

Most of the earlier psychologists studied people with psychological problems. Abraham Maslow studied successful people and decided that almost everyone wants to be happy and loving but they have particular needs that they must meet before they can act unselfishly.

Human beings are motivated by unsatisfied needs, and that certain lower needs need to be satisfied before higher needs can be satisfied. Maslow said that most of the people want more than they have. Once a person has met their most basic needs, they then develop higher needs. Maslow said, "*as one desire is satisfied, another pops up in its place*." Maslow created a hierarchy of needs with five levels:

Fig. 11.5: Theory of hierarchy of needs

Physiological Needs

Biological necessities such as food, water, oxygen, sleep, sex, etc. these needs are the strongest because a person would feel sickness, irritation, pain, discomfort, etc or may die also if they were not met.

Safety Needs

People feel unsafe during emergencies or times of disorder like rioting. Children more commonly have this need met when they feel afraid. These needs are mostly psychological in nature. We need the security of a home and family. However, if a family is dysfunction, i.e. an abusive husband, the wife cannot move to the next level because she is constantly concerned for her safety. Love and belongingness have to wait until she is no longer cringing in fear. Many in our society cry out for law and order because they do not feel safe enough to go for a walk in their neighborhood.

Love and Belonging Needs

The need to escape loneliness and alienation to give and receive love, and a sense of belonging.

Esteem Needs

The need to feel valuable; to have self-respect and the respect of others. If a person does not fulfil these needs, they feel inferior, weak, helpless, and worthless.

Self-Actualization Needs

Maslow taught that a very small group of people reach a level called self-actualization, where all of their needs are met. Maslow described self-actualization as a person's finding their "calling". He said, "A musician must make music, an artist must paint, and a poet must write".

Many people confuse self-actualization with fame or fortune but often this is not the case. While wealthy or celebrated people might reach self-actualization many psychologists believe that most people who have reached the highest level of happiness are unknown beyond their circle of family and friends. Societies develop when people reach a particular level in Maslow's hierarchy. Once people meet their physiological needs and they feel safe, they begin to develop a culture and an advanced civilization.

Emotional Development

Emotion is a state of mental excitement characterized by physiological, behavioral changes and alterations of feelings. A child expresses different kinds of emotion in response to different stimuli at different instances such as:

a. Distress or cry
b. Anxiety
c. Fear.

Characteristics of commonly seen emotions in child are:

Distress or Cry

- **At birth:** It is a primary emotion a child presents with vigorous body movements due to hunger, colic, internal cause or change in environment.
- **At six months:** Replaced by vocalization and as a manner to express or gain parents attention.
- **During preschool:** Reasons of physical pain, cry or distress is less seen.
- **During school years:** It is rapidly decreased and very rarely occurs.
- **In young adult:** Distress or cry only due to grief and usually expressed in privacy.

Types of Cry Seen in Children

1. *Obstinate cry:*
   - Child throws temper tantrums to avoid/escape dental treatment
   - It is loud and high pitched
   - As an outlet of anxiety.
2. *Frightened cry:*
   - Usually accompanied by a torrent of tears
   - Breath catching episodic sobs.
3. *Hurt cry:*
   - May be very loud, sudden outburst
   - Accompanied frequently by a small whimper
   - Usually child expresses with tear rolling down the corner of eye and does not resist/make any sound during dental treatment.
4. *Compensatory cry:*
   - It is not a cry
   - Usually it is slow, low pitched and monotone in nature
   - It is a typing of coping mechanism developed to unpleasant auditory stimulus.

Anxiety and Fear

Anxiety is one of the primary emotions acquired soon after birth. It is a personality trait and is apprehension, tension or uneasiness that stems from anticipation of danger, the source of which is largely unknown or unrecognized.

Fear is the emotional response to a consciously recognized and usually external threat or danger. It is a primitive response developed to protect the individual from harm and self-destruction.

Fears are of two types:

1. Objective fears
2. Subjective fears

Objective fears: These are acquired objectively or those produced by direct physical stimulation of the sense organs (seen, felt, smelt, or contacted) but not of parental origin, which are disagreeable and unpleasant in nature.

- Fears from previous unpleasant contact with dentistry
- Unrelated experiences like repeated hospitalization leading to fear of uniforms worn by dental team or

even characteristic smell of hospital, drugs or chemicals associated with unpleasantness arouse fear.

Subjective fears: These are based on the feelings and attitudes suggested to the child by others without the child personally experiencing them. These are imitative, suggestive or imaginative fears.

Suggestive fears are acquired by imitation by observation of others. These imitative fears are transmitted while displayed by others (parent) and acquired by the child without being aware of it. They are generally recurrent, deep seated and are difficult to eradicate. Displayed emotion in parent's face creates more impression than verbal suggestions. Even a tight clenching of the child's hand in dental office while undergoing dental treatment crates fear in child's mind about dental treatment.

These fears also develop from friends, playmates, reading books and periodicals, watching media and theater and depend on repetition.

Imaginative fears: As the child's imaginative capabilities develop, they become more intense with age and mental development with certain age.

Value of fear: Fear lowers the threshold of pain so that every pain produced during dental treatment becomes magnified.

Fear has safety value when given proper direction and control. Since fear producing stimuli can cause actual harm to the child, fear is a protective mechanism for self-protection. The nature of fear can be utilized to keep the child away from dangerous situation of either social or physical nature. If the child does not fear punishment or parental disfavor, his behavior may make him a threat to society.

The proper training of the child by parent should be not in the direction of eradicating fear, but rather in that of channeling it toward dangers that really exist, and away from situations where no danger lies. The child should be taught dental office is not a place to fear. Dentistry should not be employed as a threat or punishment. Using it in this manner creates fear of dentistry or dentist. On the other hand if the child has become attached to the dentist, fear of loss of his approval may have some value in motivating the child for dental treatment.

Mental and Emotional Development (Maturation) with Age

Birth to Two Years

It is the infancy stage corresponding to oral stage of Freud and Erikson's Trust vs. Mistrust. First form of fear or anxiety is seen in this stage, i.e. Fear of strangers other than the immediate family members. Fear developed from the conflict of basic trust vs. mistrust also may be seen.

Two Years Old

Vocabulary is limited to 12-1000 words. They are in pre-cooperative stage of lacking cooperative ability. Solitary play is preferred, as child has not yet learned to play with other children. The child is too young to be reached with words alone and must handle and touch objects in order to grasp their meaning fully.

Fear or anxiety of this age group is fear of falling, sudden jerky movements, bright lights, separation from the parents and fear of strangers.

Three Years Old

Communication is easier. Child has great desire to talk and often enjoy telling stories.

Fear of this age group is fear of strangers

Four Years Old

They are usually listeners to explanations with interest and normally responsive too verbal directions. They usually have lively minds and may be great talkers, although they tend to exaggerate in their conversation. In some situations they may be defiant.

There is increased ability to evaluate fear producing stimulations. Intelligent children display more fear, may be because of greater awareness of the danger and reluctance to accept verbal assurance without proof.

Fears of this age group: Fear of falling, of noise and of strangers is lessoned. Fear of bodily injury is present. Prick of hypodermic needle or sight of blood produces increased response disproportionate to that of pain.

Five Years Old

This is the age of readiness to accept group activities and community experience. If prepared properly by the

parents there is little fear of separation. Children of this age are proud of their possessions. Comments about clothes, toys, etc. can be effective.

Fantasy also plays very important role. It serves as a buffer for emotional problem. Children will combat on imaginative level, things they fear in reality. By so doing, the children not only gain comfort but also develop the courage and poise to meet the real situation. Children will do with pleasure in fantasy what they dislike to do in reality. The make believe play of going to the dentist and having the teeth worked on might help in dispelling undue subjective fears of a child.

Six to Twelve Years Old

Children of this age group learn from outside world and become increasingly independent of their parents. These are the years in which closely-knit groups and gangs are formed.

These are important years of learning, how to get along with other people and to abide by the rules of the society (reasoning).

Child can usually resolve fears of dental procedures if dentist explains and reasons will. Child has also learned to tolerate unpleasant situations and has marked desires to be obedient, carrying frustrations well. The child develops considerable emotional control. However, objects to people making light of his suffering, bullying, injustice or ridiculing whether it is from a friend or a dentist.

Teenage

Girls especially become concerned about their appearance. They like to be as attractive as possible. This interest in cosmetics can be used as a motivating factor for seeking dental attention. To satisfy their ego they will be willing to cooperate.

BEHAVIORAL SCIENCE AND MANAGEMENT

Terminology

Behavior: It is an observable act. It is defined as any change observed in the functioning of an organism.

Behavioral science: Is the science that deals with observation of behavioral habits of man and lower animals in various physical and social environments including behavior pedodontics, psychology, sociology and social anthropology.

Behavioral pedodontics is a study of science that helps to understand development of fear, anxiety and anger as it applies to child in the dental situations.

Behavior management is defined as the means by which the dental health team effectively and efficiently performs dental treatment and thereby instills a positive dental attitude (Wright 1975).

Behavior shaping is the procedure which slowly develops behavior by reinforcing a successive approximation of the desired behavior until the desired behavior comes into being.

Behavior modification is defined as the attempt to alter human behavior and emotion in a beneficial way and in accordance with the laws of learning.

Classification of Behavior Patterns in Children

Wilson's Classification (1933)

a. *Normal/bold:* A child is brave enough to face. Entirely new situations and is very friendly, co-operative with dentist/dental staff.
b. *Tasteful or timid:* A child is shy but accepts all dental treatment silently.
c. *Hysterical/rebellions:* A child throws frequent temper tantrums and rebellious against even painless dental procedures.
d. *Nervous/fearful:* A child appears tense anxious and expresses immense fear for any dental procedure.

Frankel's Behavior Rating Scale (1962)

Rating 1: Definitely negative

- Refusal of treatment, crying forcefully, fearful, or any other evidence of extreme negativism.

Rating 2: Negative

- Reluctant to accept treatment, uncooperative, some evidence of negative attitude but not pronounced

Rating 3: Positive

- Acceptance of treatment, at times cautions, and willingness to comply with dentist, at times reserved, but patient follows the dentist's directions co-operatively.

Rating 4: Definitely positive

- Good rapport with the dentist, interested in dental procedures, laughing and enjoying
- An ideal patient to treat
- Acts as a best model to other patient's to modify their behavior.

Lampshire Classification (1970)

1. *Co-operative:* child is physically and emotionally comfortable and relaxed. Shows full co-operation throughout procedure.
2. *Tense co-operative:* The child is tensed, anxious but co-operative at the same time.
3. *Outwardly apprehensive:* Initially avoids treatment, eventually accepts dental treatment.
4. *Fearful:* Needs emotional reinforcement to alleviate fears of dental treatment.
5. *Stubborn/defiant:* Passively resists dental treatment
6. *Hypermotive:* A child is easily and greatly agitated and resorts to temper tantrums.
7. *Handicapped:* Physical/emotional/mental impairments.
8. *Emotionally immature:* Infants, toddlers, etc.

Wright's Classification (1975)

It is a clinical classification places children in three categories:

- Co-operative
- Lacking in co-operative ability
- Potentially co-operative.

Behavior Management

Behavior management is based on scientific principles. The proper implementation requires an understanding of these principles. However it is more than pure science and requires stills in communication, empathy, coaching and listening. It is a clinical art form and still built on a foundation of science. The goals of behavior management are:

- To establish communication
- Alleviate fear and anxiety
- Deliver quality dental care
- Build a trusting relationship between dentist, parent and child
- Promote child's positive attitude towards oral/dental health.

Behavior management can be classified as follows in Table 11.3.

Table 11.3: Classification of behavior management

I.	**Basic behavior management**
	1. Communicative management
	i. Voice control
	ii. Non-verbal communication
	iii. Tell-show-do
	iv. Positive reinforcement
	v. Retraining
	vi. Parental presence/absence
	2. Behavioral shaping
	• Desensitization
	• Modeling
	• Contingency management
	3. Audio analgesia
	4. Bio-feedback
	5. Hypnosis
	6. Humor
	7. Coping
	8. Relaxation
	9. Implosion therapy
	10. Nitrous oxide/oxygen inhalation sedation
II.	**Advanced behavior management**
	1. Aversive conditioning
	• HOME and HOMAR
	2. Medical immobilization
	3. Sedation
	4. General anesthesia
	5. Treatment immobilization/physical restraints/ restraining

Basic Behavior Management

Communicative Management

Communicative management is used universally in pediatric dentistry with both the cooperative and uncooperative child. It comprises the most fundamental form of behavior management. It is the basis for establishing a relationship with the child which may allow successful completion of dental procedures and, at the same time help the child develop a positive attitude towards dental health.

Communicative management is comprised of a host of communication techniques which when integrated together enhances the evolution of a compliant and relaxed patient. It is an ongoing subjective process rather than a singular technique and is often an extension of the personality of the dentist.

Voice control:

i. **Description:** Voice control is a controlled alteration of voice volume, tone or pace to influence and direct the patient's behavior.
ii. **Objectives:**
   a. To gain the patient's attention and compliance
   b. To avert negative or avoidance behavior
   c. To establish appropriate adult-child roles.
iii. **Indications:** May be used with any patient.
iv. **Contraindications:** None.

Non-verbal communication:

i. **Description:** Non-verbal communication is the reinforcement and guidance of behavior through appropriate contact, posture and facial expression
ii. **Objectives:**
   a. To enhance the effectiveness of other communicative management technique
   b. To gain or maintain the patient's attention and compliance.
iii. **Indications:** May be used with any patient.
iv. **Contraindications:** None.

Tell-show-Do technique (Addleston 1959):

i. **Description:** Tell-show-do is a technique of behavior shaping used by many pediatric professionals. The technique involves verbal explanations of procedures in phrases appropriate to the developmental level of the patient (Tell); demonstrations for the patient of the visual, auditory, olfactory and tactile aspects of the procedure in a carefully defined, non-threatening setting (show); and then, without deviating from the explanation and demonstration completion of the procedure (Do).

   The Tell-show-do technique is used with communication skills and positive reinforcement.
ii. **Objectives:**
   a. To teach the patient important aspects of the dental visit and familiarize the patient with the dental setting
   b. To shape the patient's response to procedures through desensitization and well described expectations.
iii. **Indications:** May be used with any patient.
iv. **Contraindications:** None.

Positive reinforcement:

i. **Description:** In the process of establishing desirable patient behavior, it is essential to give appropriate feedback. Positive reinforcement is an effective technique to reward desired behaviors and thus strengthen the recurrence of those behaviors. Social reinforcement includes positive voice modulation, facial expression, verbal praise and appropriate physical demonstrations of affection by all members of the dental team.

   Non-social reinforcers include tokens and toys and gifts.
ii. **Objective:** To reinforce desired behavior.
iii. **Indications:** May be useful for any patient.
iv. **Contraindications:** None.

Retraining: The approaches to retraining falls under three main categories:

a. Avoidance
b. De-emphasis and substitution
c. Distraction

i. **Objectives:**
   a. To decrease the perception of unpleasantness
   b. To avert negative or avoidance behavior.
ii. **Indications:** May be used with any patient.
iii. **Contraindications:** None.

Parental presence/absence:

i. **Description**: This technique involves using the presence/absence of the parent to gain co-operation for treatment. Practitioners are united in the fact that communication between dentist and child is paramount and that this communication demands focus on the part of both parties. Children's respect

to their parent's presence or absence can range from very beneficial to very detrimental. It is the responsibility of each practitioner to determine the communication methods that best optimizes treatment setting; recognizing his/her own skills, the abilities of particular child, and the desires of the specific parent involved.

ii. **Objectives:**
   a. To gain patients attention and compliance
   b. To avert negative behavior
   c. To establish appropriate adult – child roles
   d. To enhance the communication environment.

iii. **Indications:** May be used with any patient.

iv. **Contraindications:** None.

Behavioral Shaping

Desensitization (1975): Wolpe's contribution has been the systematic desensitization as a behavior modification procedure uses two chief elements:

1. Gradational exposure of child to his/her fears.
2. Induced state of incompatibility with his/her fears.

The therapist creates a list of steps arranged as a hierarchy from the least to most stressful procedure. The patient while in a state of relaxation is exposed one step at a time, each step presented repeatedly until there is no evidence of stress or antagonism on the patient's part. On satisfactory completion of the hierarchy procedure the patient should be desensitized to predominant fear.

Contingency management: It is a method of modifying the patient behavior by presentation or withdrawal of reinforces.

Nitrous Oxide/Oxygen Inhalation Sedation

Description: Nitrous/oxide/oxygen inhalation sedation is a safe and effective technique to reduce anxiety and enhance effective communication.

Its onset of action is rapid, the depth of sedation is easily titrated and reversible, and recovery is rapid and complete. Additionally nitrous oxide mediates a variable degree of analgesia, amnesia and Gag reflex reduction.

The need to diagnose and treat, as well as, the safety of the patient and practitioner should be considered before the use of nitrous oxide. The decision to use nitrous oxide must take into consideration;

1. Alternative behavioral management modalities
2. Dental needs of the patient
3. The affect on the quality of dental care
4. Patient's emotional development
5. Patient's physical considerations.

Written informed consent must be obtained from a legal guardian and documented in patient's record prior to use of nitrous oxide.

The patient's record should include:

a. Percent nitrous oxide/oxygen and /or flow rate
b. Duration of the procedure
c. Post-treatment oxygenation procedure.

Objectives:

1. To reduce or eliminate anxiety
2. To reduce untoward movement and reaction to dental treatment
3. To enhance communication and patient co-operation
4. To raise the pain reaction threshold
5. To increase tolerance for longer appointments
6. To aid in treatment of the mentally/physically
7. To reduce gagging.

Indications:

1. A fearful, anxious or obstreperous patient
2. Certain mental, physical or medical disability
3. A patient whose Gag reflex interferes with dental care
4. A patient for whom profound local anesthesia cannot be obtained.

Contraindications:

1. May be contraindicated in some chronic obstructive pulmonary diseases
2. Patients with severe emotional disturbances or drug related dependencies
3. Patients with sickle cell disease
4. Patient's treated with Bleomycin sulfate.

Advanced Behavior Management

Introduction

Most children can effectively be managed using the techniques outlined in basic behavior management.

However, children occasionally present with behavioral considerations that require more advance technique.

Dentists considering the use of such technique should seek additional training that involves both didactic and experiential mentored training.

Hand–over–Mouth Exercise (HOME)

Description: 'HOME' is an accepted technique for intercepting and managing demonstrably unsuitable behavior that cannot be modified by basic behavior management techniques. Its intent is to help the hysterical/obstreperous child regain the self control that predicts that communicative management will be effective.

This technique is specifically used to re-direct in appropriate behavior, reframe a previous request and re-establish effective communication. When indicated, a hand is gently placed over the child's mouth and behavioral expectations are calmly explained. Maintenance of patent airway is mandatory. Upon the child's demonstration of self control and more suitable behavior, the hand is removed and the child is given a positive reinforcement communicative management techniques should then be used to alleviate the child's underlying fear and anxiety.

The need to diagnose and treat as well as the safety of the patient, practitioner, and staff should be considered for the use of HOME.

The decision to use HOME must take into consideration:

1. Other alternative modalities
2. Patient's dental needs
3. The effect on quality of dental care
4. Patient's physical and emotional development.

Written informed consent from the parent/legal guardian must be obtained and documented in the patient record prior to the use of HOME.

The patient's record should contain:

1. Informed consent
2. Indication for use

Objectives:

1. To redirect the child's attention, enabling communication with the dentist so appropriate behavioral expectations can be explained
2. To extinguish excessive avoidance behavior and help the child regain self control
3. To ensure the child's safety in the delivery of quality dental treatment
4. To reduce the need for sedation or general anesthesia.

Indications:

1. A healthy child who is able to understand and co-operate, but who exhibits obstreperous or hysterical avoidance behaviors
2. When all other behavior management techniques have failed and HOME is the last resort.

Contraindications:

1. In children who, due to age, disability, medication or emotional immaturity are unable to verbally communicate, understand and co-operate.
2. Any child with an airway obstruction.

Medical Immobilization/ Restraints

Description: Partial or complete immobilization of patient is sometimes is necessary to protect the patient, practitioner, and/or the dental staff from injury while providing dental care. It can be performed by the dentist, staff, parent/guardian with or without the aid of immobilization/restraining devices.

The need to diagnose and treat as well to protect the safety of patient, practitioner and staff should be considered for use of immobilization. Commonly used immobilization devises are:

For body	–	Pedi wrap
		Pappose board
		Sheets
		Bean bag with straps
		Towel and tapes, etc
For extremities	–	Velcro straps
		Posey straps
		Towel and tape
For Head	–	Head positioner
		Forearm body support
For Mouth	–	Mouth props
		Mouth blocks

Medical immobilization performed by a dentist/dental staff with/without an immobilization device must obtain

and document in the patient's record written informed consent from the parent/ guardian. The patient's record should include.

1. Informed consent
2. Type of immobilization used
3. Indication for immobilization
4. Duration of application.

Objectives:

1. To reduce or eliminate untoward movement
2. To protect patient and dental staff from injury
3. To facilitate delivery of quality dental treatment.

Indications:

1. A patient who requires immediate diagnosis and/or limited treatment and cannot co-operate due to lack of maturity/mental /physical disability.
2. When the safety of the patient and/or practitioner would be at risk without the protective use of immobilization.

Contraindications:

1. Co-operative patient
2. A patient who cannot be immobilized safely due to associated medical conditions.

Sedation

Description: Sedation can be used safely and effectively with patients unable to receive dental care for reasons of age or mental, physical or medical conditions. Written informed consent must be obtained from the parent and documented prior to the use of sedation.

Indications:

1. Fearful, anxious patients in which basic behavior management has not been successful
2. Patients who cannot co-operate due to lack of physical/emotion maturity and/or mental, physical or medical disability
3. Patients for whom use of sedation may protect them from developing psyche/and/or reduce medical risk.

General Anesthesia

Description: General anesthesia is a controlled state of unconsciousness accompanied by a loss of protective reflexes, including the ability to maintain an airway independently and respond purposefully to physical stimulation or verbal command. The use of general anesthesia sometimes is necessary to provide quality dental care for the child depending upon the patient; this can be done in a hospital or ambulatory setting.

Objectives:

1. To provide safe, efficient and effective dental care
2. To eliminate anxiety in child patients
3. To reduce untoward movement and reaction to dental treatment
4. To aid in treatment of the mentally, physically or medically compromised patient
5. To eliminate the child's pain response.

Indications:

1. Patients who are unable to co-operate due to lack of psychological or emotional maturity, and/or mental, physical or medical disability
2. Patients for whom local anesthesia is ineffective because of acute infection, anatomic variations, or allergy
3. Extremely unco-operative, fearful, anxious or uncommunicative child or adolescent
4. Patient requiring significant surgical procedures
5. Patients for whom the use of general anesthesia may protect the developing psyche and/or reduce medical risks
6. Patients requiring immediate, comprehensive oral/ dental care.

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Pediatric Pharmacology

INTRODUCTION

The term pharmacology refers to the science of drugs. In a broad sense, it deals with interaction of exogenously administered chemical molecules with living systems. There is great significance of this science when it comes to treat pediatric patients due to the fact that "infants and children are not small adults". They have significant physiological differences.

There are various differences between an adult and child pharmacology in aspects of the following:

PHYSIOLOGIC MECHANISMS

- Glomerular filtration rate (GFR)
- Tubular transport.
- Hepatic drug mobilization system.
- Blood-brain barrier permeability
- Drug absorption rate.
- Growth rate, etc.

PHYSICAL FACTORS

- Body surface area.
- Weight
- Age, etc.

These differences are explained through appropriate examples as follows:

- The newborn has low GFR and tubular transport is immature, thereby drugs have prolonged t1/2 life in newborn, GFR reaches adult rates by 5th month of age and tubular secretion takes about 7 months to nature.
- Hepatic drug metabolizing system is inadequate in newborns and blood-brain barrier is more permeable.
- Drug absorption may be altered in infants because of lower gastric acidity and slower intestinal transit.
- Transdermal absorption is faster as their skin is thin and more permeable.
- After the first year of life, drug metabolism is often faster than in adults.

Hence these factors necessitate the formulation of appropriate drug dosage for its use in children and drug dosage formulas have been devised based upon various criterions such as:

- Age
- Body weight
- Body surface area, etc.

AGE AS A CRITERION

The dose of a drug for children is often calculated from the adult dose. According to young's formula:

Child dose=(Age/Age+12) × Average adult dose

According to Dilling's formula

Child dose = (Age/20) × Average adult dose

BODY SIZE/BODY WEIGHT

Drug dosage can also be more accurately calculated on 'Body weight' or "Body surface area" basis, and for many drugs, manufacturers give dosage recommendations on mg/kg basis.

Based upon child's body weight, drug dosage is calculated by:

Clark's Rule

Child dose is 1/50th of adult dose per pound body weight or 1/70th of the adult dose per kilogram.

Child dose = (body weight in kilograms/70) × Average adult dose

Child dose= (body weight in pounds)/150) × Average adult dose

It has been argued that body surface area provides a more accurate basis for dose calculation, because total body water, extracellular fluid volume and metabolic activity are better parallel by BSA.

Since both age and weight rules underestimate the dosage for children. Total body surface area provides a better index of needful drugs and nutrients.

BSA of an Individual can be Calculated from Dubois Formula

BSA (m^2)= BW (kg))$^{0.425}$ × Height (cm)$^{0.725}$ × 00.7184

Individual dose = (BSA(m^2)/1.7) × Average adult dose

Table 12.1 gives average figures of body weight, body surface area and percentage of adult dose in children according to age.

Table 12.1: Average figures for children

Age	*Ideal body weight (kg)*	*BSA (m^2)*	*Percentage of adult dose*
Newborn	3.2	0.23	12.5
1 month	4.0	0.26	15
3 months	5.5	0.32	18
6 months	7.5	0.40	22
1 year	10	0.47	25
3 years	14	0.62	33
5 years	18	0.73	40
7 years	23	0.88	50
12 years	37	1.25	75

Other methods of calculating drug dosage in children are:

Augsburg Formula

0.7 × wt of child (pounds) = % of drug dosages.

Fred's Formula

(Age of child/150) × adult dose

Table 12.2 shows commonly prescribed drugs and their generic/trade names.

Table 12.2: Commonly prescribed drugs and their trade/generic names

	Formulation	*Generic name*
		Antibiotics
	Amikacin	Amicin, Amikacin, Amitax
	Gentamycin	Genticyn, Gentamycin, G-mycin-pedo
	Cephalexin	Sporidex, Solexin-Kid, Cephaxin, Cetalin, Cefalex, Nufex-kid, Cefalex-Pdrops
	Cephadroxyl	Cedil, Cefadrol, Cefadron, Cefadur, BID, Codroxil
	Cephazolin	Azolin, Cezolin, Crizolin, Orizolin
	Clindamycin	Clinicin, Dalacin, Dalcap
	Erythromycin	Erythrocin, Eryc-s, Erythromycin-kid(tab), Althrocin
	Roxithromycin	Roxisara, Tefrox, Bidrox, Roxid, Trony-kid, DeRox kid
	Azithromycin	Azisara, Azithral, Azid
	Clarithromycin	Celex, Claribid, Clarithro
	Penicillin	Bistrepin, Penidure
	Cloxacillin	Bioclox, Clocilin, Klox, Cloxacillin
	Ampicillin	Ampicillin, Amp-kid, Ampilac
	Ampicillin + probenicide	Ampilong
	Ampicillin + cloxacillin	Ampiclox-kid, Amplus-kid, Bluclox
	Ampicillin + sublactum	Ampitum, Sulbacin
	Amoxicillin	Novamox, Amotid, Imox, Pressmox, Symoxyl lb
	Amoxicillin+ clavulanate potassium	Augemntin, Clamp-Kid
		Antifungal drugs
	Clotrimoxazole	Candid, Surfaz
	Clotrimoxazole + corticosteroids	Clomax, Candid-b
	Nystatin	Mycostatin
	Miconazole	Candistat, Betnovate-C, Candizole
	Ketaconazole	Ketovate, Ocona
		Anti amoebic drugs
	Metronidazole	Flagyl, Metrogyl, Metron
	Tinidazole	Emidazol, Tina, Tizole, Fasigyn
		Sedative and Hypnotics
	Carbamazepine	Carmaz-kid, Tegretol, Zeptol, Carbazin
	Diazepam	Calmpose, Valium, Calmod
	Phenytoin sodium	Dilantin, Epsolin, Phetoin, Epileptin
		Analgesics
	Paracetamol	Dolo-650, Crocin(drops,syrup,tab), Cotamol, Metacin, Babygesic, Mol
	Ibuprofen	Brufen, Ibugesic, Emflam
	Ibuprofen + Paracetamol	Ibugesic-Kid, Ibugesic Plus, Ibuclin Jr, Combiflam, Anaflam, Brufamol-kid
	Mefanamic acid	Meftal, Ponstan
	Nimuselide	Nise, Nimulid-Kid, Nimulid-Plus, Nimugesic

ANTIBIOTIC PROPHYLAXIS

Taking antibiotics "before hand" is referred to as 'Antibiotic prophylaxis'. There exist various medical conditions that require patients to take antibiotics before dental treatment to ensure proper safety from post-treatment infections and other risks.

Guidelines for Antibiotic Prophylaxis for Patients at Risk

Numerous medical conditions predispose patients to bacteremia induced infections. These patients should establish optimal oral health to reduce the likelihood of bacteremia even in the absence of dental procedures. Because it is most possible to predict when a susceptible patient will develop an infection, prophylactic antibiotics are recommended when these patients undergo procedure most likely to produce bacteremia.

- An effective regimen should be directed against the most likely infecting organism.
- Antibiotics should be administered shortly before the procedures.
- When procedure involves infected tissues, additional doses may be needed.
- Appropriateness of antibiotic prophylaxis should be decided on an individual basis.

Patients who are at risk are categorized as follows for antibiotic prophylaxis.

Patients with Cardiac Conditions Associated with Endocarditis

The American Academy of Pediatric Dentistry endorses the American Heart Associations guidelines for prevention of bacterial endocarditis (Table 12.3).

In addition to those diagnoses listed in AHA guidelines, patients with a history of intravenous drug abuse and curtain syndromes (e.g. Down syndrome, Marfan syndrome) may be at risk for developing bacterial endocarditis due to associated cardiac anomalies.

Dental procedures that require and do not required endocarditis prophylaxis are mentioned in Table 12.4.

Recommended antibiotic prophylaxis regime for endocarditis is formulated in Table 12.5.

Patients with Compromised Immunity

Patients with compromised immune system may not be able to tolerate a transient bacteremia following invasive

Table 12.3: Cardiac conditions associated with endocarditis

Endocarditis prophylaxis recommended
High-risk category
Prosthetic cardiac valves, including bioprosthetic and homograft valves
Previous bacterial endocarditis
Complex cyanotic congenital heart disease (e.g. single ventricle states, transportation of the great Arteries, Tetralogy of Fallot
Surgically constructed systemic pulmonary shunts or conduits
Moderate risk category
Most other congenital cardiac malformations (other than above and below)
Acquired valvar dysfunction (e.g. rheumatic heart disease)
Hypertrophic cardiomyopathy
Mitral valve prolapse with valvar regurgitation and/or thickened leaflets
Endocarditis prophylaxis not recommended
Negligible risk category (no greater risk than the general population)
Isolated secundum atrial septal defects
Surgical repair of atrial septal defects, ventricular septal defects or patent ductus arteriosus (without residua beyond 6 months)
Previous coronary artery bypass graft surgery
Mitral valve prolapse without valvar regurgitation
Physiologic, functional or innocent heart murmurs
Previous Kawasaki disease without valvar dysfunction
Previous rheumatic fever without valvar dysfunction
Cardiac pacemakers (intravascular and epicardial) and implanted defibrillators

Table 12.4: Dental procedures and endocarditis prophylaxis

Endocarditis prophylaxis recommended
Dental extractions
Periodontal procedures including surgery, scaling and root planing, probing and recall maintenance
Dental implant placement and reimplantation of avulsed teeth
Endodontic(root canal) instrumentation or surgery only beyond the apex
Sub gingival placement of antibiotic fibers
Initial placement of orthodontic bands but not brackets
Intraligamentary local anesthetic injections
Prophylactic cleaning of teeth or implants where bleeding is anticipated
Endocarditis prophylaxis not recommended
Restorative dentistry (operative and prosthodontic) with or without retraction cord
Local anesthetic injections
Intracanal endodontic treatment; post placement and build up
Placement of rubber dam
Postoperative suture removal
Placement of removable Prosthodontic/orthodontic appliances
Taking of oral impressions
Fluoride treatments
Taking oral radiographs
Orthodontic appliance adjustments
Shedding of primary teeth

Table 12.5: Antibiotic prophylaxis for endocarditis in a pediatric patient

For dental/oral/upper respiratory tract procedures	
Standard regimen for patients at risk	
Amoxicillin	3 g orally 1 hour before procedure 1.5 g orally 6 hours after the initial dose
For penicillin allergic patients	
Erythromycin ethylsuccinate	800 mg 2 hours before procedure 400 mg 6 hours after initial dose
Erythromycin stearate	1g 2 hours before the procedure 500 mg 6 hours after the initial dose
Ampicillin	2 g IV/IM 30 minutes before procedure 1 g IV/IM 6 hours after the initial dose
Clindamycin	300 mg IV 30 minutes before the procedure 150 mg IV 6 hours after the initial dose
Initial pediatric dosages	
Amoxicillin	50 mg/kg
Clindamycin	10 mg/kg
Erythromycin	20 mg/kg
Vancomycin	20 mg/kg
Ampicillin	50 mg/kg
Gentamycin	2.0 mg/kg
Follow-up dose should be one-half the initial dose and the total pediatric dose should not exceed the adult dose	

dental procedures. The category includes, but is not limited to, patients with the following conditions:

1. HIV
2. Severe combined immunodeficiency
3. Neutropenia
4. Sickle cell anemia
5. Chronic steroid usage
6. Status post-splenectomy
7. Status post-organ transplantation
8. Diabetes
9. Lupus erythematosus, etc.

Patients with Shunts, Indwelling Vascular Catheters or Medical Devices, etc.

Patients with shunts and indwelling catheters are at a greater risk for bacteremia induced infections along with risk for bacterial endocarditism.

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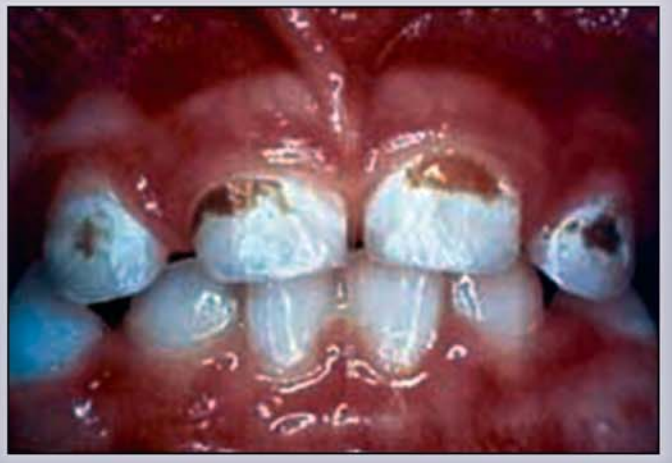


Introduction to Dental Caries

INTRODUCTION

Dental caries is one of the most prevalent diseases affecting the human race. It is often considered a disease of modern times due to its increasing risk of incidence since generations.

The word 'caries' is derived from the Latin word meaning 'rot' or 'decay'. In Greek, it means 'ker' for 'death'.

Dental caries is an infectious microbiological disease of the teeth that results in localized dissolution and destruction of calcified tissues of the teeth.

ETIOLOGY OF CARIES

There are various theories postulated regarding the mechanism of dental caries. The most accepted theories being:

1. The proteolysis theory.
2. Proteolysis chelation theory.
3. Acidogenic theory.

Proteolysis Theory

It was proposed by Gottlieb in 1934. According to this theory, the organic matrix is attacked before the mineral phase of enamel. The Proteolytic enzymes liberated by the oral bacteria destroy the organic matrix of enamel, loosening the apatite crystal, so they are eventually lost and tissue collapses.

Proteolysis Chelation Theory

This theory propounded by Schatz and Martin in 1955 postulates that the bacterial products attack the organic components of tooth structure and that the breakdown products have chelating ability and thus dissolve the tooth minerals.

Miller's Chemicoparasitic or Acidogenic Theory

This theory was proposed by WD Miller in 1890. According to this theory, the microorganisms found in the oral cavity procedure enzymes that act upon fermentable carbohydrates to produce acids which act upon the teeth causing decalcification of the inorganic portion and disintegration of the organic substance of the tooth leading to dental caries.

Other theories postulated on mechanism of caries are:

- Sulfatase theory
- Burch and Jackson hypothesis

Caries is a multifactorial disease. Various factors govern its formation, progression and arrest. There are various concepts that provide information on multi-factorial nature of caries, they are summarized as follows:

KEYE'S TRIAD (FIG. 13.1)

There is interplay of three principal factors in caries process. They are:

- The Host
- Micro flora
- Substrate

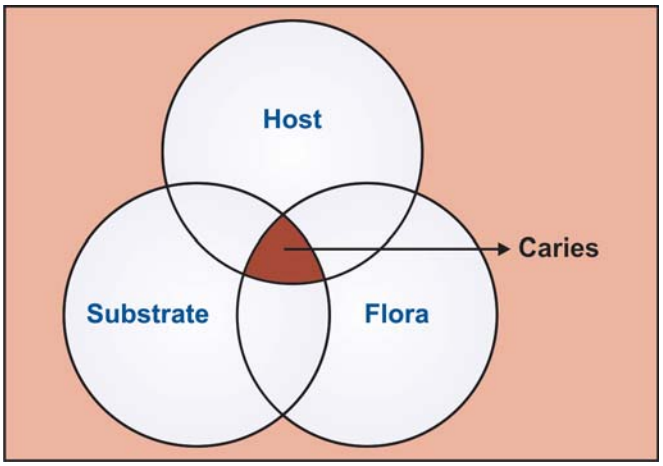


Fig. 13.1 Keye's triad

Modification of Keye's Triad (Fig. 13.2)

Newbrun in 1982 included a fourth factor, time to the still existing concept of Keye's depicting the significance of changes taking place over a period. This new concept is termed as **"Caries Tetralogy"** (Fig. 13.3).

Non-exclusive Contributory Disease Model (Fig. 13.4)

It explains the factors involved in dental caries. It includes elements "environment", "genetic" and "infectious agent".

Latest Concept

Apart from elements in caries tetralogy, "stress" is also

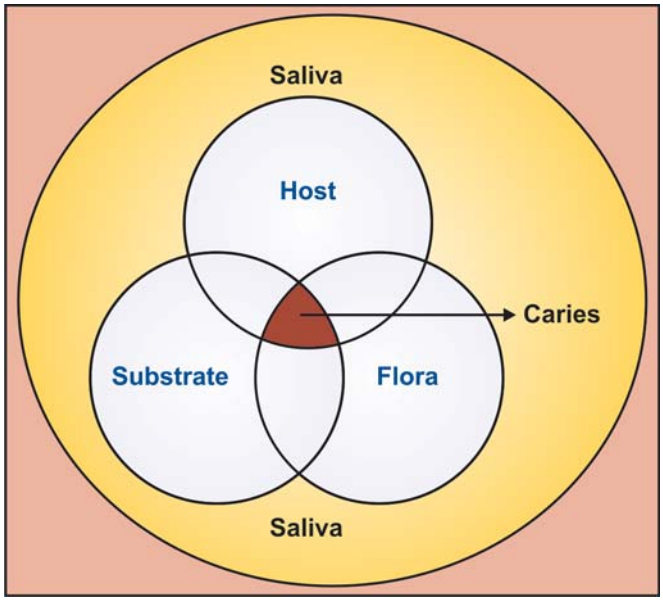


Fig. 13.2: Modification of Keye's triad

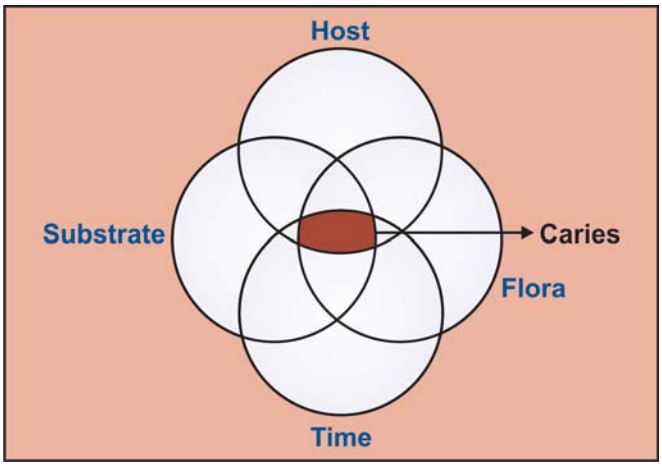


Fig. 13.3: Caries tetralogy (Newbrun)

considered to be another element considered to play a vital role in caries process.

ROLE OF EACH FACTOR IN CARIES

Host Factors

The host factors includes following variables that are responsible for dental caries. They are:

Tooth Factors

- Composition of tooth.
- Morphologic characteristics: The only morphologic feature which conceivably might predispose to caries

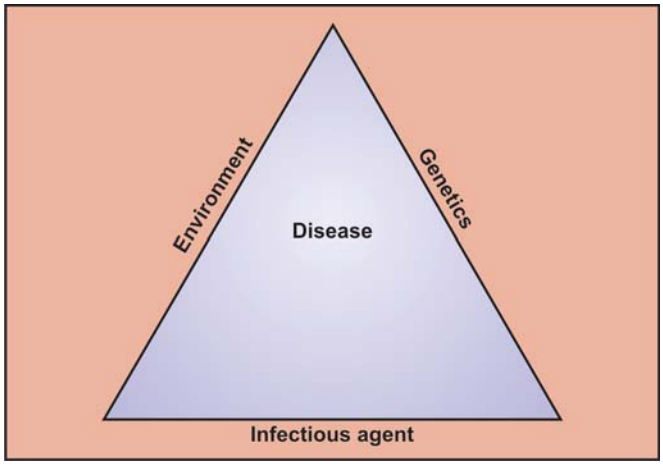


Fig. 13.4: Non-exclusive contributory disease model

is the presence of deep, narrow, occlusal fissures or buccal and lingual pits.
- *Tooth position:* Malaligned, out of position, rotated teeth may be difficult to cleanse and tends to favor food accumulation thus increasing susceptibility to caries.

Saliva

Composition of saliva: Salivary constituents related to dental caries:

- Calcium and phosphorus
- Ammonia
- Urea
- Ptyalin and amylase.

pH of saliva: pH of saliva is dependent on its bicarbonate content. As the flow rate of saliva increases, the pH increases. Saliva may be slightly acidic as it is secreted at unstimulated flow rates but it may reach a pH of 7.8 at high flow rates.

Quantity of saliva: Normally 700-800 ml of saliva is secreted per day. The quantity of saliva secreted in a given period of time, may influence caries evidence. This is especially evident in cases of salivary gland aplasia and xerostomia in which salivary flow may be entirely lacking, with rampant dental caries, the typical result.

However, mild increase or decrease in flow is of little significance in Caries development.

Viscosity of saliva: It has been suggested that viscosity is of some significance in accounting for difference in caries activity tests between different people.

Antibacterial property: The salivary antibacterial substances or enzymes are:

- Lactoperoxidase
- Lysozyme
- Lactoferrin
- IgA
- Proline rich protein like mucins and glycoproteins
- Aromatic rich proteins, etc.

Other host factors include

- Sex, age, race, familial heredity, developmental disturbances, socioeconomic status, concomitant disease, oral hygiene habits, etc.

Microflora

A variety of microbial factors have been associated with caries activity. Various observations indicate a casual relationship between *Streptococcus mutans* and the development of early carious lesion of enamel. Lactobacilli, associated with dentinal caries and Actinomyces strains with root surface.

Role of Microorganisms in Caries

1. They are a pre-requisite for caries initiation.
2. A single type of microorganism is capable of inducing caries.
3. The ability to produce acid is a pre-requisite for caries induction, but not all acidogenic organisms are cariogenic.
4. Streptococcus strains produce extracellular dextrans or levans that are capable of caries induction.
5. Organisms vary greatly in their capacity to induce caries.

Role of Dental Plaque

Plaque is a soft, non-mineralized bacterial deposit which forms on teeth that are not adequately cleaned (Loe).

Dental plaque is a complex, metabolically interconnected, highly organized, bacterial ecosystem. It is a structure of great significance as a contributing factor to the initiation of carious lesion.

An important component of dental plaque is acquired pellicle, which forms just prior to or concomitantly with bacterial colonization and may facilitate plaque formation.

Properties of Cariogenic Plaque

If acid production form readily fermentable substrates by certain plaque bacteria are the mechanism involved in enamel destruction, during the formation of a carious lesion, the metabolism and microbial composition of plaque reflect these properties:

1. Higher rate of sucrose consumption
2. Increased synthesis of intracellular polysaccharides.
3. Increased lactic acid formation.
4. Production of extracellular polysaccharides is doubled.
5. Higher levels of *Streptococcus mutans.*
6. Lower levels of *Streptococcus sanguis* and actinomyces.

Another hypothesis relating the role of plaque and caries process is:

- Non-specific plaque hypothesis that states that all plaque is cariogenic
- Specific plaque hypothesis recognizes plaque as pathogenic only when signs of associated disease are present.

Environment Factor

Diet

Diet is defined as the types and amounts of food eaten daily by an individual. The role of diet and nutritional factors deserves special consideration because of the often observed differences in caries incidence of various populations who subsist on dissimilar diets.

Essentiality of oral substrate in caries process: The presence of readily fermentable sugars/carbohydrates is responsible for caries process. The sugars found in human diet support bacterial acid production and the colonization of teeth by cariogenic bacteria.

The cariogenicity of dietary carbohydrate varies with the frequency of ingestion, physical form, and chemical

composition, route of administration and presence of other food constituents.

Cariogenicity of diet depends upon:

- Frequency of ingestion
- Physical form of diet
- Chemical composition
- Amount of fermentable carbohydrates
- Retentiveness of the food substance after consumption and hygiene procedures, e.g. Sticky, greasy, sweet foods
- Type of food substances
- pH of the food substances.

Foods with low cariogenic potential includes:

- High protein content foods
- Foods with high fiber and minimal concentration of fermentable carbohydrates
- High mineral content
- Well cooked food
- Strong buffering capacity.

Need for nutrition education and diet counseling: Nutrition education for the purpose of reducing the caries incidence in children is aimed at teaching parents the importance of reducing dietary exposures to sweets, foods and hidden sugars.

Dietary counseling aims to help parents change their and their children's eating behaviors so that they choose diets with low cariogenic or non-cariogenic snacks, limit sweet foods to meal times and perform tooth brushing after sugar exposures.

Dietary recommendations must be realistic and based on the dietary behaviors of the family. It is pointless to prescribe changes that cannot or will not be implemented.

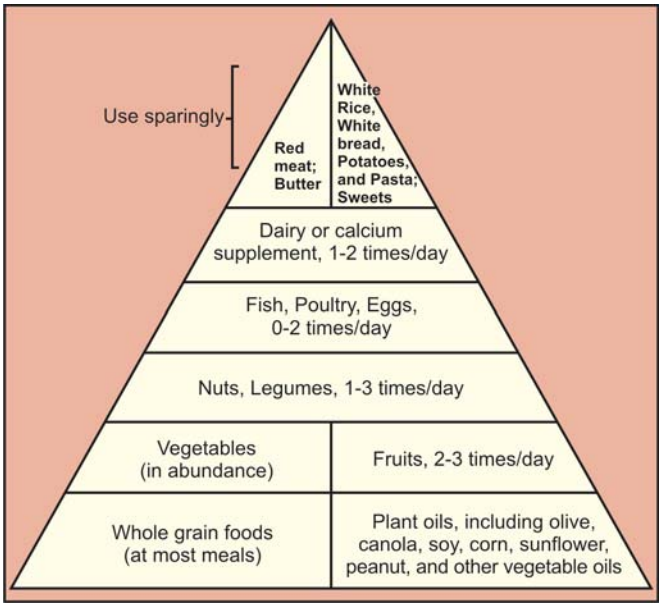


Fig. 13.5: Healthy eating pyramid for general nutrition

Additionally, modifications to the diet can only be made over time, aided by repetition and re-inforcement. The goal must be to help caregivers develop lifelong dietary habits that promote general and oral health for themselves and for those whom they influence.

Figure 13.5 depicts the healthy eating pyramid for general nutrition shows that sweets, as well as white carbohydrates and food with high cholesterol, should be eaten sparingly.

Table 13.1 shows children's foods and snacks with cariogenic potential.

Table 13.2 provides information of oral health dietary guidelines for expectant mothers and pre-school children.

Table 13.1: Cariogenic potential of children's food and snack

Non-cariogenic	*Low cariogenic*	*High cariogenic*
Cheeses	Fruits(except dried)	Candy**
Nuts*	Chocolate milk	Cookies
Dried meat sticks	Whole grain products	Cake
Plain milk		Sweetened beverages (including fruit juices)
Vegetables		Fruit roll-ups, dried fruit
Popcorn*		Breakfast bars
Flavored club soda		
Diet sodas		

**Not appropriate for infants and toddlers owing to potential for chocking
*Sticky or slowly eaten candy is extremely cariogenic

Table 13.2: Oral healthy dietary guidelines for expectant mothers and pre-school children

Dental period	*Nutrition*
Pregnant women	Follow the healthy eating pyramid, taking into account increased needs for pregnancy Take prenatal vitamin/mineral supplements as prescribed Limit intake of cariogenic foods, especially as between meal-snacks
Birth to 1 year	Avoid allowing the infant to sleep or nap with bottle Avoid excessive consumption of juice Eliminate dipping pacifiers in sweetened foods
1 to 2 years	Avoid frequent consumption of juice or other sugar containing drinks in bottle or sippy cup Encourage proper weaning Continue avoidance of bottle to bed Promote non-cariogenic foods for snacks Foster routine eating pattern and healthy eating pyramid
2 to 5 years	Discourage slowly eaten, sugar containing foods Promote non-cariogenic foods for snacks Encourage eating at meals and healthy eating pyramid

Time

The time of contact between all causative factors is most crucial factor. Greater the time taken for the food to be washed /cleansed from tooth surface by either saliva or oral hygiene measures, greater is the probability of action of oral microorganism thereby increasing the susceptibility to caries.

Stress

The recent element added to multifactorial caries process validation its inclusion due to following reasons:

- Stress is known to alter the eating habits of individuals.
- Changes in flow rate, viscosity and pH of Saliva are also increased.
- Psychological stress may increase gastrointestinal disorders leading to acidity and often acid regurgitation into mouth occurs thereby reducing pH of saliva in oral cavity and chemical erosion of tooth structures rendering them prone to action of cariogenic bacteria and enzymes.

Area of Caries Susceptibility

In primary dentition: In the primary dentition, the sequence of caries attack follows a specific pattern mandibular molars, maxillary molars and maxillary anterior teeth. Seldom are the mandibular anterior teeth or buccal and lingual surfaces of primary teeth involved, except in instances of rampant caries.

Table 13.3 shows surface by surface comparison of the teeth susceptible to caries.

Table 13.3: Surface by surface comparison of teeth susceptible to caries

Tooth	*Maxillary*	*Mandibular*
Second molar	Occlusal and lingual	Occlusal and buccal
First molar	Occlusal	Occlusal and buccal
Canine	Buccal	Buccal
Lateral	Mesial	Mesial
Central	Mesial	Mesial

In mixed dentition:

- First permanent molars are at high risk as soon as they erupt into the oral cavity.
- Distal surface of second primary molar is a common site for caries after eruption of the first permanent molar.
- At 8 years, approximately 1% of the maxillary permanent central and lateral incisors will be carious.

In permanent dentition:

- A sharp rise in the caries attack rate continues after eruption of second permanent molar and has a high occlusal surface attack rate of 20% when compared with maxillary molar with 10% susceptibility.
- The cervical portions of buccal grooves of the mandibular first and second molars and the lingual grooves of the maxillary first and second molars are also sites for morphologic defects and incomplete enamel formation.

Caries Risk Assessment (Tables 13.4 and 13.5)

Caries risk assessment is the determination of likelihood of the incidence of caries during a certain time period. It also involves the likelihood that there will be a change in size or activity of lesions already present. With the ability to detect caries in the earliest stages, health care

Table 13.4: Factors that cause high risk caries condition

Factor	*Action and mechanisms*	*Causes*
Amount of plaque (oral hygiene status)	Biofilm size determines the number of microorganisms colonizing the surface. The higher their number, the greater the amount of acid compounds produced by their metabolic activities	Lack of manual skill, interest or motivation in oral hygiene; physical or mental disabilities, stressful lifestyle; presence of crowns, bridges or filling with overhangs
Proportion of cariogenic bacteria in plaque	High proportions of cariogenic bacteria in plaque determine a high acidogenic activity and maintain a low plaque pH for prolonged periods	Genetic factors, diet with high and frequent carbohydrate intake, high retention site presence because of orthodontic appliances, fillings, or prostheses with marginal problems
Diet	High promotion of fermentable carbohydrates in diet, foods with high adhesive properties	Lack of correct dietary information, medical or social problems that make frequent or excessive carbohydrate consumption easy or necessary
Behavior	The frequent intake of carbohydrate rich foods beats the defense system of saliva and enables cariogenic flora to maintain a low pH on the dental surface	Lack of health education, stressful lifestyle, general health problems
Salivary flow	Reduction of salivary flow leads to an increase of clearance time for several substances, in particular carbohydrates. A reduced amount of saliva cannot was carbohydrates off the dental surface effectively	Systemic disease affecting salivary glands, prolonged pharmacologic treatments, genetic factors
Saliva buffer capacity	A reduced buffer capacity of saliva reduces the possibility of neutralizing acids produced in plaque by cariogenic flora	Systemic diseases affecting salivary glands, medications and genetic factors
Fluoride	Low fluoride concentration or fluoride absence reduces remineralization process	Geographic location with low fluoride concentration in drinking water, lack of fluoride toothpaste or use of other topical fluoride products

providers can help prevent cavitations. Strategies for managing caries increasingly have emphasized the concept of risk assessment.

Risk assessment is a necessary component in the clinical decision-making process. Caries risk indicators are variables that either currently are thought to cause the disease directly (e.g. Microflora) or have been shown useful in predicting it (e.g. Socioeconomic status).

These risk factors may vary with race, culture and ethnicity and may be useful in the clinical management of caries by helping to determine if additional diagnostic procedures are required, identity the subjects who require caries control measures, assess the impact of caries control measures, guide in treatment planning decisions and determine the timing of recall appointments.

Since, the etiology of caries is multifactorial, it has been suggested that risk assessment should be directed at the evaluation of all factors involved with the disease.

Risk assessment tools can aid in the identification of reliable predictors and allow health care professionals to become more actively involved in identifying and referring high risk children.

Table 13.6 incorporates available evidence into a concise, practical tool to assist both dental and non-dental health care providers in assessing levels of risk for caries development in infants, children and adolescents.

Fig. 13.5: Determination of caries risk

Factors		*High*	*Low*
Diet	Diet history	Diet high in fermentable carbohydrates	Diet low in fermentable carbohydrates
Frequency	Frequency of consumption with diet history	Frequent consumption not confined to meal times	Infrequent consumptions or confined to meal times
Plaque	Amount and nature	High plaque score	Low plaque scores
Saliva	Amount and nature	Low flow rates High lactobacilli and streptococcus counts	High flow rates low lactobacilli and streptococcus counts
Socioeconomic status		Not dentally motivated Deprived background; Low dental aspirations; high caries family	Dentally motivated patients; Privileged background; high dental aspirations; Low caries family
Past disease experience		High number of filled and missing surfaces	Low number of filled and missing surfaces
Current disease experience		High number of decayed surfaces	Low number of decayed surfaces
Attendance pattern		Irregular or pain only attenders	Regular attenders
Fluoride and chlorhexidine		Infrequent use of rinses and toothpaste; non-fluoridated water supply	Frequent use of rinses and toothpaste; fluoridated water supply
Medical history		Xerostomia; Learning difficulties; Cariogenic medication	Fit and well
Other		Partial dentures used to replace missing units	Bridgework used to replace missing units

CARIES ACTIVITY TESTS

Criteria for caries activity test:

1. The test should be valid, reproducible, simple and inexpensive.
2. The test should also be non-invasive, easy to evaluate and easy clinical application.
3. There should be minimal occurrence of false positive responses.

Advantages

1. Permits identification of risks status of individual.
2. Helps in devising preventive measures to reverse the disease process before it is too late.

Types

Common caries activity tests are summarized as follows:

1. Lactobacilli Colony Test

Lactobacilli count is performed by using serial dilutions of saliva, collected by chewing 1 gm paraffin wafer. A 1 ml aliquot for each dilution is placed in a series of Petri dish to which 10 ml of Ragosa's lactobacilli selective medium is added.

After incubating for 4 days number of colonies is counted. Count is scored from 1 to 4 depending on whether they fall within the ranges of 0 to 1000; 1000 to 10,000, 10,000 to 1, 00,000 or 1, 00,000 and above.

Table 13.6: Caries risk assessment tool

AMERICAN ACADEMY OF PEDIATRIC DENTISTRY CARIES-RISK ASSESSMENT			
Risk Factors to Consider (For each item below, circle the most accurate response found to the right under "Risk Indicators")	*Risk Indicators*		
	High	*Moderate*	*Low*
Part 1 – History (determined by interviewing the parent/primary care giver)			
Child has special health care needs	Yes		No
Child has condition that impairs salivary flow/composition	Yes		No
Child's use of dental home	None	Irregular	Regular
Time lapsed since child's last cavity	<12 months	12 to 24 months	>24 months
Child wears braces or orthodontic/oral appliances	Yes		No
Child's mother has active decay present	Yes		No
Socioeconomic status of child's care giver	Low	Mid-level	High
Frequency of exposure to between meal sugars/cariogenic foods (include ad lib use of bottle/sippy cup containing juice or carbonated beverage)	≥ 3	1 to 2	Meal time only
Child's exposure to fluoride	Does not use fluoridated toothpaste; drinking water is not fluoridated; not taking fluoride supplements	Uses fluoridated toothpaste; usually does not drink fluoridated water; does not take fluoride supplements	Uses fluoridated toothpaste; drinks fluoridated water or takes fluoride supplements
Part 2 – Clinical evaluation (determined by examining the child's mouth)			
Visible plaque on anterior teeth	Present		Absent
Gingivitis		Present	Absent
Areas of demineralization (white-spot lesions)	More than 1		Absent
Enamel characteristics: hypoplasia, defects, retentive pits/fissures	Present		Absent
Part 3 – Supplemental assessment (Optional)			
Radiographic enamel caries	Present		Absent
Levels of Mutans streptococci	High	Moderate	Low
*Based on AAPD Policy on Use of a Caries-risk Assessment Tool (CAT) for infants, children, and adolescents. Pediatric Dent 2004:26(7):25-27			
Each Child's overall assessed risk for developing decay is based on the highest level of risk indicator circled above (i.e. a single risk indicator in any area of the "high risk" category classifies a child as being "high risk")			

*American academy of pediatric dentistry caries–Risk assessment.

2. Alban's Test

The medium for test is prepared by adding 1 litre of boiling water into 61 gm Snyder's medium and adjusting pH with glacial acetic acid. Approximately 5 ml of medium is placed in sterile test tubes and refrigerated.

At the time of test, test tube is removed from refrigerator and patient is asked to spit unstimulated saliva into tube until a thin layer of saliva is covering the surface of green agar.

The tube is incubated for 4 days, with daily recordings is made to observe color changes. The changes are scored from 0 to 4 based on amount of color change from top bottom of the tube.

Scoring

0 - No color change
1+- Color change to yellow in top 1/4th of tube.
2+- Color change to yellow to ½ of tube
3+- Color change to yellow in 3/4th of tube
4+ - Color change to yellow in entire length of agar column.

3. Salivary Buffer Capacity Test.
4. Enamel Susceptibility Test.
5. Salivary Reductase Test.
6. *Streptococcus mutans* Level in Saliva.

Cariogram (Fig. 13.6)

Cariogram is a multifactorial risk assessment model for a multifactorial disease introduced by Bratthall et al in 1999. It is in the form of a pie circle diagram divided into color coded 5 sectors which are:

1. The dark blue sector 'Diet' is based on a combination of diet contents and diet frequency.
2. The red sector 'Bacteria' is based on a combination of amount of plaque and mutans streptococci.
3. The light blue sector 'susceptibility' is based on combination of fluoride program, saliva secretion and saliva buffer capacity.
4. The yellow sector 'circumstances' based on a combination of caries experience and related diseases.

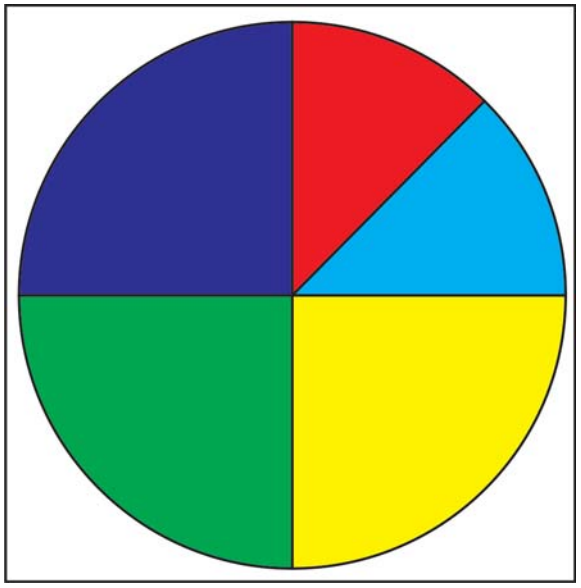


Fig. 13.6: Cariogram

5. The green sector shows an estimation of the 'chance of avoiding caries'.

Table 13.7 shows caries related factors and the data needed to create a cariogram.

CLASSIFICATION OF CARIES

Dental caries is classified into various types based upon various criterions. They are:

Based on the Location/Anatomic Site

Pit and Fissure Caries

- Pits and fissures that are deep, narrow and complex are more susceptible for retention of food debris and microorganisms and results ultimately in caries.

Smooth Surface Caries

- Develops on proximal and gingival third of buccal and lingual surfaces of teeth.
- Caries at these regions are preceded by dental plaque formation.

Root Caries

- Causes affecting radicular surfaces affecting cementum and dentine.
- Often sequelae to gingival recession followed by continuous plaque retention.

Based on the Severity of Lesion

Incipient Caries

- The earliest caries lesion seen on smooth surfaces of teeth as 'white spot'.
- Histologically the lesion has an apparent intact surface layer with subsurface demineralization.
- These lesions can undergo remineralization if intervened at right time.

Occult Caries

- These lesions are clinically not detectable except on radiographs.
- These lesions are not associated with microbial invasion and such lesions are usually seen in low

Table 13.7: Caries related factors and the data required to create a cariogram

Factors	*Comments*	*Information/data needed*
Caries experience	Past caries experience, including cavities, fillings and missing teeth because of caries. Several new cavities definitely appearing during preceding year should give a high score even if number of fillings is low	DMFT, DMFS, new caries experience in the past 1year
Related diseases	General disease or conditions associated with dental caries	Medical history, medications
Diet, contents	Estimation of the cariogenicity of the food, in particular sugar contents	Diet history, lactobacillus count
Diet, frequency	Estimation of number of meals and snacks per day, mean for 'normal days'	Questionnaire results, 24 hours recall or dietary recall (3 days)
Plaque amount	Estimation of oral hygiene. Crowded teeth leading to difficulties in removing plaque interproximally should be taken into account	Strip Mutans test or other laboratory tests giving comparable results
Mutans streptococci	Estimation of levels of Mutans streptococci in saliva	Fluoride exposure, interview patient
Fluoride program	Estimation of to what extent fluoride is available in the oral cavity over the coming period	Stimulated saliva test-secretion rate
Saliva secretion	Estimation of amount of saliva	Dentobuff test or other laboratory tests giving comparable results
Saliva buffer capacity	Estimation of capacity of saliva to buffer acids	

caries rate individuals with increased fluoride exposure.

- Such lesions do not need restorative treatment.

Cavitation Lesions

- As the caries progression reaches, the dentino enamel junction, it spreads to pulp and laterally reading to demineralization and dissolution of inorganic and organic matrix, leading to cavitation in the tooth surface.
- Both clinically and radiographically.

Based on Progression

Arrested Caries

- With changes in Oral conditions towards a healthy state; certain carious lesions ceases to progress and show signs/attempts of remineralization, such lesions are termed as arrested caries and not lesions.

Secondary/Recurrent Caries

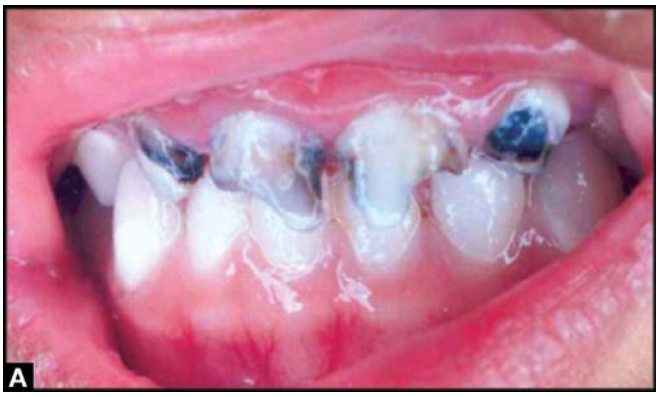
- Caries process that occurs along a restorative-tooth interface due to failure of restoration or other factors is called as 'secondary caries'.

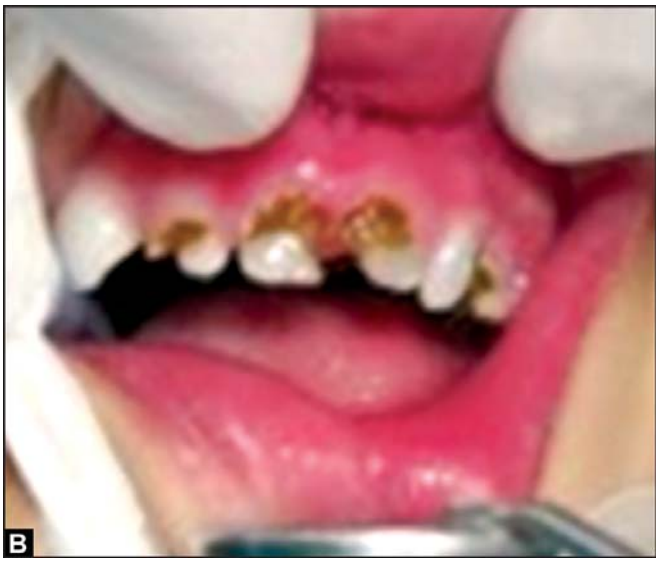
Based on Chronology

1. Early childhood caries
   - Nursing bottle caries
   - Rampant caries
2. Teenage/adolescent caries
3. Adult caries
   - Root caries, etc.

Rampant Caries (Figs 13.7A and B)

Massler (1945) defines it as "suddenly appearing, widespread, rapidly burrowing type of caries, resulting in early involvement of pulp and affecting those teeth usually regarded as immune to ordinary decay."





Figs 13.7A and B: Rampant caries

Winter's (1966) definition states "caries of acute onset involving many or all of the teeth in areas that are usually not susceptible and are associated with rapid destruction of the crown with frequent involvement of dental pulp".

Etiology:

- Poor oral hygiene.
- Primary and secondary nutritional inadequacies.
- Stress, emotional disturbances and anxiety which may initiate an unusual craving for sweets or the habit of snacking, which in turn might influence the incidence of caries.
- Decreased salivary flow and decreased caries resistance are also etiological factors.

Characteristic features: Davies believes that the distinguishing characteristics of rampant caries are the involvement of proximal surfaces of lower anterior teeth and the development of cervical type of caries.

Though it is considered that young teenagers are particularly susceptible; both children and adults of all ages are also affected by rampant caries.

Management: A comprehensive management of rampant caries is targeted at following steps:

Control of active caries lesion:

- By removal of caries.
- Temporization/Pulpal therapy.

Diet assessment and Nutrition education and counseling with patient and parent.

Oral hygiene instructions:

- Professional care
- Home care

Nursing Bottle Caries (Fig. 13.8)

Kroll (1967) defines as "a syndrome characterized by a severe caries pattern beginning with the maxillary anterior teeth in a healthy bottle fed infant or a toddler."

In recent years, it has been recognized that prolonged/improper bottle feeding, beyond the usual time when the child is weaned from the bottle and introduced to solid food, may result in early rampant caries.

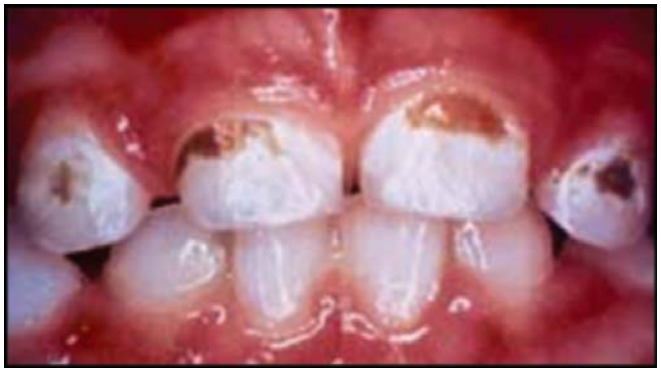


Fig. 13.8: Nursing bottle caries

Etiology:

- Prolonged bottle feeding
- Putting baby to sleep with bottle in mouth
- Additional sweeteners

- Sweetened/Honey dipped pacifiers.
- Poor oral hygiene maintenance.
- Increased lactose content in bovine milk and breast milk.

Clinical features: The clinical appearance of teeth in "nursing bottle caries" in a child 2, 3 or 4 years of age is typical and follows a definite pattern.

It is characterized by early carious involvement of maxillary anterior teeth, the maxillary and mandibular first primary molars and mandibular canines. The mandibular incisors are usually unaffected.

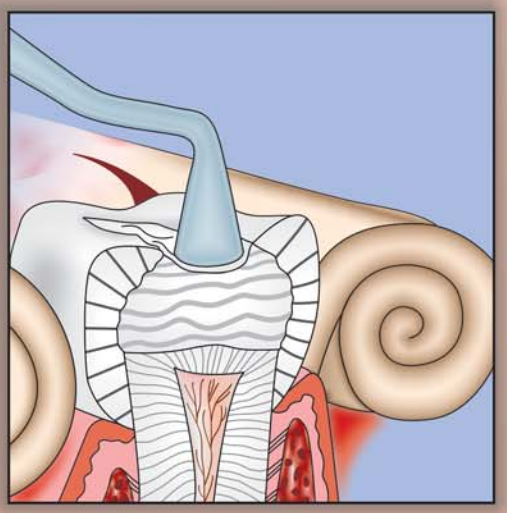
Management:

- Advice parents on nutrition, methods of breast/bottle feeding and weaning.
- Oral hygiene instructions for infants and toddlers.
- Restoration of established lesions at the earliest.

Treatment Protocol for Rampant Caries/Nursing Caries

1. **Preventive therapy**
2. **Professional care:**
   - Nutrition education and dietary assessment and counseling for both parents and patient
   - Caries risk assessment
   - Fluoride supplementation
   - Occlusal sealant therapy
   - Planning regular recall visits
   - Reinforcing and motivation home care.
3. **Home care**
   - Appropriate balance diet; eating habits, frequency and needed nutritional supplementation.
   - Oral hygiene care provided by parents to infants, toddlers and all age groups like
     — Brushing/cleaning gumpads.
     — Flossing
   - Mouth rinsing, etc.
   - Frequent dental check ups.
4. **Regeneration:** Regeneration/remineralization of incipient carious lesion by:
   - Fluoride therapy
   - Products like tooth moose
5. **Restorative**
   - Fissure sealants
   - Preventive resin restoration
   - Appropriate restorations with
     — Glass ionomer
     — Composites
     — Amalgam
   - Stainless steel crowns, etc.
   - Necessary pulpal therapy and temporization as needed in the case.

14



Pediatric Restorative Dentistry

INTRODUCTION

"Prevention is better than cure", but when the situation gets beyond this level, the main concern is directed towards controlling or eliminating the cause to rehabilitate.

Pediatric operative dentistry aims to eliminate the established/establishing carious lesion affecting primary and young permanent teeth and restore the lost tooth structure to maintain its function and integrity; so as to prevent the loss of teeth and development of related subsequent problems.

STRUCTURAL DIFFERENCES BETWEEN DECIDUOUS AND PERMANENT TEETH

The structural differences between primary and permanent teeth is the key factor governing the principles of cavity preparation; design and choice of restorative materials used in pediatric restorative dentistry. They are as follows:

1. Shorter crowns:
   - Restricts the depth of cavity preparation due to proximity of pulp chamber and pulp horns.
2. Narrow occlusal surface
   - Reduced width of the cavity than in permanent teeth.
3. Cervical constriction
   - Probability of pulp exposure is greater due to cervical construction during proximal cavity preparation if depth of cavity is increased.
4. Reduced thickness of enamel and dentine than in permanent teeth.
5. Occlusal orientation of enamel rods cervically essentiates not to provide beveling at the gingivo-cavo surface line angle as it may lead to many unsupported enamel rods that may fracture under stress of a restoration.
6. Broad and flat contact areas suggest the proximal preparations to be wider than in permanent teeth.
7. Histological differences in enamel and dentine composition necessitate more etching time (especially in fluoride treated teeth) in use of composite resins.

AIDS IN CARIES DIAGNOSIS

The boundaries of caries diagnosis and caries intervention are changing. As with any disease process the early initiation of therapy for dental caries is often the most effective means to ensure resolution. However, for any therapy to be effective, early diagnosis is paramount to success. Unfortunately, current tools used in dental caries detection are not sensitive enough to diagnose the disease process in its early stages and frequently once a diagnosis is made, restoration is the only effective means of treatment.

Diagnosis of dental caries is often regarded as synonymous with detection of the clinical signs of the tissue damage caused by the disease, i.e. carious lesions and cavities.

The early detection of caries is further complicated by the overall restraints of the oral cavity. Changes of curvature of the tooth surfaces, the various compositional profiles within the same tooth and between teeth, also make matters more difficult. Diagnosis of occlusal decay in particular has proved to be an arduous task. This is because of increase in fluoridated dentifrice usage resulting in the surface enamel remaining intact overlying a slowly progressing "occult" carious lesion. There is a need to develop better diagnostic methods for occlusal caries.

The use of appropriate diagnosis methods must be made according to the situation, for example:

- Use in clinical situation
- Laboratory studies and research work
- Community studies.

The need for each of these will vary; hence, different approaches are required. The search for an ideal caries diagnostic test continues. Such a test must be accurate, sensitive, specific, reproducible, and reliable, not transfer *S. mutans* or other bacteria from affected area to an unaffected area and also cost effective.

Methods Available for the Clinical Diagnosis of Dental Caries

Clinical Method/Visual-Tactile (Table 14.1)

Method: This method is a combination of light, mirror, and the probe for detailed examination of every tooth

Table 14.1: Detailed criteria for visual inspection of occlusal surfaces introduced by Ekstrand et al

Classification	*Visual inspection*
0	No or slight change in enamel translucency after prolonged air drying (>5 seconds)
1	Opacity or discoloration hardly visible on wet surface, but distinctly visible after air drying
2	Opacity or discoloration distinctly visible without air drying
3	Localized enamel breakdown in opaque or discolored enamel and/or grayish discoloration from the underlying dentin
4	Cavitations in opaque or discolored enamel exposing the dentin

surface. It is by far the most commonly applied method in general practice worldwide. Greene Verdman Black, the father of conservative dentistry in 1924, suggested the use of a sharp explore from the tooth surface, i.e. if there was a "catch" then the surface was counted as being decayed, a concept supported by other authors also. Today, this concept has been challenged. It has been proved that the explorer point may fracture the demineralized enamel leading to cavitation. If left alone such a lesion could have reverted back to normal.

The use of a mirror and a sharp probe is the most common method of diagnosing tooth decay, but it could not be notoriously unreliable, as the sticky fissures may not be carious. Recently, the sensitivity of probing in a standardized system was found to be only 17%. Because of this a mirror and a blunt probe is used to remove plaque and deposits from the fissure and then visual examination of the fissure for decay is carried out.

Use of visual examination only, is known as the European method, while use of sharp or blunt probe in visual tactile system, is popularly known as the American System, for diagnosis of dental caries. Visual diagnosis is being increasingly relied upon today, because of the following reasons:

- Probing can irreversibly traumatize potentially remineralizable non-cavitated lesions of the enamel.
- The probe might be causative in transportation of decay causing bacteria from place to place.
- Probing may provide no more accuracy in diagnosis than visual inspection alone.

An examiner detects caries by observing the change in translucency of enamel. This is done by observing a clean, dry, and well-illuminated field. Magnifying glasses are sometimes used to aid in the diagnosis and have been shown to modify treatment planning.

Radiographic Method

Earlier, bitewing radiographs were used for diagnosis of proximal decay because caries tends to occur most frequently just below the contact point either mesially or distally. Today bitewing radiographs are in use for detection of hidden occlusal dentine lesions. The use of radiographs must interpreted with caution as it presents a two dimensional picture of a three dimensional object. Another aspect is that net mineral loss must exceed at least 20 to 30% in order to radiographically visible. It is also one of the most inevitable diagnostic aids in diagnosis of caries in restored teeth and commonly used method as an aid to the diagnosis and the subsequent treatment of caries.

It makes it possible to study parts of the teeth inaccessible to other diagnostic methods.

The depth of a lesion can be evaluated and hence, the relation between the lesion and the pulp of the tooth.

Radiograph is a non-invasive method while probing may cause a break of the enamel covering a subsurface lesion.

The radiograph provides a lasting documentation and repeated examinations allow an evaluation of disease activity and consequently the efficacy of therapeutic measures.

Tooth Separation

Separating the teeth for visualizing the posterior proximal surfaces has been known since the last century and is now regaining popularity. This method uses orthodontic modules or bands and achieves slow separation. Taking impressions of the proximal surfaces thus separated have been used to assist in the detection of cavitations.

Advantages

- Permits the differentiation of cavitated and non-cavitated lesions.

- Permits an assessment of the buccolingual extent of the lesion.
- Noninvasive reversible method.
- It is well tolerated, quick, effective and inexpensive.
- Versatile, can be used for anterior or posterior teeth in adults as well as children.

Disadvantages

- Requires an additional visit.
- Occasional discomfort.
- Occasional failure of separation.
- Potential danger of ingestion/inhalation.
- Potential exacerbation of gingival inflammation.

Fiber Optic Transillumination (FOTI)

- A qualitative method used since 1970s in which while light is from a cold-light source is passed through a fiber to an intra-oral fiber-optic light probe that is placed on the buccal or lingual side of the tooth.
- The surface is examined using the transmitted light, seen from the occlusal view.
- Demineralized area appear darker compared to surrounding sound tissue. This contrast between the sound and carious tissue is used for detection of lesions.

Dyes in Caries Detection (Fig. 14.1)

Dyes have widespread use in medicine, biology and dentistry. If an object is hard to distinguish from its background, the color induced by a dye can make it easier to visualize or, if several objects have a similar appearance, coloring by a dye may discriminate between them and allow identification. The property of dyes to enhance contrast, by their color can be used in clinical dentistry and in investigations *in vitro* or *in vivo*. They have been used for:

- Indication of affected dental tissues
- Improvement of diagnostic methods
- Enhancement of patient awareness
- Information about specific processes.

Clinically used dyes are often visually observed, which means a qualitative assessment of the staining, while quantification of the staining, if performed at all, is confined mostly to laboratory experiments. Various dyes such as silver nitrate, methyl red, and Alizarin stain have been used to detect carious sites by change of color. The difficulty lies in removing the dye from the altered enamel areas. The altered areas of enamel are characterized by more reactive calcium that reacts with carboxylic and sulfuric acid groups of the dyes.

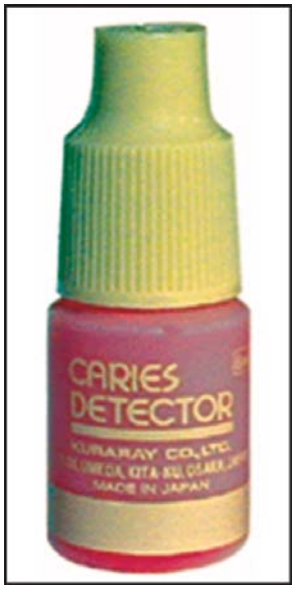


Fig. 14.1: Caries detector

Digital Radiographic Methods

A digital image is an image for images, which are the Digital Image Receptor (DIR) and Video Camera for indirect digital imaging.

Digital, filmless techniques for Intraoral radiography have been developed for several reasons,

- Conventional film absorbs only a few percent of the X-rays that reach it, utilizing very little of the radiation to which patient has been exposed.
- Poor dark room procedure can lead to both unnecessarily high doses of radiation and loss of diagnostic information.
- Development of films is time consuming and the developing and fixing solutions are hazardous to the environment.
- Can be easily tele transmitted.
- Consistent image quality.

When viewed from a distance, the image appears continuous, but closer view reveals individual pixels. With the advent of digital radiography, following of lesion progression and reversal with time is now possible.

Electrical Resistance Measurements (ERM)

The idea of an electrical method of caries detection dates back to 1878, when it was believed to have been first proposed by Magitot. It is based on the differences in the electrical conductance of carious and sound enamel. Two instruments were developed and tested in the 1980's, the Vanguard Electronic Caries Detector and the Caries Meter-L.

Because intact enamel is believed to be an electrical insulator, carious lesions reaching the dentin might be detected by measuring the electrical resistance of the small areas of the occlusal surface. The results obtained by electrical resistance measurements indicate that this technique can be valuable aid in the diagnosis of occlusal carious lesions, which are imperceptible to the unaided eye.

Light-Induced Fluorescence (Lasers in Caries Detection)

Fluorescence is a well known phenomenon in science and technology. In simple terms light at one wavelength (excitation wavelength) is absorbed by the tissue and emitted at a second longer wavelength (emission wavelength). The phenomenon occurs only when there is a specific substance that is excited by a specific wavelength of light. Visible light within the blue-green region has been used as the light source for the development of a sensitive method for early detection of smooth surface and fissure caries at an early stage. In this method the tooth is illuminated with a broad beam of diffuse monochromatic light within the blue green region from an argon laser source. The fluorescence of enamel, occurring in the yellow green region, is observed through a yellow high pass filter to exclude the tooth scattered blue laser light. Demineralized areas appear dark. Laser induced enamel fluorescence was later used for the development of a method for quantitative assessment *in vitro* of enamel demineralization. Mineral loss was seen to be strongly correlated with the relative loss of fluorescence. Demineralized tissues absorb dyes like Fluorl TGA, sodium florescein and fluoresce strongly. This is called as Dye Enhanced Laser Fluorescence. A portable diode laser system was developed (Diagnodent). It enables to recognize at an early stage, pathological changes that prove difficult or even impossible to detect.

Advantages:

- Carious lesions can be detected and their mineral loss measured.
- Convenient and relatively fast. Suitable for quantifying mineral loss around different restorations.

Ultrasonography

This method utilizes a sonar device in which a beam of ultrasound waves is directed against the tooth surface and if reflected is picked up by an appropriate receive. This method can be readily adopted to easily accessible areas but not for interproximal surfaces. Its main use therefore, has remained in *in vitro* testing studies. It is also more sensitive than visual-tactile method; however, it is not a quantitative method.

Xeroradiography

This is a technique that uses modified xerographic copying technique to images produced by diagnostic X-rays. These differ from conventional X-rays by demonstrating broader latitude of enhancement called "edge enhancement". Due to these small structures and areas of subtle density, differences are made more visible. This offers convenience, reduction in radiation dose and is economical too. It is twice as sensitive as conventional D-speed films.

Disadvantages:

- Exposure time varies with different brands.
- Electric charge over the film causes discomfort to the patient, as saliva acts as a medium for conduction of electricity.

Digital Fiber Optic Transillumination (DIFOTI)

It is relatively a new methodology that was developed to reduce the perceived short coming of FOTI by

combining FOTI and a digital CD camera. Images captured by the camera are sent to a computer for analysis.

Novel Methods for Caries Detection

Endoscopic Filtered Fluorescence Method (EFF)

Pitts and Longbottom in 1987 explored the use of EFF for the clinical diagnosis of carious lesions and compared results with conventional alternatives on occlusal and proximal sites. This work developed to include the use of intraoral video system for caries detection, the prototype "videoscope". Now that commercial intraoral cameras are increasingly available in practices, this may prove to be of practical clinical importance. Fluorescence that occurs when the tooth is illuminated with a blue light in the wavelength of 400-500 nm is read. Sound enamel and carious enamel show difference in fluorescence. When this fluoresced tooth is viewed through a specific broadband gelatin filter. Similarly a white light source can be connected to an endoscope by a fiber optic cable so that tooth can be viewed without the filter. This technique is referred to as white light endoscopy.

The EFF method has been shown to be highly sensitive for occlusal caries in enamel, but sensitivity is poor for occlusal caries in dentin. Specificity is poor for occlusal surfaces but high for approximal lesions at both thresholds. The method is reasonably good at detecting approximal lesions in enamel but not lesions in dentin.

Advantages:

- Easy for clinical application.
- Provides magnified image.

Disadvantages:

- Dry field and isolation mandatory.
- High cost.
- Time consuming.

CLASSIFICATION OF CAVITIES

GV Black Classification of Cavities (Fig. 14.2)

The origin of this classification goes back to the beginning of last century before the advent of magnification, good illumination, and radiographs, and well before the present sophisticated understanding of the significance of bacterial flora, saliva, fluoride, and the caries processes.

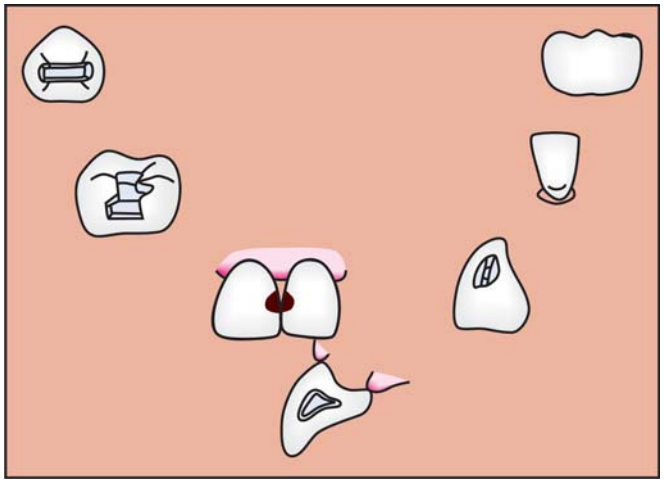


Fig. 14.2: Black's classification of cavities. Class I, II, III, IV, V, VI

At that time, there was no alternative method of identifying caries other than by counting visible lesions.

The Black classification represented a system of cavity designs for restoration rather than caries lesions per se.

Class I Lesion

- Lesions that begin in the structural defects of teeth such as pits, tissues and defective grooves.
- Location include – occlusal surface of molars and premolars.
- Occlusal 2/3rd of buccal and lingual surfaces.
- Lingual surface of anterior teeth.

Class II Lesions

- They are found on proximal surfaces of the cuspids and molars.

Class III Lesions

- Lesions found on the proximal surfaces of anterior teeth that do not involve or necessitate the removal of the incisal angle.

Class IV Lesions

- Lesions found on the proximal surfaces of anterior teeth that involve the incisal angle.

Class V Lesions

- Lesions that are found at the gingival third of the facial and lingual surfaces of the anterior and posterior teeth.

Class VI Lesions

- *Simon's modification* Lesions involving Cuspal tips and incisal edges of teeth.

Charbeneu's Classification

- Class II cavities: Cavities on single proximal surface of bicuspids and molars.
- Class VI: Cavities on both mesial and distal proximal surfaces of posterior teeth that will share a common occlusal isthmus.
- Lingual surfaces of upper anterior teeth.
- Any other unusually located pit/fissure involved with decay.

Sturdevant's Classification

Cavity	*Feature*
Simple cavity	A cavity involving only one tooth surface
Compound cavity	A cavity involving 2 surfaces of a tooth
Complex cavity	A cavity involves more than 2 surfaces of a tooth

Finn's Modification of Black's Cavity Preparation for Primary Teeth

Class I

- Cavities involving pits and fissures of the molar teeth and the buccal and lingual pits of all teeth.

Class II

- Cavities involving proximal surface of molar teeth with access established from the occlusal surface.

Class III

- Cavities involving proximal surfaces of anterior teeth which may or may not involve labial or lingual extension.

Class IV

- Restoration of proximal surface of an anterior tooth which involves the restoration of an incisal angle.

Class V

- Cavities present on the cervical third of all teeth, including proximal surface where the marginal ridge is not included in the cavity preparation.

Baume's Classification

a. Pit and fissure cavities.
b. Smooth surface cavities.

Classification of Mount and Hume (Table 14.2)

This new system defines the extent and complexity of a cavity and at the same time encourages a conservative approach to the preservation of natural tooth structure. This system is designed to utilize healing capacity of enamel and dentine.

PRINCIPLES OF CAVITY PREPARATION

Conventional Concept – Black's Concept

Outline Form

The locations that the peripheries of the completed tooth preparation will occupy on tooth surfaces. It can:

- Internal outline form
- External outline form.

Features:

1. Extend cavity margins to sound tooth structure.
2. Include all fissures.

Table 14.2: Mount and Hume classification

Site	*Size*			
	Minimal 1	*Moderate 2*	*Enlarged 3*	*Extensive 4*
Pit and fissure 1	1.1	1.2	1.3	1.4
Contact area 2	2.1	2.2	2.3	2.4
Cervical 3	3.1	3.2	3.3	3.4

3. Extend outline form to provide sufficient access.
4. Cavity margins should be placed in self cleansable areas.
5. Pulpal floor and axial wall should have average depth of 0.5 mm into dentin.
6. Margins should be extended to include all defective enamel.
7. 0.2-0.3 mm clearance from adjacent tooth while preparing proximal box in Class II cavities.
8. Margins of cavity preparation should not be in contact with the opposing tooth.
9. If less than 0.5 mm of tooth structure exists between 2 carious surfaces; then they should be joined.

Factors influencing outline form:

1. Embrasure area
2. Contact with opposing tooth
3. Caries index of individual
4. Position of tooth
5. Masticatory forces
6. Convexity of tooth
7. Extent of caries
8. Proximity of lesion to other defects
9. Esthetic considerations
10. Partial edentulism
11. Restorative material to be used
12. Existing restorations.

Resistance Form

It is the shape given to the cavity to enable the tooth as well as the restoration to withstand the stresses of mastication to which it is subject.

Features:

1. Flat pulpal floor, gingival wall.
2. Utilizing box form of cavity preparation
3. Cavity prepared in such a way that strong cusps and ridge areas remain with adequate dentine support.
4. Rounded internal line angles.
5. Butt joint at cavosurface margin
6. Removal of unsupported enamel
7. 90° cavosurface angle
8. Adequate bulk of restoration.
9. Adequate depth and width of the cavity.
10. Reverse curve in case of Class II cavities, when the proximal outline becomes offset buccolingually.
11. Gingival cavosurface bevel given only in case of permanent teeth and not in primary teeth.
12. Width of cavity should not exceed more than 1/4th – 1/5th of later Cuspal distance.
13. Pulpal floor should be 0.5 mm below DEJ.
14. Cavity wall should be divergent occlusally at marginal ridge areas.

Retention Form

It is comprised of those factors of cavity design that prevents the restoration form being displaced.

Features:

1. Parallel or an inverse taper of 5 degree under the triangular ridges of Cuspal areas in the case of Class I and Class II cavities.
2. Retention cores and grooves as used in Class III and Class V cavities.
3. Occlusal dovetail.
4. Pins placed into dentin.
5. Acid etching of enamel.
6. Proximal box of Class II design is divergent gingivally to contribute to the retention form.

Convenience Form

It includes shaping the cavity to facilitate access for instrumentation for condensation, adaptation and finishing.

Features:

1. Modification of cavosurface margins for ease of placement of restorative material.
2. Extension of buccal and lingual walls for visibility and access to deeper portions of cavity.
3. Proximal lesion can be instrumented from facial/lingual embrasures in a tooth wide accessible embrasures and intact marginal ridges.

Removal of any Remaining Infected Dentin

In the case of a small carious lesion, the infected dentin would be completely removed as above mentioned

principles are achieved. However, when a large carious lesion exists, some amount of infected dentin still remains in spite of above procedures. In such cases, it has to be removed.

Finishing Enamel Walls

1. To place margins on sound tooth structure.
2. To have smooth walls and rounded angles.
3. To facilitate placement a finishing of the restorative material.
4. Placement of taper or bevel for the appropriate restorative material.

Cleaning of the Cavity

It is the final step of debridement and maintaining clean debris free; cavity to aid in optimal restorative condition.

Class I Cavity Preparations in Primary Teeth and Its Modifications (Figs 14.3 A to C)

First Primary Molar

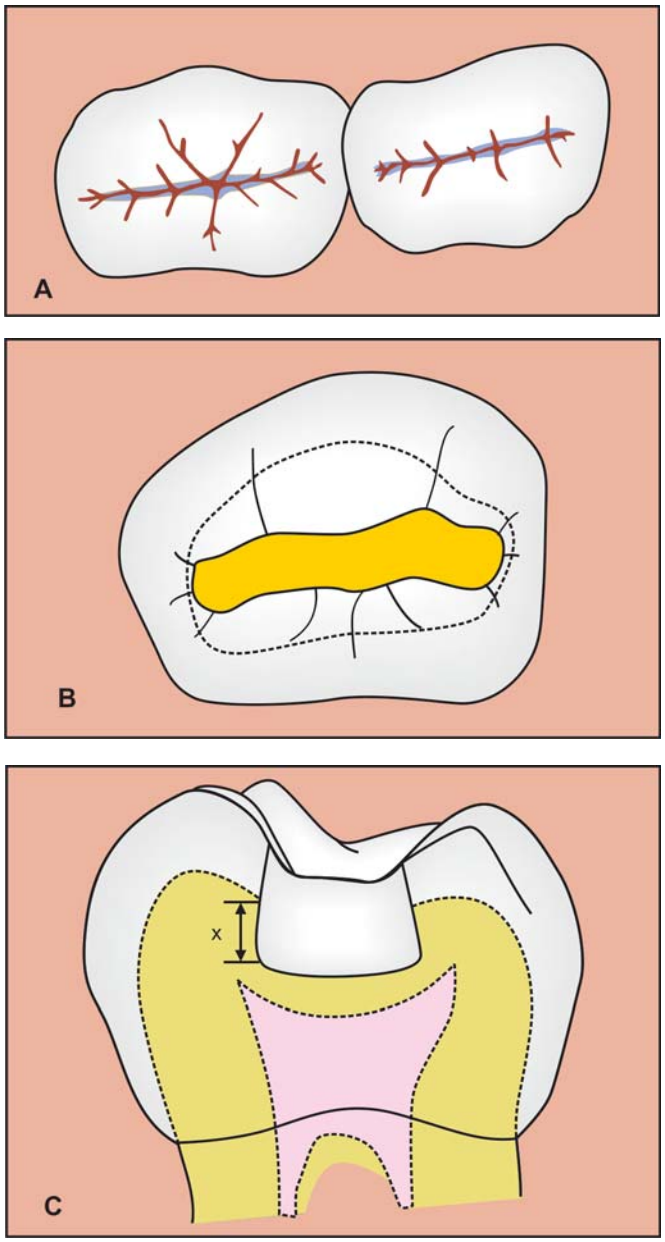
- The mesial pit is affected only after the central pit. Hence, the outline form includes the central pit, buccal and lingual developmental grooves and distal triangular fossa.
- The ridge of enamel joining the mesiobuccal and mesiolingual cusp need not be involved in cavity preparation due to:
  - Proximity of mesiobuccal pulp horn
  - Strength will be compromised.
- If the occlusal surface is extensively destroyed by nursing bottle caries, a wide outline form may be needed.

Second Primary and First Permanent Molars

- The outline form must include all fissures on the occlusal surface.
- The intercuspal dimension should be small.

Mandibular

- Separate buccal and lingual restorations are placed.
- Buccal or lingual extension must have the cavity walls parallel or converging occlusally.



Figs 14.3A to C: Class I outline forms. A. Outline form in primary 1st and 2nd molar, B. External outline form, C. Internal outline form (x-0.5 mm depth into dentine)

- Extension is **0.5 mm** into dentine.
- Retention groove may be placed in dentine.

Maxillary

- Caries is limited to the central pit, distal pit, the groove separating the cusp of carabelli and mesiolingual cusp.

- The oblique ridge is not included unless if caries undermines it.
- The depth of lingual developmental groove and its continuity with the distal pit and developmental groove necessitates an occlusolingual restoration.
- Inclusion of the lingual pit, groove and accessory groove between the lingual groove and the groove separating the mesiolingual cusp and cusp of carabelli is extension for prevention.
- The width of the lingual extension is minimal, compensated by increase in depth.

Second Permanent Molars

- Lingual extensions are usually not needed.
- Mesial and distal pits need separate cavities.
- Extruding over the central groove and mesial and distal pits will involve the oblique ridge.
- The difficulties faced with mandibular 2nd permanent molar are:
  - Tooth is affected very soon after eruption.
  - The tooth may only be partly erupted with the distal aspect covered by an operculum of tissue.

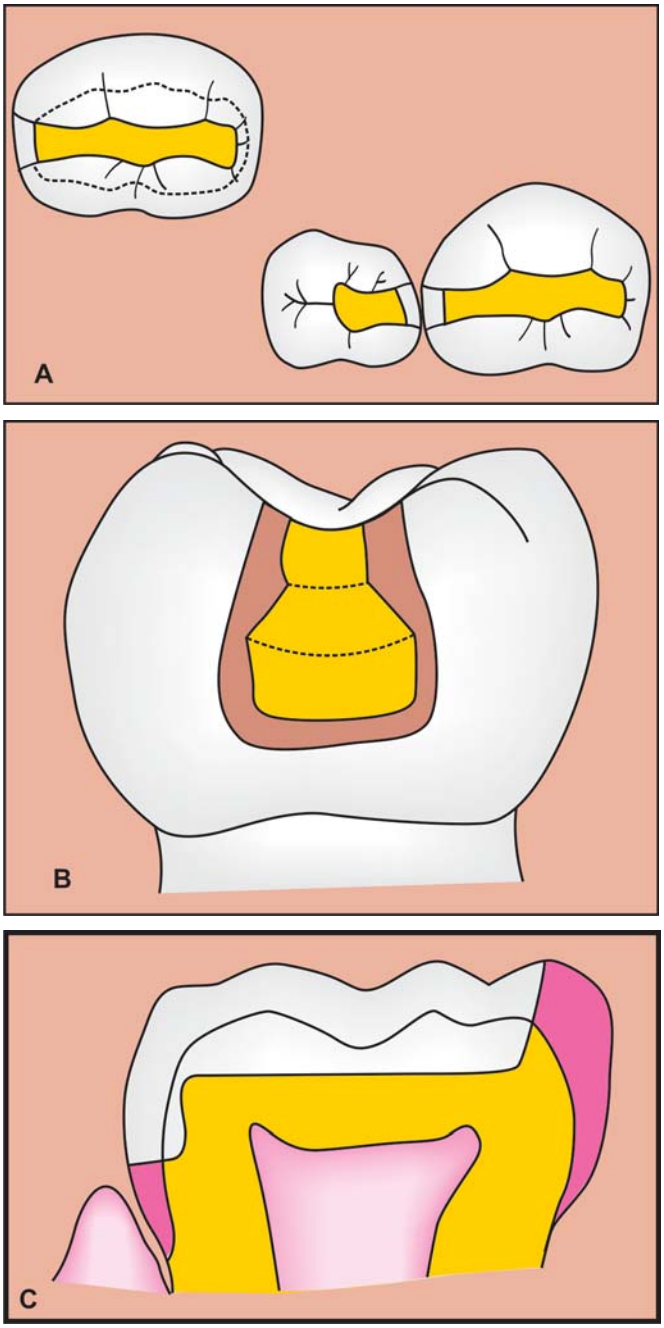
Developmental Pits occur in these Areas

- Midway down the mesiobuccal developmental groove in lower second, primary first and second permanent molars.
- Midway down the lingual developmental groove in the maxillary second primary, first and second permanent molars.
- Between the 5th cusp and mesiolingual cusp of 2nd primary and first permanent molars.
- Lingual pit in maxillary permanent incisors, rarely in permanent canines, laterals are affected.
- Caries prone developmental pits and accessory grooves are included.

Class II Cavity Preparation (Figs 14.4 A to C)

Fin's Modification

Cavity involving proximal surfaces of bicuspids and molars with access established from occlusal surface.



Figs 14.4 A to C: Class II cavity preparation. A. External view, B. Proximal view, C. Cross-sectional view

Cavity Preparation

Primary molars:

- Cavity design depends on
  - Presence of caries
  - Potential to develop caries
  - Preservations of round tooth structure.

- Class II cavity includes a proximal box joined by an isthmus to an occlusal extension.
- If the occlusal aspect is unaffected the proximal box alone is prepared with turbine hand piece using a small, pear shaped bur.
- Occlusal preparation will include carious stained fissures with a depth of 1.5 mm.
- Pulpal floor is flat mesiodistally, rounded buccolingually.
- Remaining caries is removed with slow speed round bur/excavators.
- Narrow occlusal aspect of cavity is prepared placing the buccal and lingual margins are easily cleansable areas.
- Occlusal approach is at 90° to occlusal table.
- Cavosurface angles or proximobuccal and proximolingual walls are at 70°.
- If initial flaring is wide, a preformed crown is used.
- Angulation of bur to the long axis of the tooth must be clear the contact with tooth buccolingually.
- The bur is advanced to gingiva, making a mesiodistal movement of 90°.
- Decay begins at or below contact area; hence the gingival seat is extended past this contact.
- Clearance from opposing tooth favors matrix band placement.
- Buccal and lingual limits of the gingival seat are placed clear of adjacent tooth contact to clean the margin of the restoration.
- Occlusal aspect is kept narrow.
- Buccal and lingual cavosurface angles are ~ 90°.
- Depth of proximal box is 1 mm, the axial walls and gingival seat placed into dentine, so are the proximal retentive grooves to avoid unsupported enamel.
- Isthmus width is = 1/3rd of intercuspal distance.
- In Class II cavity an adequate depth and non-coincidence of axiopulpal line angle and the isthmus is important.
- Axiopulpal line angle is rounded, grooved, tunneled etc. to give bulk to the weak isthmus area.

Retention:

- By extension into occlusal fissures
- By diverging walls of proximal box
- 'U' groove within the dentine of the buccal and lingual walls of the proximal box No.1, 2 round fissure bur is used.
- By a groove in dentine of the gingival seat.

Finishing: Final refinement and gingival seats is made with hatchets and chisels.

Modifications: According to

- Occlusal pattern
- Extent of caries
- Age of the patient.

- If 2 Class II cavities occur on same tooth, with an intact oblique ridge, a single restoration is given.
- If ridge is undermined a stainless steel crown is preferred.
- Another method is reduction of a weakened cusp and overlaying it with enamel.
- When marginal ridge is undermined.
- When proximobuccal/lingual cusps are undermined before the marginal ridge finally breaks down.

Modifications for proximal lesions

- Tunnel cavity (Fig. 14.5)
- Internal preparation
- Slot cavity/Mini box preparation
- Proximal approach.

Class III Cavity Preparation

Finn's Modification

- Cavities involving proximal surfaces of ant teeth which may or may not involve labial or lingual extension.

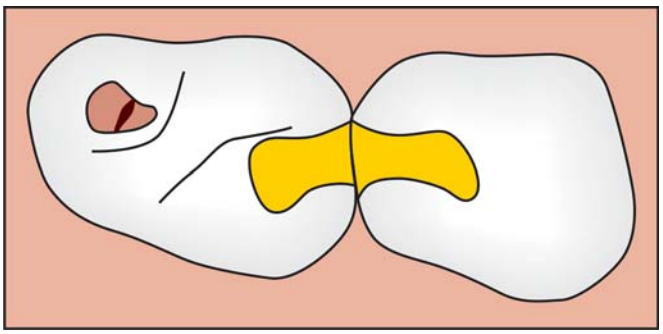


Fig. 14.5: Tunnel cavity

Primary Incisor Tooth

The outline form is triangular with the base gingivally; the buccal and lingual walls should be parallel.

- Pear shaped bur is used.
- Gingival wall inclines occlusally, parallel the enamel rods, this also gives retention.
- Cavity depth is 0.5 mm into DEJ
- Retentive grooves are made at DEJ with No.2 round bur.

Modification:

- In closed contact, an indirect approach is given.
- A dovetail or local extending into the middle of the tooth gives resistance to the lateral displacement.
- Interproximal area must resemble letter C with its open end facing the retentive lock.
- When Class III and V lesions co-exist in the same tooth, Class V forms the retentive lock.

Primary Canine

- The dovetail lock is rarely used.
- When the distal cavity is large, a palatal or facial lock is useful for maxillary and mandibular teeth respectively.

Modified Class III Cavity (Fig. 14.6)

- A dovetail is used lingually or labially
- Retentive features may be given as Labiogingival and linguogingival angles.

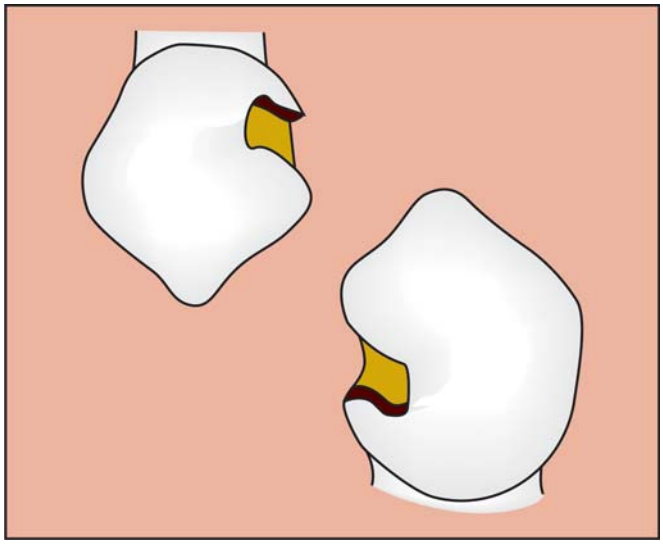


Fig. 14.6: Modified class III cavity

Class IV Cavity Preparation

Finn's Modification

- A restoration of proximal surfaces of anterior teeth which involves the restoration of incisal edge.

Class V Cavity Preparation (Fig. 14.7)

Finn's Modification

- Cavities present on cervical third of all teeth including proximal surface where the marginal ridge is not included in cavity preparation.

Outline form:

- Includes the carious site
- Decalcified areas and lesions 2 mm apart are included as extension for prevention.
- A kidney shaped gently curved outline with square sharp outline of mesial and distal margins is preferred.
- No. 330 bur is used.

Retention form:

- Dentinal under cuts
- The gingival enamel margin should follow a regular curve parallel to the gingival attachment unless the lesion extends subgingivally.

Preparation Alternatives to Conventional Cavity

Air Abrasion

There has recently been a resurgence of interest in air abrasion technology with several different commercial units available. With air abrasion machines, aluminium

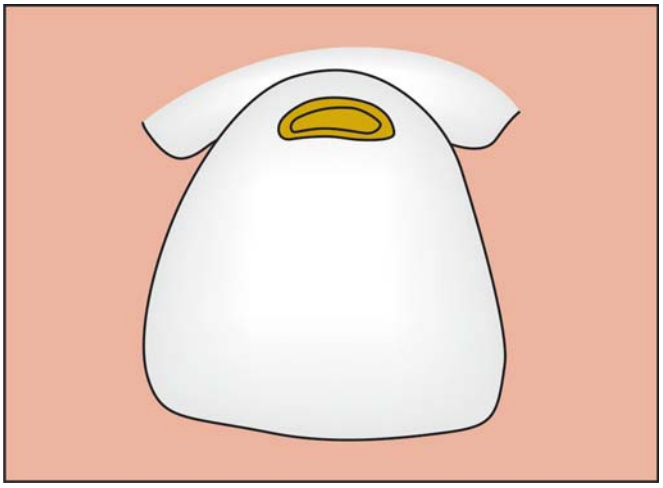


Fig. 14.7: Class V cavity in primary anterior

oxide particles (27 or 50 µm) are blasted against the teeth under a range of pressures (30-160 psi) with variable particle flow rates.

One very obvious concern is the safety aspect due to the presence of quantities of free aluminium oxide in the surgery environment. In theory aluminium oxide is considered harmless. It is found in a wide variety of products from toothpastes to polishing wheels. The size of the particles is considered too big to enter the distal airways or alveoli of the lungs. What dust does enter the lungs should be easily removed by ciliary action. However, anyone who has used one of these units will know that control of the dust is an ongoing challenge; rubber dam and very good suction help, but it still seems to spread.

Air abrasion produced a cavity preparation with both rounded cavo-surface margins and internal line angles. The surface it creates is irregular with many fine voids and defects. Initially it was considered that this surface might provide enough retention without etching but studies show this as erroneous.

Some of the clear advantages proposed for air abrasion are:

- Elimination of vibration, less noise, and decreased pressure.
- Reduction in pain during cavity preparation; 85% of patients do not require local analgesia.
- Less damaging pulpal effects than with conventional hand-piece usage, when used at higher pressures of 160 psi and with smaller particle size of 27 µm.
- Less fracture or crazing of enamel and dentine during cavity preparation.
- Root canal access through porcelain crowns without fracturing porcelain.

Air abrasion has been proposed for:

- Cleaning and removing stains and incipient caries from pits and fissures prior to sealant and PRR's.
- Small Class I, III, IV and V cavity preparations and selected class II preparations.
- Repair and removal of composites, glass ionomer, and porcelain restorations.
- Cleaning and preparation of castings, orthodontic bands, and brackets prior to cementation.

What it cannot do is remove leathery dentinal caries or prepare extensive cavities requiring classical retentive form.

To use it successfully, the clinician must learn a new technique as the tip does not touch the tooth and therefore there is no tactile feedback. The tip width and the tip to tooth distance seem to have most influence on the cavity width and depth. Increasing the distance produces larger shallower cuts. Increasing the tip diameter produces larger deeper cuts. Therefore, the most precise removal of tooth tissue is achieved with a small inner diameter tip (0.38 mm), held 2 mm from the tooth surface. If cutting a class I cavity, it is essential to protect the adjacent tooth. Care must also be taken around the soft tissues to prevent surgical emphysema. Glass/mirror surfaces may be damaged by the dust.

In the preparation of PRRs, this technique gives as good a result as conventional methods. It was thought that cavities would be smaller with air abrasion but this has not been realized practically.

In conclusion, air abrasion may be useful in preparation of small cavities with reduced patient discomfort, when combined with acid etching to obtain a good bond with adhesive materials, and when correctly and carefully used. However, the dust is a practical problem.

Ozone Therapy

Dental treatments are constantly evolving. One such innovation, ozone therapy (heal ozone) has hit the media headlines, spiking much public interest. The technology is available and costly devices for delivery of ozone for dental purposes exist, but as yet the superiority of this modality over conventional treatment has not been proven with properly conducted clinical trials.

The theory of the action of ozone is that it kills micro-organisms, by oxidizing their cell walls to rupture their cytoplasmic membranes, that is, it is bactericidal. In laboratories, it has been shown that ozone can substantially reduce the numbers of microorganisms within carious dentine on short exposures of 10-20s. However, the clinical significance of this has not been established. It has been postulated that the use of ozone together with a remineralizing regime of fluoride paste and rinse, oral hygiene instruction, and dietary advice

would be beneficial and that it would arrest primary root caries to a greater extent than remineralizing regime alone. It has also been suggested that ozone treatment can stabilize pit and fissure caries preventing further deterioration. However, the authors will stay with more traditional methods of caries control until proper controlled trails of reasonable duration (>4 years) have been reported.

Lasers (Table 14.3)

The public perception of lasers in dentistry is that they can do remarkable things painlessly, so obviously this appeal to a greater number of people. However, the number of dentists offering lasers as an option in their practices is still small. The cost of equipment is obviously a significant factor, but as with all new technologies, it is important that each dentist considers the proven clinical outcomes, that is, what the recorded literature states regarding the safety, efficacy, and effectiveness.

Table 14.3: Laser types and applications

Carbon dioxide lasers	Soft tissue incision/ablation Gingival troughening Aesthetic contouring of gingivae Treatment of oral ulcers Frenectomy and Gingivectomy De-epithelialization of gingival tissue during periodontal regenerative procedures.
Nd: YAG	Similar to above plus removal of incipient caries but because of the depth of penetration there is a greater risk of collateral damage than with dioxide lasers.
Er: YAG	Caries removal Cavity preparation in both enamel and dentine. Preparation of root canals.
Argon laser	Resin curing Tooth bleaching Treatment of ulcers Aesthetic gingival contouring Frenectomy and Gingivectomy

With lasers this is further complicated by the fact that there are many different types of lasers, with different uses and new types and applications being produced constantly.

ADVANCED RESTORATIVE DENTISTRY

The Hydrochloric Acid—Pumice Microabrasion Technique

This is a controlled method of removing surface enamel in order to improve discolorations that are limited to the outer enamel layer. It is achieved by a combination of abrasion and erosion – the term "abrasion" is sometimes used. In the clinical technique that will be described no more than 100 µm of enamel are removed. Once completed the procedure should not be repeated again in the future. Too much enamel removal is potentially damaging to the pulp and cosmetically the underlying dentine color will become more evident.

Indications

- Fluorosis
- Idiopathic speckling
- Post orthodontic treatment demineralization
- Prior to veneer placement for well-demarcated stains
- White/brown surface staining, e.g. secondary to primary predecessor infection or trauma (Turner teeth).

Armamentarium

- Bicarbonate of soda/water
- Copalite varnish or Vaseline
- Fluoridated toothpaste
- Non-acidulated fluoride (0-2 years: drops)
- Pumice
- rubber dam
- rubber prophylaxis cup
- Soflex discs (3M)
- 18% hydrochloric acid.

Technique

1. Perform preoperative vitality tests, take radiographs and photographs.
2. Clean the teeth with pumice and water wash and dry.
3. Isolate the teeth to be treated with rubber dam, and paint Copalite varnish around the necks of the dam or Vaseline under the dam.

4. Place a mixture of sodium bicarbonate and water on the dam behind the teeth as protection in case of spillage.
5. Mix 18% hydrochloric acid with pumice into a slurry and apply a small amount to the labial surface on either a rubber cup rotating slowly for 5s or a wooden stick rubbed over the surface for 5s before washing for 5s directly into an aspirator tip. Repeat until the stain has reduced upto a maximum of 10 x 5s applications per tooth. Any improvement that is going to occur will have done so by this time.
6. Apply the fluoride drops to the teeth for 3 minutes.
7. Remove the rubber dam.
8. Polish the teeth with the finest Soflex discs.
9. Polish the teeth with fluoridated toothpaste for 1 min.
10. Review in 1 month for vitality tests and clinical photographs.
11. Review biannually checking pulpal status.

Critical analysis of the effectiveness of the technique should not be made immediately, but delayed for at least 1 month as the appearance of the teeth will continue to improve over this time. Experience has shown that brown mottling is removed more easily than white, but even where white mottling is incompletely removed it nevertheless becomes less perceptible. This phenomenon has been attributed to the relatively prismless layer of compacted surface enamel produced by the 'abrasion' technique, which alters the optical properties of the tooth surface.

Long-term studies of the technique have found no association with pulpal damage, increased caries susceptibility, or significant prolonged thermal sensitivity. Patient compliance and satisfaction is good and any dissatisfaction is usually due to inadequate preoperative explanation. The technique is easy to perform for the operator and patient, and is not time consuming. Removal of any mottled area is permanent the appearance by the HCl – pumice microabrasion technique has not harmful effects and may make it easier to mask some lesions with veneers.

Localized Composite Resin Restorations

This restorative technique uses recent advances in dental materials science to replace defective enamel with a restoration that bonds to and blends with enamel.

Indications

- Well-demarcated white, yellow, or brown hypo-mineralized enamel.

Armamentarium

- Rubber dam/contoured matrix strips
- round and fissure diamond burs
- enamel/dentine bonding kit
- New generation, highly polishable, hybrid composite resin
- Soflex discs and interproximal polishing strips.

Technique

1. Take preoperative photographs and select the shade.
2. Apply rubber dam or contoured matrix strips.
3. Remove demarcated lesion with a round diamond bur down to the amelodentinal junction (ADJ)/ Dentinoenamel junction (DEJ).
4. Chamfer the enamel margins with a diamond fissure bur to increase the surface area available for retention.
5. Etch the enamel margins – wash and dry.
6. Apply the dentine primer to dentine and dry.
7. Apply the enamel and dentine bonding agent and light cure.
8. Apply the chosen shade of composite using a brush lubricated with the bonding agent to smooth and shape, and light-cure for the recommended time.
9. Remove the matrix strip/rubber dam.
10. Polish with graded Soflex discs, finishing burs and interproximal strips if required. Add characterization to the surface of the composite.
11. Take postoperative photographs.

The localized restoration is quick and easy to complete. Despite the removal of defective enamel down to the DEJ there is often no significant sensitivity and therefore no need for local anesthesia. If the hypoplastic

enamel has become carious and this extends into dentine then a liner of glass ionomer cement (correct shade) prior to placement of the composite resin will be necessary. Local anesthesia will probably be required in these cases. Advances in bonding and resin technology make these restorations simple and obviate the need for a full labial veneer. Disadvantages are marginal staining, accurate color match, and reduced composite translucency when lined by glass ionomer cement.

Composite Resin Veneers

Although the porcelain jacket crown (PJC) may be the most satisfactory long-term restoration for a severely hypoplastic or discolored tooth, it is not an appropriate solution for children for two reasons:

1. The large size of the young pulp horns and chamber.
2. The immature gingival contour.

Composite veneers may be direct (placed at initial appointment) or indirect (placed at a subsequent appointment having been fabricated in the laboratory). The conservative veneering methods may not just offer a temporary solution, but a satisfactory long-term alternative to the PJC. Most composite veneers placed in children and adolescents are of the 'direct' type, as the durability of the indirect composite veneers is as yet unknown.

Before proceeding with any veneering technique, the decision must be made whether to reduce the thickness of labial enamel before placing the veneer. Certain factors should be considered:

- Increased labiopalatal bulk makes it harder to maintain good oral hygiene. This may be courting disaster in the adolescent with a dubious oral hygiene technique.
- Composite resin has a better bond strength to enamel when the surface layer of 200-300 mm is removed.
- If a tooth is much discolored some sort of reduction will be desirable, as a thicker layer of composite will be required to mask the intense stain.
- If a tooth is already instanding or rotated, its appearance can be enhanced by a thicker labial veneer.

New generation, highly polishable, hybrid composite resins can replace relatively large amounts of missing tooth tissue as well as being used in thin sections as a veneer. Combinations of shades can be used to stimulate natural colour gradations and hues.

Indications

- Discoloration
- Enamel defects
- Diastema
- Malpositioned teeth
- Large restorations.

Contraindications

- Insufficient available enamel for bonding
- Oral habits, e.g. woodwind musicians.

Armamentarium

- Rubber dam/contoured matrix strips
- Preparation and finishing burs
- New generation, highly polishable, hybrid composite resin
- Soflex discs and interproximal polishing strips.

Technique

- Use a tapered diamond bur to reduce labial enamel by 0.3-0.5 mm. Identify the finish line at the gingival margin and also mesially and distally just labial to the contact points.
- Clean the tooth with slurry of pumice in water. Wash and dry and select the shade.
- Isolate the tooth either with rubber dam or a contoured matrix strip. Hold this in place by applying unfilled resin to its gingival side against the gingiva and curing for 10s.
- Etch the enamel for 60 seconds, wash, and dry.
- Where dentine is exposed apply dentine primer.
- Apply a thin layer of bonding resin to the labial surface with a brush and cure for 15s. It may be necessary to use an opaquer at this stage if the discoloration is intense.

- Apply composite resin of the desired shade to the labial surface and roughly shape it into all areas with a plastic instrument, then use a brush lubricated with unfilled resin to 'paddle' and smooth it into the desired shape. Cure 60 seconds gingivally, 60 seconds mesioincisally, 60 seconds distoincisally, and 60 seconds from the palatal aspect if incisal coverage has been used. Different shades of composite can be combined to achieve good matches with adjacent teeth and a transition from a relatively dark gingival area to a light more translucent incisal region.
- Flick away the unfilled resin holding the contour strip and remove the strip.
- Finish the margins with diamond finishing burs and interproximal strips and the labial surface with graded sandpaper discs. Characterization should be added to improve light reflection properties.

The exact design of the composite veneer will be dependent upon each clinical case, but will usually be one of four types: intraenamel or window preparation; incisal bevel; overlapped incisal edge; or feathered incisal edge.

Tooth preparation will not normally expose dentine, but this will be unavoidable in some cases of localized hypoplasia or with caries. Sound dentine may need to be covered by glass ionomer cement prior to placement of the composite veneer.

Porcelain Veneers

Porcelain has several advantages over composite as a veneering material: its appearance is superior; it has a better resistance to abrasion; and it is well tolerated by the gingival tissues. However, it is vital that the porcelain fits exactly and that the film thickness of the luting cement is kept to a minimum. These luting cements are only moderately filled composite resins and they absorb water, hydrolyse, and stain. This coupled with the apical migration of the gingival margin in young patients can result in an unacceptable aesthetic appearance in a relatively short time.

Instruction in standard porcelain veneer preparation is covered in restorative dentistry textbooks. If there are occasions when they are used at an earlier age then the same principles apply. However, a non-standard application that is being used more frequently at a younger age is the restoration of the peg lateral incisor. This utilizes a no-preparation technique and the technician is asked to produce a three-quarter wrap-around veneer finished to a knife edge at the gingival margin. An elastomeric impression is taken after gingival retraction to obtain the maximum length of crown, and cementation should be under rubber dam.

Adhesive Metal Castings

The development of acid-etched, retained cast restorations has allowed the fabrication of cast occlusal onlays for posterior teeth and palatal veneers for incisors and canines. These restorations are manufactured with minimal or no tooth preparation and are ideal for cases where there is a risk of tooth tissue loss.

Indications

- Amelogenesis imperfecta
- Dentinogenesis imperfecta
- Dental erosion, attrition or abrasion
- Enamel hypoplasia.

Armamentarium

- Gingival retraction cord
- Elastomeric impression material
- Facebow system
- Semi-adjustable articulator
- Rubber dam.

Technique

1. Obtain study models (these are essential) and photographs if possible.
2. Perform a full mouth prophylaxis.
3. Ensure good moisture isolation.
4. Place retraction cord into the gingival crevices of the teeth to be treated and remove immediately prior to taking the impression.
5. Take an impression using an elastomeric impression material – a putty/wash system is the best and check the margins are easily distinguishable.

6. Take a facebow transfer an interocclusal record in the retruded axis position.
7. Mount the casts on a semi-adjustable articulator.
8. Construct cast onlays, a maximum of 1.5 mm thick occlusally in either nickel/chrome or gold.
9. Grit – blast the fitting surfaces of the occlusal onlays.
10. Return to the mouth and check the fit of the onlays.
11. Polish the teeth with pumice and isolate under rubber dam where possible.
12. Cement onlays.
13. Check occlusion.
14. Review in 1 week for problems and regularly thereafter.

When used to protect the palatal aspect of upper anterior teeth there may be an aesthetic problem as the metal may 'shine through' the translucent incisal tip of young teeth. The durability of this form of restoration has now been confirmed by 10 year evaluation studies.

Indirect Composite Resin Onlays

An alternative to cast metal onlays are indirect composite onlays. In addition to the obvious aesthetic advantages these restorations can be modified relatively easily. This is particularly useful for conditions such as erosion where the disease process may well be ongoing and therefore the tooth and/or restoration may require repair or additions. Studies suggest that these restorations are durable in the anterior region; however, in response to patient demand indirect composite onlays are increasingly being used in the posterior region, where their durability is currently unclear. The disadvantage of these restorations is that they need to be thicker than their cast counterparts, are bulkier and can cause greater increases in vertical dimension. However, in young patients, providing the occlusion remains balanced and there is no periodontal pathology, then increases in vertical dimension appear well tolerated.

ATRAUMATIC RESTORATIVE TREATMENT (ART)

The Atraumatic Restorative Treatment (ART) is a procedure based on removing carious tooth tissues using hand instruments alone and restoring the cavity with an adhesive restorative material. At present the restorative material is glass-ionomer. This procedure has been developed because millions of people in less-industrialized countries and certain special groups such as refugees and people living in deprived communities are unable to obtain restorative dental care. Their teeth gradually decay until extraction is the only treatment option. These people have not benefited from the developments that have brought about improved oral health and care in the industrialized world. The absence of electricity and the idea that restorative dental care always requires special electrically driven equipment are the main reasons for this situation. In contrast, the ART approach enables treatment of cavities in teeth to be provided for people residing in areas where electricity is not available or, alternatively, in areas which have electricity, but where the community cannot afford expensive dental equipment.

Glass-ionomers are very useful dental restorative materials. In addition to its use as a restorative material, glass-ionomers can be applied in the very early stages of caries development. The glass-ionomer sticks to the tooth and halts or slows the progression of lesions, mainly because it slowly releases fluoride.

ART is, however, just one component of oral health care which must start with health promoting messages about a prudent diet and good oral hygiene using a fluoride containing toothpaste. Sealing pits and grooves in the chewing surfaces of teeth is another preventive action to consider. Removing carious tooth tissue with hand instruments alone, and restoring the cavity with an adhesive material - that is ART - will conserve as much tooth structure as possible and prevent further decay. This approach is a breakthrough towards achieving the goal that all people should retain as many teeth as possible: "Teeth for life".

Principles of ART

- Removing carious tooth tissue using hand instruments only.
- Restoring the cavity with a glass ionomer.

Table 14.4: List of instruments needed for ART

Instruments	*Materials*	*Other*
Mouth mirror	Cotton wool roll	Examination gloves
Explorer	Cotton wool pellet	Mouth mask
Pair of tweezers	Clean water	Operating light
Dental hatchet	Glass-ionomer restorative material	Operation bed/headrest extension
Spoon excavator, small	Liquid, powder and measuring spoon	Stool
Spoon excavator, medium	Dentine conditioner	Methylated alcohol
Spoon excavator, large	Petroleum jelly	Pressure cooker
Applier/carver	Wedge	Instrument forceps
Glass slab or paper mixing pad	Plastic strip	Soap and towel
Spatula	Articulation paper	Sheet of textile
		Sharpening stone and oil

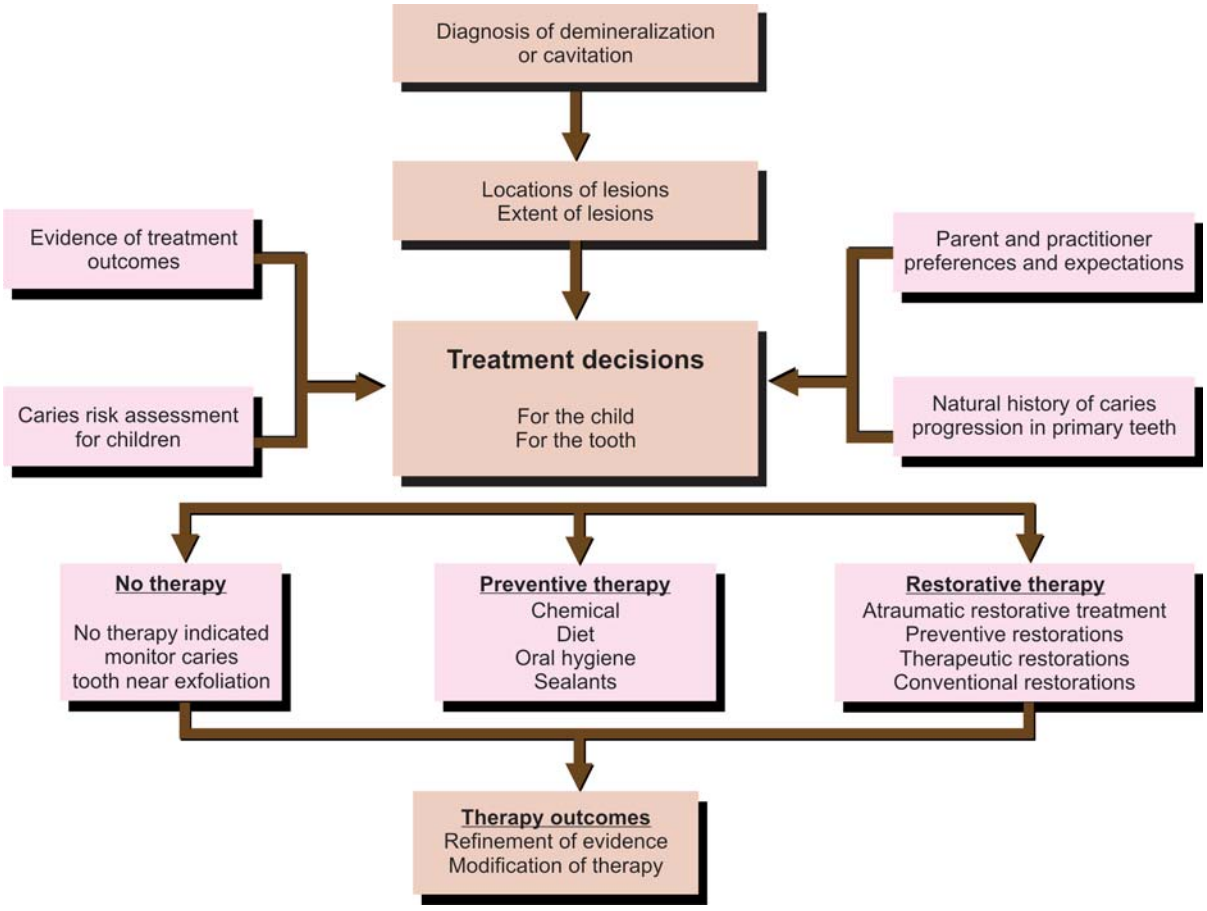


Fig. 14.8: A concept for primary teeth diagnosis and therapy based on caries risk assessment

Reasons for Using Hand Instruments Rather than Electric Driven Handpieces

- It makes restorative care accessible to all population.
- The use of a biological approach, which requires minimal cavity preparation that conserves sound tooth tissues and causes less trauma to the low cost of hand instruments compared to electrically driven dental equipment the limitation of pain that reduces the need for local anesthesia to a minimum and reduces psychological trauma to patients.
- Simplified infection control; hand instruments can be easily cleaned and sterilized after every patient.

Reasons for using Glass Ionomers

- As the glass ionomer chemically bonds to both enamel and dentin, the need to cut sound tooth tissue to prepare the cavity is reduced.
- Fluoride is released from the restoration to prevent and arrest caries.
- Glass ionomers are biocompatible, does not cause any irritation to pulp and gingiva and has a coefficient of thermal expansion similar to tooth structure.

List of Essential Instruments and Materials (Table 14.4)

Figure 14.7 emphasizes the basis of treatment decisions according to the data collected from the patient through a systematic procedure thereby devising a perfect customized treatment plan targeting the patient's complaint.

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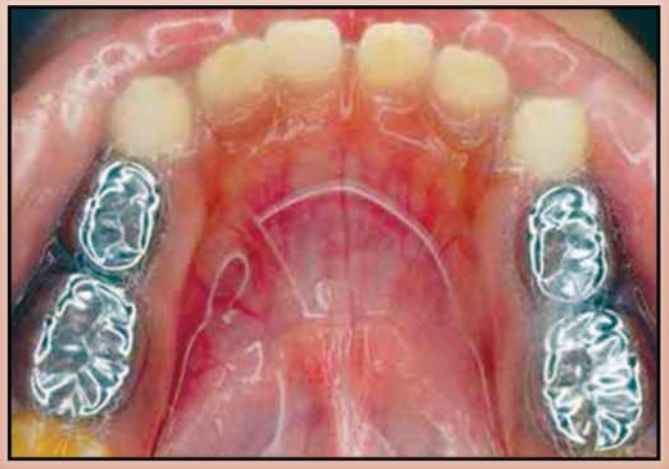


Stainless Steel Crowns

INTRODUCTION

The most commonly faced dental problems in children include dental caries (Nursing bottle caries), malocclusion (cross bites) and fractured teeth. There is no good reason for leaving decayed primary teeth, untreated malocclusion and fractured teeth in a child's mouth. The importance of primary teeth should be considered in helping speech, mastication, maintaining arch length and cosmetic function.

As the treatment of primary and young permanent teeth with advanced carious lesion has been a constant and difficult problem for the dentist, the stainless crowns has become an important factor in the restoration of the extensively carious lesion by maintaining arch integrity for the permanent teeth and by instilling in the child positive attitudes towards oral health.

Humphrey introduced stainless steel crowns, which is a semi-permanent restoration used for both primary and young permanent dentition. Stainless steel crown has provided an effective and practical means of restoring badly decayed or broken down teeth where amalgam would not be retained.

Dawson, Messler, Einwag reported stainless steel crown were superior to large multisurface amalgam restoration particularly in first primary molars and have a long clinical life span than two or three surface amalgam restoration.

Stainless steel crowns are available in different sizes with performed anatomy. Although chrome steel restoration has been used for nearly 50 years very little significant research has been done.

The technique for placing a stainless crown in a simple one and in the hands of many clinicians can certainly be performed far quicker than a multisurface restoration.

Modern crown are so well constructed that trimming them to fit is almost a thing of the past and tooth preparation is minimal and very quick. The fracture rate for stainless steel crowns is very low indeed, and far better than that reported for amalgam restorations.

The dentist treating children should recognize the durability, preventive aspect and cost effectiveness of the stainless steel crown as a restorative choice for the primary dentition.

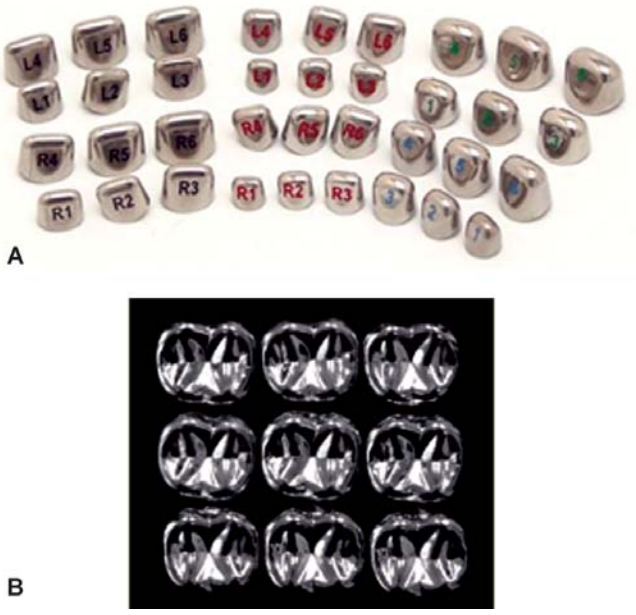
TYPES OF STAINLESS STEEL CROWNS

Stainless steel crowns are available in six sizes for both primary molars and permanent first molars. Sizes 4 and 5 are the most often used. A size 7 is available for extra large teeth. These sizes are supplied in kit form, with the user needing to record only those sizes frequently used.

1. **Untrimmed crowns:** (e.g. Rocky Mountain) these crowns are neither trimmed nor contoured, require lot of adaptation, thus are time consuming.
2. **Pretrimmed crowns** (e.g. Unitek stainless steel crowns, MN and Denovo crowns, Denovo co Arcadia, CA).

   These crowns have straight, non-contoured side but are festooned to follow a line parallel to the gingival crest. They still require contouring and trimming (Fig. 15.1).
3. **Precontoured crowns** (e.g. Ni-Cr ion crowns and Unitek stainless steel crowns, MN, Sankuin copper crowns).

These crowns are festooned and are also precontoured although a minimal amount of festooning and trimming may be necessary.



Figs 15.1 A and B: Stainless steel crowns. A. Anteriors, B. Posteriors

CLASSIFICATION OF STAINLESS STEEL CROWNS

Knowledge of the different stainless steel crowns is necessary to determine how they can affect the adaptation of the various crowns to the type of preparation recommended. The different type of crowns includes Ion crown, Unitek, Rocky Mountain, Ormco. The original alloys used by manufacturers for steel crown construction were stainless steel. Rocky Mountain and Unitek crown still are stainless steel; but the ion crown is Iconel, a nickel-chromium alloy.

Stainless Steel Crowns

Stainless steels are low carbon alloy steel that contains at least 11.50% chromium. Chromium contributes to the formation of a very thin surface film, probably oxide that protects against corrosive attack.

There are three general classes of stainless steel:

- The heat hardenable 400 series martensitic types.
- The non-heat hardenable 400 series ferrite types.
- The austenitic types of chromium-nickel-manganese 200 series and chromium-nickel 300 series.

Austenitic Types

The austenitic types are used by Rocky Mountain and Unitek (Monrovia, California, USA) for their crown-referred to as 18-8 since they contain about 18% chromium and 8% nickel. In addition, they contain small amounts of other alloying elements, carbon (0.08% - 0.15%) and the iron. Generally, the stainless steels contain about 70% iron. The austenitic types have high ductility, low yield strength, and high ultimate strength, which make them outstanding for deep drawing and forming procedures. They are readily welded and can be work hardened to high levels, although not as high as can be obtained by heat-treating the appropriate types of the 400 series.

The austenitic types provide the best corrosion resistance of all the stainless particularly when they have been annealed to dissolve chromium carbides and then rapidly quenched to retain the carbon in solution.

Ion Crown

The ion nickel-chromium crowns from 3M are made of a nickel-chromium alloy and are constructed of Iconel 600, a relatively new addition to the category of preformed crowns, and are primarily nickel-chromium.

These stainless steel crowns exhibit the following properties:

- Annealing does not increase their strength.
- They work harden; strength increases from manipulation with pliers.
- Their high chromium content reduces corrosion.
- Soldering with flux reduces their corrosion resistance.

Aluminium Crowns (Fig. 15.2)

Aluminium crowns are pretrimmed, anatomical, temporary gold anodized.

Fig. 15.2: Aluminium crown

Advantages

- It eliminates metallic taste
- The pretrimmed aluminium crowns helps in minimizing office time.

Availability

They are available in 80 different sizes.

- 30-first permanent bicuspids
- 12-second permanent bicuspids
- 24-first permanent molars and
- 12-second molars.

Composition

Stainless Steel Crowns

(18-8) Austenitic types of alloy is used

For example Rocky Mountain and Unitek

- 17-19% Chromium
- 10-13% Nickel
- 67% Iron
- 4% Minor elements.

The austenitic types provide the best corrosion resistance of all the stainless steel.

Nickel-Base Crowns

They are Iconel 600 type of alloy.

- 70% Nickel
- 14% Chromium
- 6-19% Iron
- 0.04% Carbon
- 0.35% Manganese
- 0.2% Silicon

These alloys have good formability and ductility necessary for clinical adaptation of crowns and wear resistance to resist opposing occlusal forces. The metallurgical characteristics of nickel-chromium crown allow these crowns to be fully shaped and strain hardened without a defect during manufacture.

OBJECTIVES

According to Full, Walker, and Pinkham the objectives of stainless steel crown restorations are:

- To achieve biologically compatible, masticatory competent and clinically acceptable restoration.
- To maintain the form and function and where possible, the vitality of the tooth should be maintained.

The advantages of stainless steel crowns are:

- More durable and inexpensive.
- Reduction in operating time at chair side.
- These stainless steel crowns are far superior to multi-surface amalgam restorations with respect to both life span and replacement also and a most advantageous system of restoration because of its retention and resistance.
- Stainless steel crowns are acceptable to both the parents and dentist.
- They are also more cost effective because of comparatively simple procedures involved in restoring even severely affected primary molars.

INDICATIONS

Extensive Caries

Restoration of primary molars requiring large multi-surface restoration in children with rampant caries. If large approximal lesions are left untreated for a long time, the adjacent teeth will move into the space, resulting in a loss of arch length. The timely placement of a stainless steel crown restores the natural contour of the tooth while minimizing this space loss.

Stainless steel crowns are more cost effective than multisurface silver amalgams in primary teeth because fewer teeth require retreatment.

Extensive decay in primary and permanent teeth, where the caries on three or more surface or caries extent beyond the anatomic line angles, e.g. caries on mesial surface of the maxillary and mandibular first molar. The proximity of the pulp on the mesial side make placement of an acceptable amalgam restoration difficult.

Primary incisors with class IV and class V lesions occurring mesially and distally, i.e. the primary anterior teeth that is extensively decayed from the nursing bottle syndrome.

Developmental Enamel Defects

In patients with amelogenesis or dentinogenesis imperfecta or those who have extensive enamel hypoplasia of an acquired nature often complain of pain when eating. The enamel on these teeth is quickly worn away, exposing the more sensitive dentin. Placement of silver amalgam restorations in these teeth requires an unconventional type of preparation, which generally fails

in the long-term. To prevent failure, a stainless crown should be fitted as soon as possible after eruption of these teeth. Due to the extreme fragility of the enamel, great care must be exercised when fitting a stainless steel crown to these teeth so that large flakes of enamel are not dislodged.

Another indication for the use of stainless steel crowns as intermediate restorations is in children with a severe class II division I malocclusion but whose first permanent molars are severely hypoplastic or grossly carious. In this situation the mandibular first molars can be extracted to allow the second molars to drift anteriorly. The maxillary first permanent molars should then be restored with stainless steel crowns. When the second molars erupt, the first molars can be extracted and the premolars moved into the available space with an appliance.

Following Pulp Therapy

Croll and Killian recommended stainless steel crown as the treatment of choice for teeth that have undergone Pulpotomy, assuming there is a minimal leakage in crowned teeth compared to those restored with amalgam. Kopel states that following pulp therapy a correctly fitted stainless steel crown is probably the restoration most likely to give an adequate seal and thus eliminate subsequent bacterial contamination caused by micro leakage.

As a Preventive Restoration

If the patient has a high susceptibility to caries and in a handicapped child whose lack of oral hygiene may encourage further decay. For instance, development class V lesion is a sign of poor oral hygiene and cariogenic diet. When this occurs in the preschool age child who also has class II lesion in the same tooth, the stainless steel crown is indicated particularly in the first primary molar.

For Replacing Prematurely Lost Anterior Teeth

Double stainless steel crowns on abutment teeth can be used for replacing the lost maxillary anterior teeth.

Harrison JD reported crown and bridge preparation, design and use as for replacing missing anterior teeth. Wright, Cashion and Hoover reported the esthetic stainless steel crown bridge replacing prematurely lost or missing anterior teeth in young patients.

The esthetic stainless steel crown bridge is relatively easy to fabricate and offers an additional treatment modality for replacing missing anterior teeth in specific patient population.

For Temporary Restoration of a Fractured Tooth

According to McDonald and Avery stainless steel crown is one of the most stable restorations for the temporary protection of the fractured tooth. If the coronal fracture is extensive with a vital pulp exposure (especially Ellis class III), this crown may be the temporary restoration of choice to provide the protection and space maintenance required during the tooth initial recovery.

A stainless steel crown restoration may be the practical choice until the patient is in a better position to receive more complete dental care.

The principal disadvantages of the restoration include its unsatisfactory esthetic appearance and the inability to inaccurately evaluate pulpal responses during the recovery period without removing and usually ruining the crown. If skillfully placed, however, this restoration will protect the fractured tooth in harmony with the adjacent and opposing tooth.

Single Tooth Cross Bite

Croll and Lieberman reported the "reverse" stainless steel crown is a well-known method of correcting single tooth anterior crossbites. When an elongated preformed incisor stainless steel crown is luted with the lingual surface facing labially, the resulting slope contacts the opposing mandibular incisors and normal occlusal forces move the incisors out of crossbite relationship. The reverse crown method corrects, "Dental tipping" type single tooth crossbite rapidly, comfortably, inexpensively, and without the need for special patient cooperation. Such treatment is also useful in certain cases of primary tooth anterior crossbite and was once used for treatment of a 10-month-old.

Two disadvantages of using reverse stainless steel crown are the unsightly silver appearance of the crown

form, and the limitation of working with an inclined slope that is already formed.

Severe Bruxism

According to Morris and Braham since the teeth of the primary dentition exhibit excessive wear, they can be restored with stainless crowns. This reduces the adverse effects of excessive masticatory forces on the early erupting permanent teeth.

In the mixed dentition phase, the stainless steel crown adapted to the primary molars will assist in preventing undue wear of the first permanent molars.

As an Abutment for Space Maintainer (Fig. 15.3)

Stainless steel crowns are often used as abutments in space management appliance when the use of bands is contraindicated from the presence of extensive carious lesions.

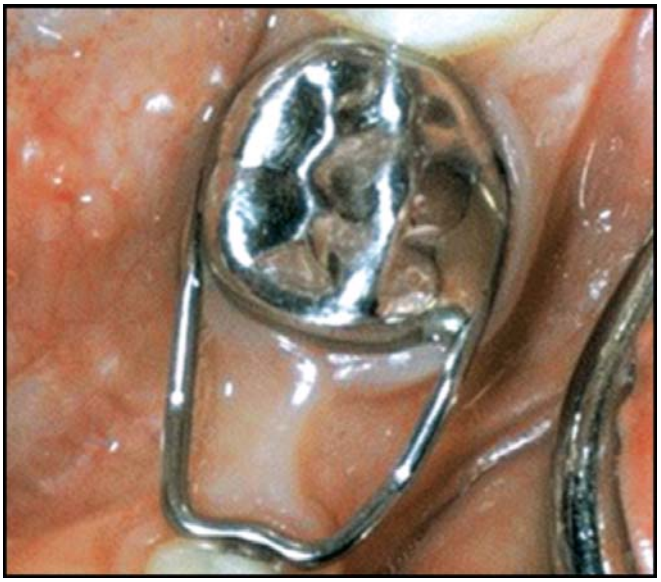


Fig. 15.3: As an abutment for space maintenance

Abutments to Prosthesis

Stainless steel crowns are useful extracoronal restorations on the abutment teeth to a removable prosthesis in children. This overcomes any problems caused by poor oral hygiene and prevents secondary caries occurring in these teeth.

CONTRAINDICATIONS

According to Nash:

1. Primary posterior teeth in which conservative amalgam restorations can be placed. Quality of amalgam restorations continues to be the preferred restoration in many primary molars.
2. *Exfoliation:* Teeth expected to exfoliate within a brief period (6 to 12 months) and radiograph shows over half the primary root resorbed.
3. *Abutments for space maintainers:* The preformed crown should be considered as a means of restoring a primary tooth, not as a method of fabricating a space management appliance. Bands can be placed on primary teeth to fabricate appliances to preserve arch circumference, a more conservative measure than reducing a tooth for crown placement. Even when the adjacent tooth requires crown placement, it is advisable to maintain separate functions. A well-placed crown can have a band and loop device cemented to it rather than have the loop directly attached to the crown.

   When the space management device has served its purpose, it can be removed readily, leaving the crown intact and undamaged. The use of crowns as abutments for space maintainers can result in poor adaptation of the crown to the tooth to accommodate the demands of the space maintainer. In addition, cutting the space maintainer from the crown leaves a roughened surface, a nidus for plaque development.
4. *Poor esthetics in the anterior teeth:* Restoring fractured or missing anterior teeth using stainless steel crowns usually not accepted both parents and patients due to unesthetic metallic appearances.
5. *Cost:* The cost-effectiveness of any restoration should be considered in treatment planning; in many instances, a temporary restoration can be placed in molars approaching exfoliation.
6. *Periodontal problems:* In primary molars and incisors gingival inflammation may arise with improperly adapted stainless steel crowns. In permanent molars periodontal pocket deep may increase and bone loss can occur on improperly adapted stainless steel crowns.

7. *Patients with nickel allergies:* Placement of stainless steel crowns on these patients cause a localized and systemic allergic reaction.

CLINICAL PROCEDURE

Factors to be Considered in Preoperative Evaluation

Dental Age of the Patient

This is recorded by the root development of the underlying tooth. When a primary tooth can be expected to exfoliate within 2 years of restoration, amalgam restoration can be done. However, failure of extensive amalgam restoration in the primary teeth can be frustrating. This can be overcome by an initial placement of stainless steel crown.

Cooperation of the Patient

If the child is stubborn and does not want to cooperate, first a positive behavior has to be instilled. If the child is unable to cooperate because of age (i.e. < 3 years), then a chair side general anesthesia should be considered. In this case, since it is difficult to check the correct occlusion, it is always better to keep the stainless steel crown at the level or slightly below the level of the adjacent tooth, so that the child does not have disturbed occlusion due to premature contact.

Motivation of the Parents

Whether the parents are willing to come for dental visits and for the follow-up.

Medically Compromised and Disabled Children

For example in children with a heart problem, antibiotic prophylaxis has to be taken as in tooth reduction and subgingival procedure. In poor general condition of the child chair side GA has to be taken into account.

Procedure

Armamentarium

Burs and stones

- No.169 or No.69L FG
- No.6 or No.8 RA
- No.330 FG
- Tapered diamond FG
- Green stone or heatless stone
- Wire wheel.

Pliers/Instruments

- No.115 pliers
- No.114 pliers (Johnson contouring pliers)
- No.800-417 crown pliers (Unitek corp)
- No.112 ball and socket pliers (optional)
- Sharp scaler or instrument-American No.7
- Crown and bridge scissors
- No.110 Howe pliers
- No.138 pliers (Gordon contouring pliers)
- Paper pad and glass slab
- A gate spatula
- Dental floss
- Rubber dam armamentarium
- Rough or white polishing wheels
- Articulation paper
- Local anesthetic.

Selection of Crown

The correct size crown may be selected prior to the tooth preparation by the mesiodistal dimensions of the tooth to be restored, and a Boley gauge can be used for this purpose.

If the crown is not selected before the tooth reduction, after the tooth reduction it can be selected as a trial and error procedure which approximates the mesiodistal width of the crown. The smallest crown that completely covers the preparation should be chosen. To produce steel crown margins of similar shapes, examine the contours of the buccal and lingual marginal gingivae.

- Buccal and lingual marginal gingivae of the second primary molar resembles smiles with greatest occlusal-gingival height of the clinical tooth crown about midway on the buccal and lingual surfaces.
- Buccal marginal gingivae of the most mandibular first primary molar and many maxillary first molars are similar to a stretched out 'S', having greatest occlusal-gingival height located at the mesiobuccal.

- The contour of the lingual marginal gingivae of all the first primary molars resembles a smile.
- The occlusal-gingival height is located about midway in buccolingual direction.

Tooth Preparation

Evaluate the Preoperative Occlusion

- Make an alginate impression of upper and lower dental arch of the patients.
- Pour the cast in the dental stone.
- Note the dental midline and the cusp fossa relationship bilaterally.

Local Anesthesia

According to Full, Walker, Pinkham preparation of a tooth for a stainless steel crown requires rigorous instrumentation with high-speed carbide but; a procedure begun without deep anesthesia may result in severe compromises in quality. Because of the possible impingement of soft tissue by the insertion of the crown before festooning and the possibility of pain caused by the probing of the gingival crevice with an explorer. The clinician may choose to ensure anesthesia of the gingival tissues.

In the maxilla, an infiltration of the buccal and occasionally on the palatial side of the tooth are required if pulp therapy is planned. It is not necessary to place the anesthetic gel on the palatial side over the apex of the tooth.

According to Stephen Wei, it can be placed in the loose soft tissues adjacent to the tooth from the buccal side after anesthetic gel has begun to produce anesthesia. In the mandible, inferior alveolar nerve block is given supplemented by an infiltration of the long buccal nerve.

According to Duggal MS, Gautam SK, Robertson local anesthesia is given for tooth preparation involving the gingival margins, which can cause some discomfort. Sometimes, it is possible to use a topical anesthesia, such as a benzocaine ointment on the gingival cuff.

Isolation

Rubber dam: It is not only advantageous from a behavioral and access standpoint, it is also very much an advantage in preventing the aspiration of loose crown. Because of the danger of aspiration, the rubber dam should be utilized whenever possible during the fitting of the steel crown form. When it is not possible to utilize the rubber dam, as in the case of crowning the terminal tooth in the arch, gauze oral screen should be used to prevent the inadvertent loss and possible aspiration of a crown form.

Use of rubber dam for isolation is mandatory. When it is not possible to use rubber dam, as in case of terminal teeth in arch, cotton rolls, which are held in position by cotton roll retainer or a gauze oral screen should be used to prevent the possible aspiration of a crown.

1. Before placing a rubber dam, check the child's occlusion. Observe the following:
   - The opposing tooth has extruded due to longstanding carious lesions.
   - There has been mesial drift due to carious lesions changing the occlusion of the adjacent tooth.
   - Tooth reduction is needed so that the restored tooth can be returned to normal function.
2. Use of a rubber dam in preparing a tooth for a stainless steel crown for the following reasons:
   - To protect surrounding tissue
   - To improve visibility and efficiency.
   - For better behavior management.
   - To prevent ingestion of the stainless steel crown during preparation.
3. One can alter the rubber dam by cutting the interproximal rubber to avoid cutting the dam with rotating instruments. Wedges can also be used to protect the dam and tissue. An alternate method is to punch a large hole and slip it over the most posterior tooth to the tooth receiving the stainless steel crown. Then stretch the dam forward to the canine area.

Remove the decay with large round bur in a slow speed handpiece. After caries removal and pulp therapy, if necessary, the previously carious area can be built up

with a quick setting reinforced ZnOE cement and/or $ZnPO_4$ cement.

Recommendations for Preparation

How much occlusal reduction should be done?: Though various views have been expressed regarding the occlusal reduction it is found that about 1.5-2 mm of reduction has to be done to obtain occlusal clearance. However, as much of the tooth structure as possible must be left for retention.

Which surface should be reduced first, proximal or occlusal?: If the proximal reduction is done at the initial step, even with utilization of wedged rubber dam, some amount of gingival bleeding will occur. If blood gets on the preparation, it will make the diagnosis of very small pulp exposure difficult.

Thus the best plan is to reduce the occlusal as the initial step, removing any caries as part of that step. Next perform the necessary pulp therapy then proceed with proximal surface reduction.

Considerations for retention capability of the crown: It is not only mechanical preparation which is required for the retention but the cementing medium also plays an important an important role for the retention of stainless steel crown.

According to More and Pink, retention is primarily created by contouring the crown and adapting its margins to the tooth. Mathewson et al used five different dental cements to retain stainless steel crowns on extracted primary molars and found that little or no mechanical retention unless cement was used. They concluded that retention is attributed to the adhesiveness of the cements.

Special recommendations for preparation of tooth: One has to concentrate on making the stainless steel crown more physiologically acceptable to the gingival as it is seen in our clinical practice and also that cement increases the retentive capacity of all types of preparations. Reducing supragingival bulge with reduction extending 0.5 mm below the gingival crest helps to obtain an acceptable gingival response.

Aims of Tooth Reduction

- To provide sufficient space for the steel crown.
- To remove the caries.
- To leave sufficient tooth for retention of the crown.

Steps in Stainless Steel Crown Procedure

- Occlusal reduction
- Proximal reduction
- Buccal and lingual reduction
- Evaluation criteria for tooth preparation
- Selection of crown
- Initial adaptation and retention
- Festooning of crown
- Crown contouring
- Crown crimping
- Crown finishing
- Crown polishing
- Final adaptation
- Response of gingival tissue to SSC
- Radiographic confirmation of the gingival fit
- Cementation
- Postoperative instructions.

Occlusal Reduction (Fig. 15.4)

A conventional, small pear-shaped carbide bur such as the No.330 is adequate for all instrumentation except for the removal of large masses of decay and the preparation of grossly decayed axial contours. These

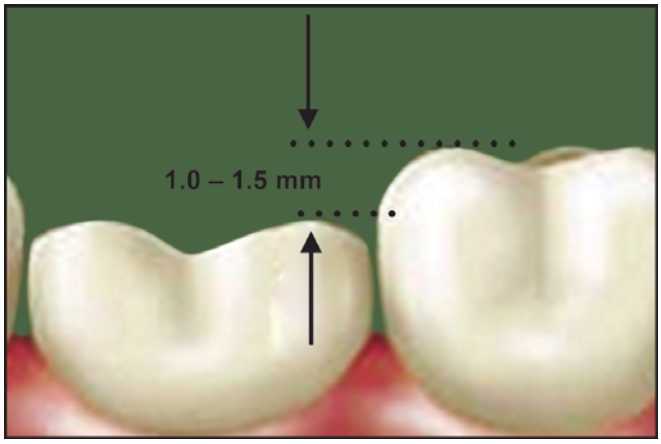


Fig. 15.4: Occlusal reduction

procedures call for the use of round burs and a flame-shaped diamond or a tapered fissure bur.

Both the side and the end of the No.330 bur are used to reduce the occlusal surface of the tooth about 1.5 mm. Because all aspects of the occlusal morphology are reduced in equal amount, the resultant occlusal table approximates the original contours and anatomy of that surface. The probability of a consistent 1.5 mm reference grooves (depth cuts) into the fissures and cusps of the tooth will aid in deciding the extent of occlusal reduction.

After gross reduction, a gingivally inclined bevel should be instrumented along the buccoocclusal and linguo-occlusal line angles. This removal of acute line angles will greatly facilitate the placement of the contoured stainless steel crown.

Humphrey recommended that the cusps be reduced, if necessary, and that the four sides of the tooth be reduced but as much tooth structure as possible be left for retention.

Rapp advises that the occlusal surface of the tooth be reduced so the height of the preparation is approximately 4 mm from the gingival margin. Mink and Bennett on the other hand, suggest a uniform occlusal reduction of 1 to 1.5 mm using a 1mm bur to make grooves in the occlusal surface to guide the reduction. Troutman recommended that the occlusal surface be reduced at least 1mm. Kennedy suggested that it can be reduced 1.5 to 2 mm.

Similar variations exist in the timing of the reduction of the occlusal surface relative to the interproximal reduction. However, the best plan seems to be to reduce the occlusal as the initial step, removing any caries as a part of that step, and subsequently perform whichever type of pulp therapy is deemed necessary before continuing with the preparation. The rationale behind this approach is that if the proximal surface is reduced as the initial step, even utilizing a wedged rubber dam, it will be likely encounter some gingival bleeding. If blood gets on the preparation, it will make the diagnosis of even a very small pulp exposure difficult.

The most common problem encountered in attempting tooth preparation for steel crowns is inadequate reduction. The recommendation of Kennedy to reduce the occlusal surface 1.5 to 2 mm is not excessive and should definitely be considered. Whether to use a large round bur, a tapered fissure bur, a diamond wheel, or a flame-shaped diamond stone to accomplish this reduction is irrelevant and should simply be determined by the preference of the operator.

Proximal Reduction (Figs 15.5A and B)

The second step in the process of preparing the tooth for a stainless steel crown should be interproximal reduction. It has been observed that may of the difficulties encountered in placing a stainless steel crown are the result of attempting to fit a round or oval crown form over a rectangular tooth preparation. Irregularities, projections, or sharp angles on the circumference of the

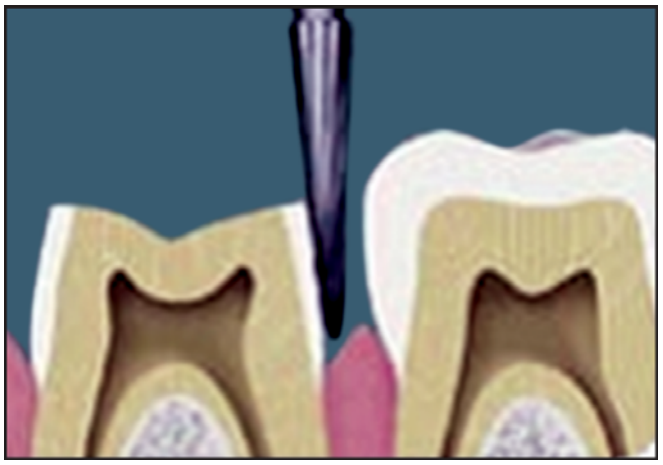


Fig. 15.5A: Proximal reduction

Fig. 15.5B: Ledge on proximal surface prevents seating of the crown, if detected it should be removed with a tapered bur

prepared tooth will prevent the crown form from being properly seated, will cause time consuming repeated adjustments, and will prevent the crown form from properly fitting the tooth preparation. The primary principle of the technique for fitting stainless steel crowns is to make the tooth preparation fit the crown form rather than attempt to make the crown fit the tooth preparation.

By examining the crown form, prior to preparation of the tooth, the crowns of all manufacturers are somewhat oval and rhomboid. This confirms to the rhomboid shape of the primary tooth. In accomplishing the interproximal reduction therefore, be careful to maintain that form in the preparation.

By beginning on the lingual and following the contour of the proximal surface of the tooth, one can more easily accomplish an even and uniform reduction of the surface, thereby maintaining this rhomboid shape. Making a slice also helps to eliminate the interproximal ledge, which seems to be the most frustrating problem in the restoration of a tooth with a stainless steel crown. Beginning the slice at the marginal ridge with the no.691 or 169L bur not only will result in the frequent formation of a ledge but also will rapidly dull and wear out the tip of the bur.

It is important to maintain the shape of the tooth, because errors sometimes made in the preparation of the proximal slices include preparing the slices parallel to each other or reducing the buccal aspect of the tooth excessively. Thus the shape of the tooth is destroyed and a preformed crown cannot be adapted satisfactorily. The slice should pass through the contact and terminate slightly below the gingival tissue on the enamel portion of the tooth. An undercut area of enamel should remain in the cervical portion of the tooth.

The proximal slice should allow the trimmed and contoured crown to seat and snap into the retentive area. The margin of the crown will be adapted in the retentive area at the gingival portion of the tooth. Over reduction in vertical plane may produce an excessively tapered tooth. Additionally, ledges may be created on the tooth, which will prevent the crown from seating. Precaution should be taken in the proximal reduction because but may damage adjacent tooth while preparation. To prevent damage stainless steel bands and wooden ledges are used.

If the bur is angled toward the tooth, it will produce an excessively tapered preparation. If the bur is angled away from the tooth, the adjacent tooth may be damaged.

To confirm that there is enough clearance between the preparation and adjacent teeth, an explorer can be used. If the space is adequate, it should be possible to pass the shank of the instrument through the space. Cut a bevel around all of the cut edges of the preparation at an angle of about 30° and then increase this angle to achieve a 60° bevel. This process must be repeated all the way around the occlusal aspect of the preparation, and also the mesiobuccal, mesiolingual and the distobuccal and along the distolingual line angles.

Buccal and Lingual Reduction

The third step in the preparation concerns the reduction of the buccal and lingual surfaces. This area seems to be the most controversial.

The question is whether to:

- Reduce the entire buccal and lingual surfaces and remove, if not the entire bulge, at least a significant portion of it.
- Permit the buccal and lingual cervical bulges to remain and reduce only the occlusal third of the preparation. (Mink and Bennett).

Evaluation Criteria for Tooth Preparation

- The occlusal clearance is 1.5 to 2 mm. (A sheet of wax may be used to indicate area, of insufficient reduction).
- The proximal slices converge toward the occlusal and lingual, following the normal proximal contour.
- An explorer can be passed between the prepared tooth and the proximal tooth at the gingival margin of the preparation.
- The buccal and lingual are reduced at least 0.5 mm with the reduction ending in a featheredge 0.5 to 1 mm in to the gingival sulcus.
- The buccal and lingual surfaces converge slightly toward the occlusal.
- The occlusal third of the buccal and lingual surfaces is gently rounded.

- All point and line angles in the preparation are rounded and smoothed.

Selection of Crown

Two general ways of selecting the crown size appropriate for the prepared tooth are measurement of the tooth and trial and error. The measurement of the tooth before preparation may significantly expedite the inexperienced clinician's choice. The experienced clinician will probably use the trial and error method. Crown should be the smallest one that can be inserted over the cervical convexities of the tooth with the operator's finger pressure.

The placement of a stainless steel crown over a prepared tooth is best accomplished by use of either a lingually or buccally based path of insertion. This method permits the initial contact of the crown's buccal or lingual perimeter to be gingival to the tooth's greatest convexity, and permits the crown to be inserted over the remaining convexities of the tooth. This technique of crown placement minimizes the chance of distorting the festooned crown's gingival perimeter beyond its elastic yield limit.

Initial Adaptation and Retention

The flattened proximal surfaces combined with rounded line angles should form a somewhat oval rhomboidal in preparation. This greatly aids in rapid crown adaptation because of the shape of the steel crown forms.

Rapp stated that the retention of the stainless steel crown restoration originates form contact between the tooth and the margins of the crown. Milk and Bennett reported that it is seldom necessary to reduce the buccal and lingual surfaces of the crown except on the buccal surface of the mandibular primary first molar or where an abnormal bulge of enamel may be present. Humphrey advocated leaving as much tooth structure as possible, when preparing a tooth to be crowned to aid retention. The rationale for maintaining this bulging tooth structure is that it will contribute to the retention of the crown.

Full et al hypothesized that retention is attained by elastically deforming the crown into the undercut areas of the primary tooth. They recommended conservative preparation of the tooth's cervical perimeters to retain undercut areas of the primary tooth. Myers suggested a preparation that retains the general morphology of the tooth. He argued that the cervical portion of the tooth the retained so that the metal crown will "snap" into place. Henderson noted that it is frequently impossible to obtain sufficient retention without placing the margin of the crown subgingivally.

Festooning of Crown

The establishment of the proper gingival extension of the crown and the adaptation of the gingival third of the crown to the tooth are possibly the most difficult clinical objectives of the technique. Use an explorer to carefully inspect the adaptation of the crown's gingival edge to the tooth cementoenamel junction. This test of fit should be done around the entire perimeter of the crown. Efficient use of this technique requires a tactile sense that develops with experience.

The first step in festooning is the achievement of an appropriate occlusogingival dimension for the crown. Care should be taken not to over reduce this dimension. Over reduction will compromise the crown's retention by incomplete utilization of the cervical undercut. The preferred instrument for trimming of the crown is the curved crown and bridge scissors.

Crown Contouring

Initial crown contouring is performed with a 114 pliers (ball and socket pliers) in the middle third of the crown to produce a belling effect. This will give the crown a more even curvature. Contouring of proximal metal surface is not done with these pliers as they are already in contact with the adjacent teeth. Though occasionally, No.112 (Abell) pliers is used to contour the proximal surface to establish correct contact. Adaptation of the gingival third of the crown is done with the 137 Gordon pliers.

Crown Crimping (Figs 15.6A and B)

Any marked gingival crimping of the crown can also be done with Unitek 800-412 pliers. The tight marginal fit aids in:

1. Mechanical retention of the crown.

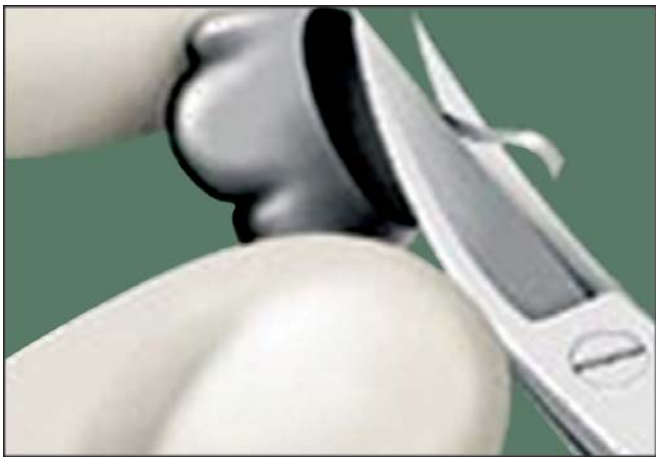


Fig. 15.6A: Crown trimming

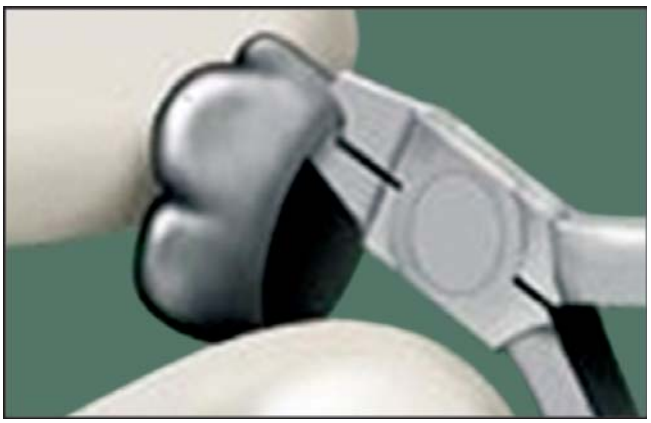


Fig. 15.6B: Crown crimping

2. Protection of the cement from exposure to oral fluids.
3. Maintenance of gingival health.

During the trial flitting and cementation, the crown should be placed form lingual and rolled towards buccal surface. In this way maximum undercut on the buccal surface is more easily covered.

Crown Finishing

1. Use a large green stone to make a knife edge finish at the cervical margin. Operate the handpiece in such a manner that the burs and shavings are spun to the inside of the stainless steel crown. This technique should aid in retention.
2. Smooth and polish the margins with a rubber wheel.
3. Polish the entire crown with a wire brush. Rouge, whiting or a fine polishing material can be used to give the crown a fine luster.
4. Remove the rubber dam if the crown is to be cemented with the dam off.
5. Try on the crown and check occlusion. Evaluate the opposite arch for proper cuspal and occlusal interdigitation.
6. Check the mesial and distal contacts. If they need expansion use No.112 pliers. It may be necessary to add silver solder to the contacts if the pliers do not work.

Crown Polishing

Accumulation of the plaque and inflammation of gingiva is commonly seen in practice of restorative dentistry due to rough and unpolished restoration. To avoid these complications, the crown should be polished prior to cementation with a rubber wheel to remove all scratches.

While polishing the crown, margin should be blunt since knife-edge finish produces sharp ends which act as areas of plaque retention. A broad stone wheel should run slowly, in light brushing strokes, across the margins towards the center of the crown. This will draw the metal closer to the tooth without reduction of the crown height and thus improves the adaptation of crown.

A wire brush can be used to polish the margins to a high shine. To give a fine luster to crown, rouge, whiting or a fine polishing material can be used.

Final Adaptation

Spedding reported that two principles related to stainless steel crown length and crown margin shapes that are based on an understanding of the tooth morphology and gingival tissue contours.

1. The crown should be of a correct length and its margins can be adapted closely to the tooth. This can be achieved when the finished crown is correctly seated on the prepared tooth with its occlusal surface in the occlusal plane and its margins placed just apical to the marginal gingival crests.
2. After the correct size crown is placed on the prepared tooth, the crown height can be reduced by removing

about 1 mm of the crown initially with a crown and bridge scissors or with a caborundum wheel on a slow speed straight headpiece, which shapes the margins simultaneously.

3. For shaping the crown margins, sickle scaler is used to mark 3 light points on the metal at the mesiobuccal, buccal and distobuccal and at the mesiolingual, lingual and distolingual surfaces at the crest of respective marginal gingival without compressing the marginal gingiva. This mark on the metal corresponds to the greatest diameter of the tooth. Final finished margins are placed approximately 1 mm below these marks. The correctly shaped finished crown margins are parallel to the contours of the marginal gingival of the tooth, about 1.0 mm into the gingival crevice.

Checking the Final Adaptation of the Crown

1. The crown must snap into place.
2. It should not be able to be removed with finger pressure.
3. The crown should fit so tightly that there is no rocking on the tooth. Moderate occlusal displacement forces at the margin do not displace the crown.
4. The properly seated crown will correspond to the marginal ridge height of the adjacent tooth and is not rotated on the tooth.
5. Crown is in proper occlusion and should not interfere with the eruption of the teeth.
6. There should not be any high points when checked with articulating paper.
7. The crown margin extends about 1 mm gingival to gingival crest.
8. No opening exists between the crown and the tooth at the cervical margins. Crown margins closely adapted to the tooth and should not cause gingival irritation.
9. Restoration enables the patient to maintain oral hygiene.

Radiographic Confirmation of the Gingival Fit

According to Myers before cementation, a bitewing is taken to verify proximal marginal integrity. If the crown is too long, there is still an opportunity to reduce the length. If it is too short, then add an orthodontic band or adaptation of another crown is indicated.

Cementation

The luting cements for stainless steel crowns have undergone tremendous improvements. Traditionally zinc phosphate, polycarboxylate, and reinforced zinc oxide eugenol have been used to cement die stainless steel crowns to seal the crown margins. However, newer materials with superior physical properties of adhering to tooth structure and releasing fluoride are now available.

Myers et al reported that a cavity varnish must be routinely applied before a stainless steel crown is cemented on a vital tooth. Shiflett and white determined that dentin bonding agent, resin-modified glass-ionomer, adhesive composite resin cement and glass-ionomer cement significantly reduced microleakage compared to the traditional cements. This could enhance the clinical efficacy of all forms of stainless steel crowns.

Zinc Phosphate Cement: Zinc phosphate cement is formed by mixing zinc oxide with phosphoric acid. Zinc phosphate cement is used mainly for luting or mechanically locking a restoration by filling in voids and defects. It is used primarily with stainless steel crowns. A second use is for cementing stainless steel bands for space maintainers. Zinc phosphate cements are easily handled and manipulated and have many years of clinical use.

Zinc Oxide-Eugenol Cements: Zinc oxide-eugenol cements consist of a mixture of zinc oxide and eugenol. The zinc oxide-eugenol cements are used primarily for cavity base in deep lesions such as those that require indirect pulp capping.

Zinc oxide-eugenol cement is extremely compatible with pulpal tissue as it may stimulate healing with secondary dentin formation. According to Phillips, it possesses low strength values and is very soluble in oral fluids. It is used as the sub-base for pulpotomies, with or without Formocresol added.

Reinforced Zinc Oxide-Eugenol Cements: The reinforced zinc oxide-eugenol cements contain additives to the liquid (eugenol) or powder (zinc oxide). These additives can

be resins, accelerators or minerals. The improved zinc oxide-eugenol cements can be used for cementation of stainless steel crown on vital teeth, as cavity liners in deep lesions (as in indirect pulp capping procedures), and for provisional restoration in prevention programs in children with rampant caries.

The reinforced zinc oxide-eugenol cements are less soluble in oral fluids than are zinc oxide cements. Phillips reported that the reinforced zinc oxide-eugenol cements have greater strength than zinc oxide-eugenol but less strength than zinc phosphate cement. These cements are used for final cementation of cast gold restorations. The main advantages of these cements are their minimal pulpal irritation, easy manipulation, and optimal margin-sealing properties. They have adequate strength and can be used to cement stainless steel crowns in vital primary teeth. Unfortunately, these materials are soluble in oral fluids, cause gingival inflammation, and have minimal mechanical luting properties.

Polycarboxylate cements: Polycarboxylate cements consist of a mixture of zinc oxide powder and a polyacrylic acid liquid. Polycarboxylate cements have a minimally irritating effect on the pulp, the same as zinc oxide-eugenol. These cements have been used to directly bond stainless steel orthodontic brackets to enamel. Polycarboxylate cements have higher bond strength than either zinc phosphate or improved zinc oxide-eugenol cement. However, this strength is not related to increased physical properties such as tensile strength, compressive strength, or film thickness.

The main advantage of polycarboxylate cements is the low irritant factor to oral tissue. There is some adhesion to tooth substance and stainless steel alloys. Other physical properties are similar to those of zinc phosphate cement. The disadvantages are the requirements for precise proportioning and optimal manipulation, plus the need for a clean, uncontaminated tooth surface.

Glass ionomer cement: Glass ionomer cements are quite new and very promising. Their powder is aluminosilicate glass and their liquid a mixture of polyacrylic, itaconic and tartaric acids. Just as silicophosphate is a hybrid of silicate and zinc phosphate, the glass ionomers are a hybrid of silicate and polycarboxylate. These cements have comparable strengths with zinc phosphate, release fluoride as do the silicophosphates, chelate or bond to tooth structure as do their polycarboxylates, and are as pulpally compatible as the polycarboxylates. They could prove to be the best cement available for steel crown cementation. Disadvantages with the glass ionomers are their radiolucency and the present lack of long-term clinical efficacy.

Steps in cementation (Figs 15.7 and 15.8):

1. Apply Lidocaine (Xylocaine) ointment or petroleum jelly to contact areas before cementation to assist in cement removal after cementation.
2. Use cotton rolls to isolate the quadrant containing the tooth to be restored. Every effort should be made to prevent postoperative sensitivity. If the tooth is vital, place a liner such as Dycal (LC Caulk Co.) in the deep portions of the crown preparation, followed by Copalite (LD Caulk Co.)

Fig. 15.7: Crown cementation

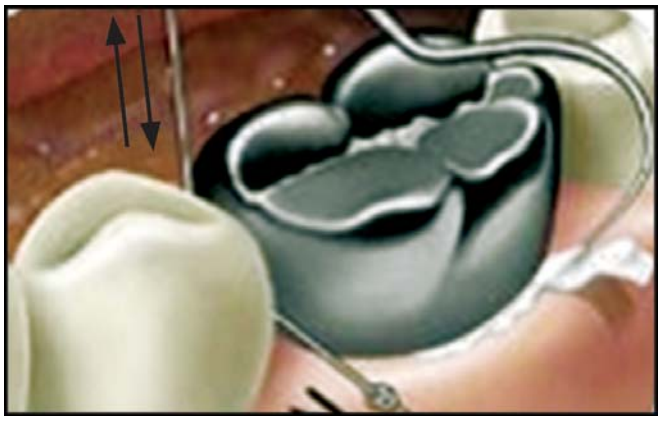


Fig. 15.8: Removal of excess cement

3. Zinc phosphate is still commonly used for cementation of stainless steel crowns. Cool the glass slab thoroughly under cold water and dry it with a clean towel. Place powder on one end of the slab. In the middle of the slab measure out 5 drops of liquid for each unit to be cemented. Use the spatula to divide the powder into small increments that are approximately 3 mm on a side. Move on increment across the slab and incorporate it into the liquid, mixing it for 20 seconds across a wide area. Allow this first portion to set for approximately 1 minute before continuing. This setting time will aid in neutralizing the acid. Continue to add small increments of powder, mixing each for 10 to 20 seconds, using a circular motion and covering a wide area of the slab. Check the consistency by picking up the cement on the spatula and holding it over the slab. If the cement is in the right consistency, it will string out slightly between the spatula and slab before it runs back onto the slab. If it runs quickly off the spatula, it is too thin; if it must be nudged off the spatula, it is too thick.
4. Fill the crown with cement.
5. Seat the crown, usually first on the lingual side and then the buccal side. Make sure it is firmly seated. Support the child's mandible with one hand as you seat the crown with the other.
6. If the tooth is isolated with cotton rolls, place a strip of Burlew dry foil over the crown to help to keep the tooth free from moisture until the cement sets.
7. Remove excess cement with a scaler or explorer. Polish the crown with acidulated phosphate fluoride prophylaxis paste. Gently but firmly check all the areas of the gingival sulcus for retained cement. Excess cement can produce gingival inflammation and discomfort.

Postoperative Instructions

1. Regular diet may be resumed after anesthetic effects are worn off.
2. It is not unusual for the gum tissue around the newly restored tooth to appear slightly irritated and inflamed for several days. Using salt-water rinses while irritation persists can ease this (1/2 teaspoon of table salt in 6 ounces of warm water). Brushing and flossing the gums offers no substitute.
3. Tylenol should handle any postoperative discomfort. However, please call if pain persists as a stronger medication can be prescribed.
4. Gum tissue around the tooth may bleed somewhat for upto several hours after the appointment. Should bleeding persist into the next day, please call our office.
5. The area around the crown should be brushed gently today, gradually increasing to normal toothbrush pressure in a few days. It is important to brush the area well; clean teeth will aid in fast healing of irritated gum tissue.
6. Stainless steel crown on primary teeth will come out with the primary tooth when the permanent tooth is ready to erupt.
7. Stainless steel crowns on permanent teeth may need to be replaced by a casted crown when the child is in his/her mid to upper teens or later in life.
8. The patient is instructed to brush the gingival tissue and to floss the teeth daily.

Myer's Criteria for the Evaluation of Crown Defects

Stainless steel crown is non-ideal if:

1. *Length:* The margin is short of the gingival crest or is extended below the cementoenamel junction.
2. *Crimp:* A space is detected between the margin of the crown and the tooth.
3. *Contour:* The walls appear flat and do not approximate normal contour.
4. *Contact:* A space exists between the crowned tooth and adjacent tooth.
5. *Position:* The crown is markedly rotated or has an unusual axial inclination.
6. *Polish:* There is evidence of plier marks or scratches.
7. *Cement:* Excess cement remains in the sulcus.

STAINLESS STEEL CROWN FOR PERMANENT MOLARS

There are problems involving permanent posterior teeth for which the stainless steel crown may provide the most

desirable short-term solution. In placing the crown, emphasis should be given recognition of the wide variation of available crowns, occlusal considerations, the importance of the rubber dam, and the use of pre-cementation radiograph to confirm marginal adaptation of the crown.

Indications

- The stainless steel permanent crowns can be used to make a useful long-term provisional restoration for a broken down first permanent molar that has been partially restored and must be kept under observation before construction of a cast restoration.
- The crowns are useful for restoring the occlusion and greatly reducing the sensitivity from enamel and dentine dyscrasias in young patients.
- When there are financial considerations regarding the need for a cast restoration, placement of a stainless steel crown may be considered as economical.

Radiological Considerations

Along with a preoperative diagnostic radiograph of the affected tooth and associated structures, precementation radiographs are essential to assess precise marginal adaptation of the crown by showing interproximal areas where marginal coverage is difficult to assess.

Anesthesia

According to Full, Walker, Pinkham preparation of a tooth for a stainless steel crowns requires rigorous instrumentation with a high-speed carbide bur; a procedure begun without deep anesthesia may result in severe comprises in quality. Because of the possible impingement of soft tissue by the insertion of the crown before festooning and the possibility of pain caused by the probing of the gingival crevice with an explorer, the clinician may choose to ensure anesthesia of the gingival tissues.

Isolation

The entire procedure should be done with the use of rubber dam. The major advantage of the rubber dam is that gingival marginal fit can be visualized around the entire circumference of the tooth being restored with the possible exception of the center of the proximal surfaces, which can be evaluated with a pre-cementation radiograph.

TOOTH PREPARATION

Occlusal Reduction

Initially, the tooth is reduced occlusally in a similar manner to the reduction for a cast gold crown. The general anatomical form of the crown in reduced dimensions should be maintained while assuring between 1 to 2 mm occlusal clearances in the entire envelope excursive movements. This is achieved readily with the barrel shaped diamond bur. The occlusal reduction is achieved first to facilitate better control and vision for the next step.

Proximal Reduction

The proximal slices eliminate all contact with adjacent teeth and create the space required to adapt the crown and to restore contact if indicated. Proximal preparation achieved with the flame-shaped diamond, or the 169 long carbide burs. It is helpful to place a wooden wedge or flattened round toothpick between the teeth to prevent interference of the rubber dam and to avoid laceration of the gingival.

Buccal and Lingual Reduction

In the next step, slightly reduce the convexity of the buccal and lingual surfaces of the tooth. It is important to reduce these surface convexities in the gingival third of the tooth so that stainless steel crown may assume the original convexity and thus produce an over contoured, enlarged buccolingual dimension.

A fine, feather-edged gingival margin at the crest of the gingival should be produced, which will be covered by a thin smooth edge of the crown. When caries extends subgingivally, the margin must extend subgingivally also to furnish full coverage of the preparation after complete caries removal. The edge of the crown must be designed to embrace securely the margin around the entire periphery of the tooth.

Selection

The measurement of the tooth before preparation may significantly expedite the inexperienced clinician's choice. The experienced clinician will probably use the trial and error method. The selected crown should be the smallest one that can be inserted over the cervical convexities of the tooth with the operator's finger pressure.

Crown Placement

After achieving proper orientation of the crown on the tooth, the marginal areas are critically examined. Only the areas immediately below the proximal contacts cannot be easily seen.

A pair of crown-crimping pliers is used to crimp the margin of the crown. These pliers scallop the periphery, which is then smoothened with a pair of contouring pliers.

The crimped crown is again seated on the tooth and the margins are re-examined visually and with the explorer. Any open area disclosed by this examination can be marked with an indelible, fine-pointed pencil or felt-tip marker to indicate where additional crimping and contouring may be necessary. When ideal adaptation has been achieved, the rubber dam is removed. The crown is reseated and occlusion is evaluated.

The use of a wooden tongue blade split lengthwise serves as an excellent bite stick for applying force in a particular area while seating the crown. Prematurity, coronal orientation, length of the crown, and stability of the restoration are all verified and deficiencies are corrected.

Radiographic Confirmation of the Gingival Fit

Before cementation, a bitewing radiograph is taken to verify proximal marginal integrity. If the crown is too long, there is still an opportunity to reduce the length. If it is too short, the add-on procedure or adaptation of another crown is indicated.

Final Finishing

After all occlusal and gingival adjustments have been accomplished, it may be necessary to re-crimp the crown as the metal may expand minutely each time the crown is seated and removed. The margins of the crown are then refined and smoothened with a greenstone and a large rubber wheel that removes all scratches. Final treatment of the margin can be accomplished readily by buffing with a rag wheel and then polishing with rouge. It is most important to thoroughly clean the interior of the crown with a wet cotton swab or small brush before cementation.

Cementation

The rubber dam is replaced and the tooth is cleaned and dried with a liberal water spray and gentle application of warm air. A creamy mixture of cement is prepared and the crown is then filled about three quarters full, making sure that all margins are covered. It is then seated on the tooth with gentle finger pressure or with a tongue blade and mild biting force. Removal of excess cement is important to prevent gingival irritation.

The stainless steel crown is a vital component of contemporary restoration procedures for the deciduous dentition. Because economy is one of the most advantageous characteristics of the stainless steel crown, instruments and instrument transfer should be minimal. The technique also should require conservative preparation of the tooth's cervical perimeter to usefully the retention that these natural undercuts afford.

One must be aware of the different anatomic and metallurgic characteristics of the specific crown form to be utilized. Appropriate adjustments must be made in the preparation of the tooth and the crown form must be manipulated accordingly. The described technique is consistent with the requirements of clinical efficiency and maintenance of natural tooth morphology.

The following criteria should be utilized to evaluate the completed steel crown restoration:

- The crown is smooth and polished.
- The crown margins are smooth and closely adapted to the tooth preparation.
- The contacts are properly established.
- The crown is in the proper occlusion.
- The crown extends about 1mm gingival to the gingival crest, and there is no gingival blanching.
- All excess cement is removed.

16

Oral Hygiene Education

INTRODUCTION

"Prevention is better than cure" is the emerging philosophy and becoming the first principle in the evolving trends in dentistry.

Though dental caries and periodontal diseases are considered to be multifactorial, it is well accepted that the main cause of both the diseases are dental plaque, which is largely preventable by elimination of colonization of microorganisms in dental plaque.

The above goal can be achieved by the child patient's participation in a comprehensive prevention program that includes effective daily plaque removal.

For any oral hygiene program to be successful, the child patient and parent must be motivated to perform effective plaque removal procedures on a continued and regular basis.

It is the prime duty of a dental health professional to outline a regime that is simple, effective and comfortable, requiring a minimal armamentarium and can be accomplished in a reasonably brief period of time. An oral hygiene regime should be individualized according to the needs and abilities of the child and parent; and it is the responsibility of the dental professional to provide information on the techniques and potential benefits of an oral hygiene regimen to aid the parent and child in achieving this goal.

Plaque control is brought about by:

a. *In office methods* that include professional oral prophylaxis that motivates home care procedures.
b. *Home care program:* It includes daily regime of brushing teeth daily in an appropriate systematic manner with use of adjuncts like flossing and rinsing practices, etc.

Mechanical removal of dental plaque is advocated by use of tooth brushes, floss, etc. However, chemical removal of plaque by means of mouth rinses are advised to adolescents and children who posses the dexterity to swish and spit the rinses.

TOOTH BRUSH

Tooth brush is the universally accepted oral physiotherapy device that promotes mechanical removal of dental plaque.

The parts of the tooth brush include:

- Bristle head:
  - Toe (extreme end of head)
  - Heel (closest to handle)
- Shank
- Handle

Classification

Based upon Type of Bristle Material

- Natural bristles
- Synthetic nylon bristles

Based Upon Mechanism of Action

- Manual tooth brush
- Powered tooth brush
- Semi tooth brush

Based Upon Bristle Texture

- Soft
- Medium
- Hard

Other Criterions

- Size
- Shape of head, etc.

ADA Specifications

ADA Specifications for Acceptable Brushes

• Length of head	1-1.25 inches
• Width	5/16-3/8 inches
• Surface area	2.54 to 3.2 cm
• Number of rows	2-4 rows of bristles
• Number of tufts	5-12 tufts/row
• Number of bristles	80-85 bristles/tuft

Acceptable Dimensions for an Ideal Pedodontic Brush

• Length of head	1 inch
• Width	5/16 inch
• Number of rows	2-3 rows of bristles
• Number of tufts	24-33 tufts

- Diameter of each nylon filament -0.007”-0.008”

Types of Designs in Arrangement of the Tufts

- Oral or convex
- Curved
- Straight
- Short oral
- Straight multitufted/serrated/non-serrated.
- Short bristles.

It is ideal for pedodontic tooth brushes to bare long handles with hand grips to improve the hold of the brush by the child.

The ADA Council on Dental Therapeutics (1984) does not recommend one specific tooth brush as being superior to another for removing dental plaque and controlling dental disease.

Once the appropriate tooth brush is selected for an individual child, the child/parent should be instructed about storage and maintenance of handling the tooth brush.

Guidelines for Tooth Brush Usage

- Brush teeth thrice daily.
- Rinse the brush thoroughly in running water before and after use.
- Store the brush in an open, ventilated area vertically.
- Ensure that bristles of one brush do not touch other brushes.
- Use appropriated sized caps for brush heads to prevent microbial and dust particle setting.
- Never leave the brush wet; it should be mildly moist.
- Never use another child’s brush.
- Replace the brush at regular intervals either once in 3 months or on frayed tooth brush head whichever is the earliest.
- A parent should always supervise and accompany the child during brushing. A parent is considered the best model for the child to teach tooth brushing.

Dental Floss

It is the most generally recommended device for the removal of interproximal plaque in non-spaced dentition. Floss is available in variety of types like waxed, unwaxed, tapes, flavored, etc. and floss holders of various designs are available to facilitate ease of use and manual dexterity.

Tooth Brushing Methods

The overall objectives of tooth brushing are to remove plaque and food debris and atraumatically stimulate the gingival tissues. A variety of tooth brushing methods aimed at achieving these objectives are described below. All involve the use of one of four basic brushing motions or combinations thereof. These include:

1. Horizontal reciprocating (scrub method)
2. Vertical sweeping (roll and physiologic methods)
3. Circular (Fones method)
4. Vibratory (Charters, Stillman and Bass methods)

Brushing techniques should be individualized according to the patient’s level of intelligence, co-operation and manual dexterity.

Scrub Method (Fig. 16.1)

- It is most commonly used technique.
- The bristles are applied at 90 degrees to the tooth surface and the brush is moved back and forth.
- The disadvantage of this technique is its inability to remove *plaque from interproximal areas* and also resultant abrasion and gingival recession.

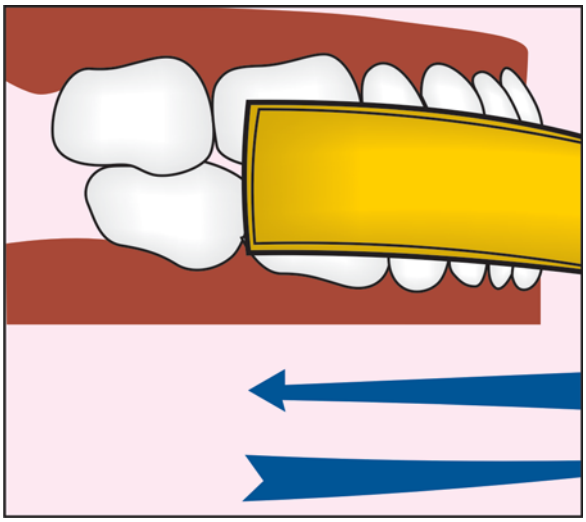


Fig. 16.1: Scrub technique

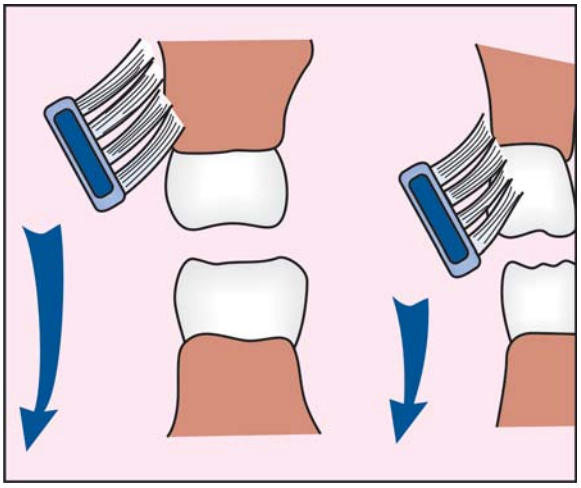


Fig. 16.2: Roll technique

Roll Technique (Fig. 16.2)

- Bristles are placed high on attached gingival apically at 45° angle.
- The sides of the bristles are firmly rolled against the gingival in a coronal direction to blanch the tissues momentously.

Physiologic Technique (1930)

- It requires a soft tooth brush and sweeping it from a coronal portion apically towards the gingival margin and the gingival.
- This method is so called as it is considered that action of brushing stimulates the passage of food over the crown towards the gingiva.

Fones Technique (1934)

- The brush is firmly pressed against the teeth and the gingiva with the bristles at right angles to the buccal surfaces and handles parallel to the occlusal plane.
- The patient occludes the teeth and the brush is moved in wide circular motion.

Charter's Technique (1928)

- It is primarily used to clean large interproximal spaces.
- The brush is placed at 45 degrees to the long axis of the teeth. The bristles are firmly forced into interproximal areas with a slight rotary and vibratory movement.
- The sides of the bristles should contact the gum margins producing a massaging action.
- The occlusal surface is brushed firmly with bristles in a slight rotary movement to force the bristles gently into pits and fissures.

Stillman Technique (Fig. 16.3)

- It is also termed as "Press and Roll technique".
- The technique begins with light pressure applied on gingival through bristles and then the bristles are rolled across towards the occlusal surface of the teeth.
- This cleans and vigorously massages the gingival tissues.

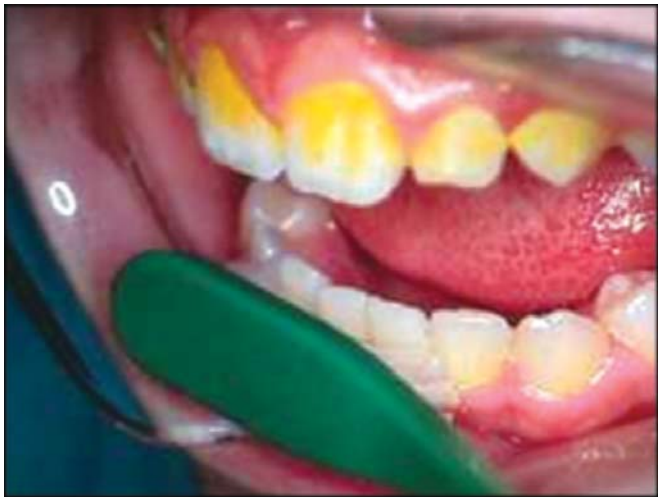


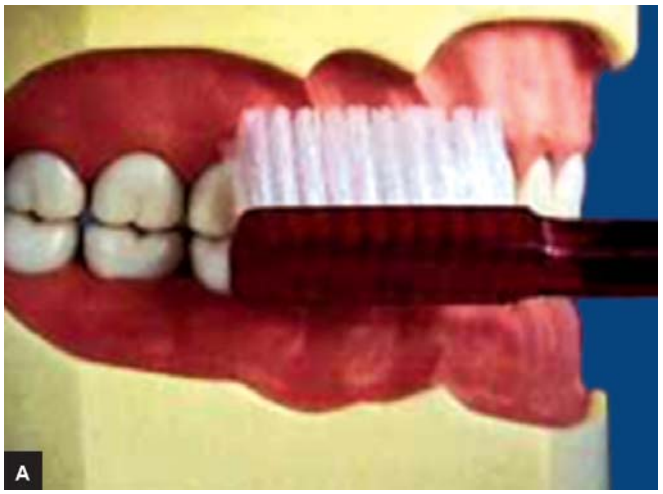
Fig. 16.3: Stillman's technique

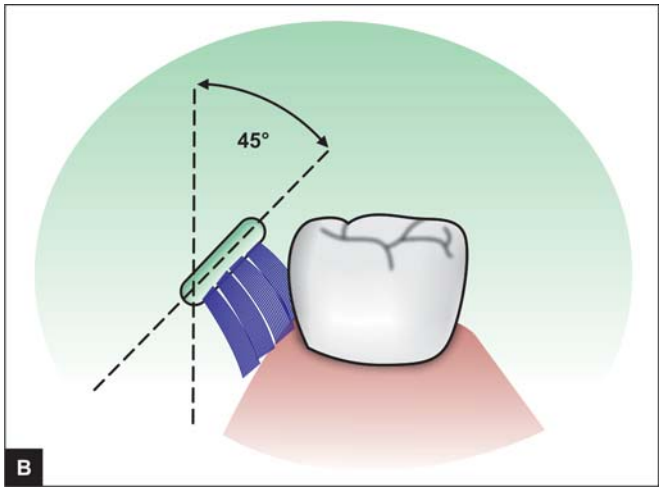
Bass Technique (1948) (Fig. 16.4)

- The brush is placed at 45 degrees angle to the long axis of the teeth and the bristles are gently forced into gingival sulcus and the interproximal area and moved in short back and forth strokes with a vibratory action for 1-15 seconds for each area.
- The occlusal surfaces are brushed with short anteroposterior strokes.

Modified Bass Technique

- This method combines the vibratory motion of the bass technique with the vertical sweeping strokes of





Figs 16.4 A and B: Bass technique

the roll technique and short vibrating horizontal strokes.

- It provides better plaque control in the interdental areas.

ORAL HYGIENE REGIMEN FOR CHILDREN

A successful oral hygiene regimen includes following components:

Assessment of Patient and Parent Dexterity

- Patient dexterity varies from child to child based upon factors like
  - Age
  - Physical strength
  - Emotional/mental status
  - Disabilities if any.
- Parent dexterity is nothing but training and educating the parent to assist the *child in performing homecare in maintaining oral hygiene.*

Disclosing Plaque (Fig. 16.5)

- Use of plaque disclosing agents is employed in education and motivation of the child and parent participating in the oral hygiene regimen.
- It helps to point out areas of plaque retention to both the child and parent before and after brushing and helps to reinforce their brushing technique if needed.
- Some of the plaque disclosing agents available are as follows:
  - Skinners solution.
  - Bismarck Brown (Easlick's disclosing solution)
  - Merbromin.
  - Erythrosine (F D and C Red No.3 or No.28)
  - Fast green.
  - Fluorescein
  - Two-tone (Thicker plaque stains blue; thinner plaque stains red).

Fig. 16.5: Plaque disclosing agent

- Meruchrome.
- Proflavin.
- Tartarazine
- Plaklite, etc.

A Systematic Approach to Brushing

Advise the patient and parent to follow a systematic pattern in brushing the teeth. Katz, McDonald and Stookey have described the concept of **"circuits of brushing"** as a systematic approach to tooth brushing which involves dividing the dental arches into buccal, lingual and occlusal circuits.

The patient or parent is instructed to start in same area of mouth and proceed through the circuits in same fashion every time the teeth are cleaned, so that none of the surfaces is missed out during brushing.

Time Devoted to Brushing

- A minimum 3 minute period is probably required to cover the tooth surfaces to be cleaned with an appropriate number of brushing strokes.

Frequency of Brushing

- It is the most essential feature.
- The most commonly recommended frequency is thrice daily regime for high-risk patients while twice daily regime for patients at moderate or minimal risk.

Appropriate Brushing Technique

Brushing technique varies for different age groups in compliance to their manual dexterity, etc (Table 16.1).

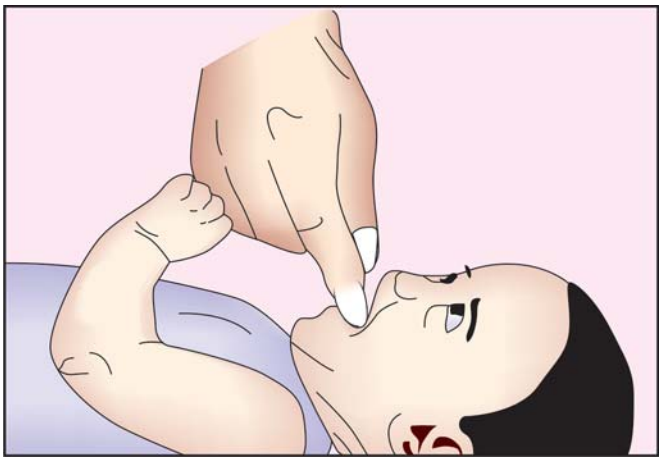


Fig. 16.6: Infant oral care

Figure 16.6 shows a diagrammatic representation of infant oral hygiene

AAPD GUIDELINES ON PERIODICITY OF EXAMINATION AND ORAL HEALTH CARE

Birth to 12 Months

- Complete the clinical oral examination with appropriate diagnostic tests to assess oral growth and development, pathology, and/or injuries, provide diagnosis.
- Provide oral hygiene counseling for parents and care givers, including the implications of the oral health of the care giver.
- Remove supragingival and subgingival stains or deposits as indicated.
- Assess appropriateness of feeding practices, including bottle and breastfeeding and provide counseling as indicated.

Table 16.1: Brushing techniques for different age groups

Age group	*Brushing technique*
Infants	Use moistened gauze pads to clean the gum pads
Toddler	Use moistened gauze pad initially followed by use of dampened soft bristle brush
Pre-school child (3 years)	Scrub technique
Early school child (3 years to 7 years)	Modified bass and roll technique
Pre-adolescent child (7 to 12 years)	Fones technique
Adolescent	Bass technique

- Provide dietary counseling related to oral health.
- Provide counseling for non-nutritive oral habits.
- Provide required treatment and/or appropriate referral for any oral disease or injury.
- Provide anticipatory guidance for parent/guardian/caregiver.
- Consult with the child's physician as needed
- Based on evaluation and history, assess the child's risk for oral disease.
- Determine the interval for periodic re-evaluation.

12 to 24 Months

- Repeat birth to 12 month procedures or as indicated by individual patients risk status/susceptibility.
- Assess appropriateness of feeding practices, including bottle, breastfeeding, and no spill training cups, and provide counseling as indicated.
- Review patients fluoride status-including any child care arrangements, which may impact systemic fluoride intake and provide parental counseling.
- Provide topical fluoride treatments every 6 months or as indicated by the individual patients need.

2 to 6 Years

- Repeat 12 to 24 month procedures every 6 months or as indicated by individual patients risk status/susceptibility to disease. Provide age appropriate oral hygiene instructions.
- Complete a radiographic assessment of pathology and /or abnormal growth and development, as indicated by individual patients needs.
- Scale and clean the teeth every 6 months or as indicated.
- Provide pit and fissure sealants for primary and permanent teeth as indicated.
- Provide counseling and services as needed for Orofacial trauma prevention.
- Provide assessment/treatment or referral of developing malocclusion as indicated.
- Provide required treatment and/ or appropriate referral for any oral diseases, habits or injuries as indicated.
- Assess speech and language development and provide appropriate referral as indicated.

6 to 12 Years

- Repeat 2 to 6 years procedures every 6 months or as indicated by individual patients risk status.
- Provide preventive therapy as indicated.
- Check on developing malocclusions and their immediate care.
- Assess and reinforce age appropriate oral hygiene instructions.
- Attend upon patient's dental needs as indicated.

12 Years and Older

- Repeat 6 to 12 years procedures every 6 months or as indicated by individual patient's risk status/susceptibility to disease.
- At an age determined by patient, parent/guardian and pediatric dentist, refer the patient to a general dentist for continuing oral care.

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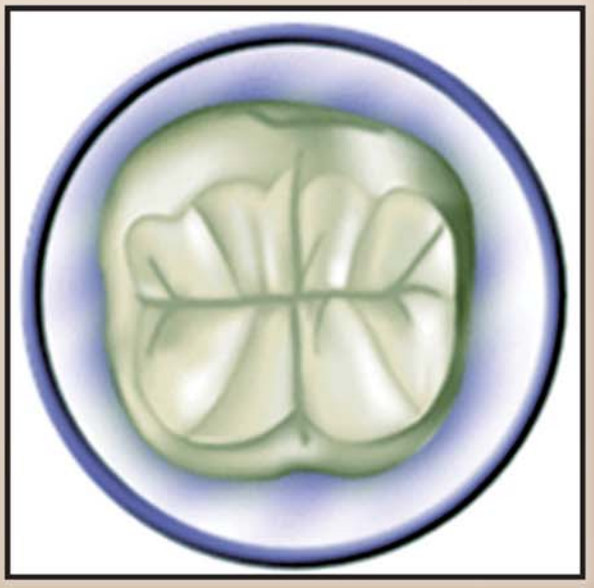


Preventive Dentistry: Occlusal Sealants

INTRODUCTION

Occlusal sealants are considered as an effective caries reducing agent when proper patient selection and application techniques are followed.

Scientific evidences shows that sealants are effective in both preventing caries and in arresting the progression of caries continue to mount. Today occlusal sealants are the most effective and important preventive measure available for the prevention of caries in children and adolescents.

Though fluorides are highly effective in reducing the number of carious lesions occurring on the smooth surfaces of enamel and cementum, unfortunately they are not effective in protecting the occlusal pits and fissures.

PREVALENCE OF OCCLUSAL CARIES IN CHILDREN

Caries potential is directly proportional to the shape and depth of the pits and fissures.

The high prevalence and rapid onset of occlusal caries is multifactorial in nature.

- Narrow isolated crevices and grooves that harbor food and microorganisms are the most important anatomical features leading to development of occlusal caries.
- Inaccessibility of the area to any mechanical means of debridement (Fig. 17.1).

MILESTONES OF PIT AND FISSURE SEALANTS

Hyatt-1923 - Proposed **"Prophylactic odontotomy"**
- Non-carious fissures prepared and restored with silver or copper oxyphosphate as soon as it erupts and when fully erupted a small occlusal cavity is prepared and restored with amalgam

Bodecker-1929 - Proposed **"Fissure eradication".**
- Mechanical eradication of fissures making them easily cleansable.

Buonocore-1955 - Proposed the method of adhering resin to an acid etched enamel surface

Mid 1960's Use of sealants

Bowen and Associates-1965 - Developed B-GMA resin and its use as sealant

OCCLUSAL SEALANTS IN PRIMARY TEETH

There are certain features that need to be kept in mind about sealant therapy in primary teeth as it varies from that in permanent teeth.

- The differences in the structure of the primary enamel compared to permanent tooth enamel effects the bond strength.
- Prismless enamel in primary teeth accounts for lower retention rates.
- Need for an extended etching time.
- More importantly is the difficulty of sealant application is isolation and moisture control in treating the young child.

This fact emphasizes the importance of technique in the sealant procedure.

TERMINOLOGIES TO KNOW

Pit is defined as a small pin point depression located at the junction of developmental grooves or at terminals of these grooves. The central pit describes a landmark in the central fossae of molars where developmental grooves join (Ash 1993).

Fissure is defined as **deep clefts between adjoining cusps**. They provide areas for retention of caries producing agents. These defects occur on occlusal surfaces of molars and premolars with tortuous configurations that are difficult to assess from the surfaces. These areas are impossible to keep clean and highly susceptible to advancement of carious lesion (Orban's 1990).

MORPHOLOGY OF FISSURES

Nango (1960) in a study of crown sections described four principal types of fissures based upon alphabetical description of share.

- V-type
- I-type
- U-type
- K-type

CLASSIFICATION OF PIT AND FISSURE SEALANTS

Mitchell and Gordon (1990) classification classifies sealants as follows:

Based Upon Polymerization

- Self activation
- Light activation.

Based Upon Resin System Used

- Poly urethanes
- Cyanoacrylates
- Bisphenol A-glycidyl methacrylate (Bis-GMA).

Based Upon Fillers

- Unfilled
- semi-filled
- filled.

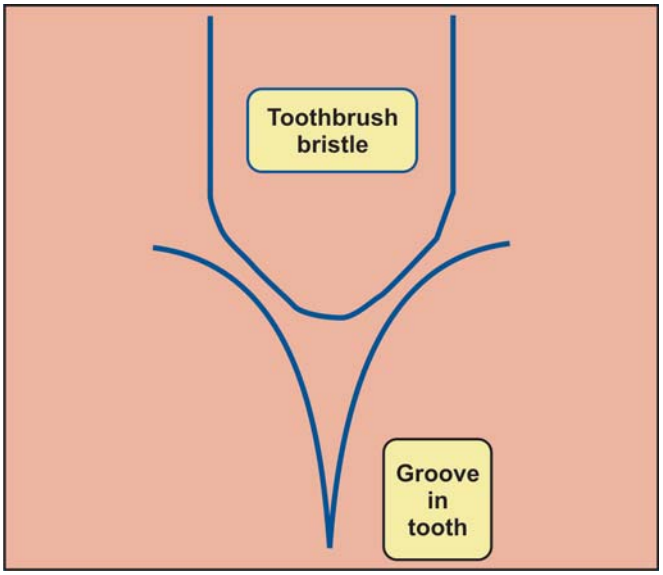


Fig. 17.1: Inaccessibility of pits and fissures to cleaning

Based Upon Color

- Clear
- Tinted
- Opaque.

REQUISITES OF AN EFFICIENT SEALANT (BRAUER 1978)

- A viscosity allowing penetration into deep narrow fissures.
- Adequate working time.
- Rapid cure.
- Good and prolonged adhesion.
- Low sorption and solubility.
- Resistance to wear.
- Minimum irritation to tissues.
- Cariostatic action.

A sealant is indicated if:

- A deep occlusal fissure, fossa or lingual pit is present, especially if it catches the tine of the explorer.
- An intact occlusal surface is present where the contra-lateral tooth surface is carious or restored, this is because teeth on opposite sides of mouth are usually equally as prone to caries.

A sealant is contraindicated if:

- Patient behavior does not permit use of adequate dry field techniques throughout the procedure.
- There is an open occlusal carious lesion.
- Caries exist on other surfaces of the same tooth.
- A restoration is already present.

A sealant is probably indicated if:

- The fossa selected for sealant placement is well isolated from another fossa with a restoration.
- The area selected is confined to a fully erupted fossa, even though the distal fossa is impossible to seal due to inadequate eruption.
- There is an incipient lesion in the pit and fissure; this decision would be a matter of professional judgment.

Guidelines for sealant use in individual care program (Fig. 17.2).

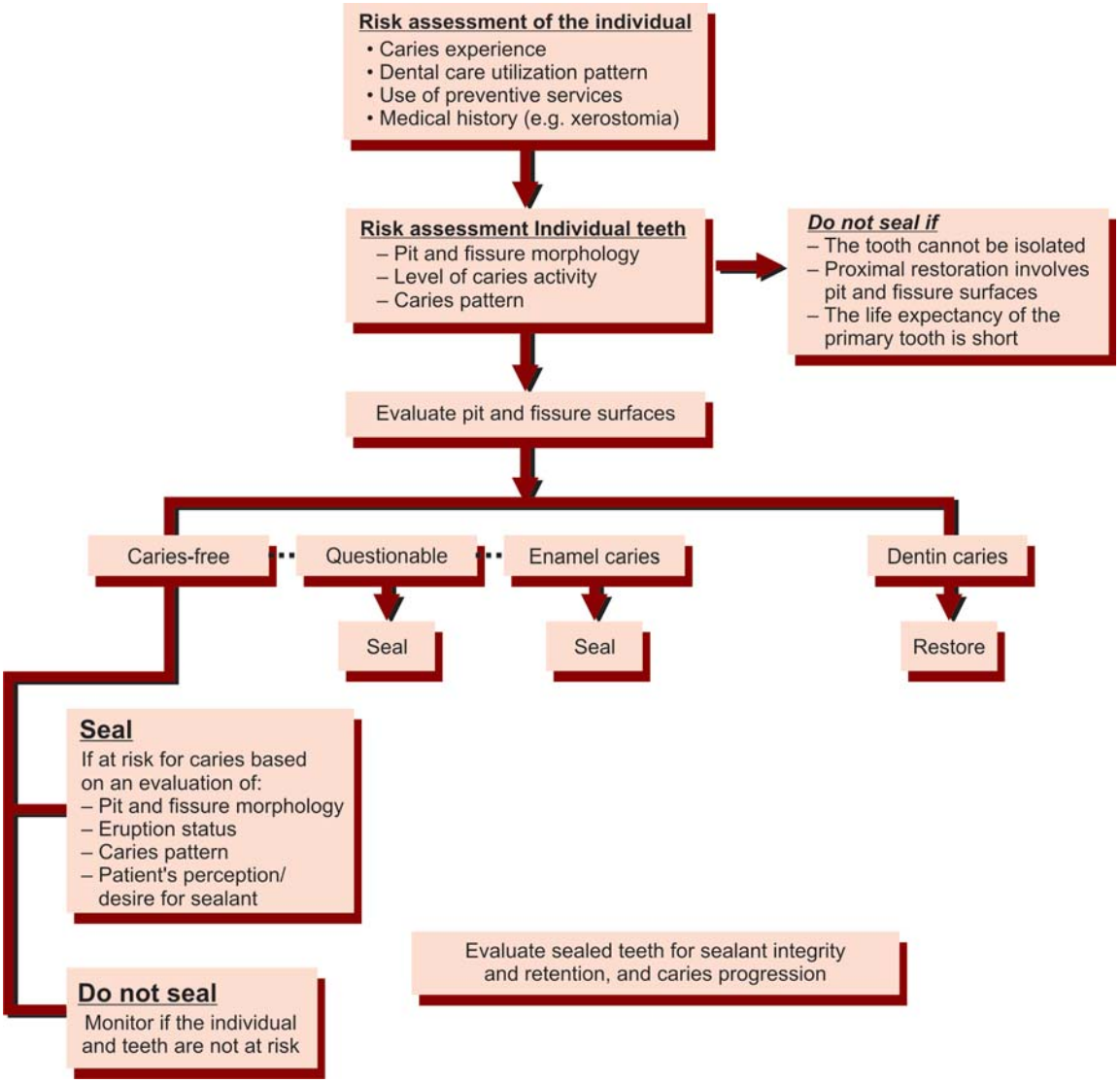


Fig. 17.2: Guidelines for sealant use in individual care program

OCCLUSAL SEALANT TECHNIQUE

Occlusal sealants are defined as the application and mechanical bonding of a resin material to an acid etched enamel surface thereby sealing existing pits and fissures from the oral environment. This mechanism prevents bacteria from colonizing in the pits and fissures and nutrients from reaching the bacterial already present.

Age and Tooth Selection

3-4 years	Primary molars
6-7 years	First permanent molars
11-13 years	Second permanent molars

Simensons Recommendations

- Group 1: Caries free patients judged at no risk to decay.
- Group 2: Patients judged to be at moderate risk to decay.
- Group 3: Patients with rampant caries at a high-risk to decay.

Clinical judgment for sealant application is based upon:

- Age of the patient
- Oral hygiene status
- Level of caries activity
- Life expectancy of the primary teeth
- Eruption status
- Pit and fissure morphology
- Ability to isolate and control moisture
- Fluoride environment and history, etc.

Placement Technique

Step I: Isolation (Fig. 17.3)

- Moisture contamination is greatest cause of sealant failure.
- Maintenance of a dry field is critical in sealant retention.
- Isolation is achieved with cotton rolls or rubber dam.
- After adequate isolation is obtained, the surface is dried and re-examined for presence of any carious lesion, if any caries is suspected; placement of a preventive resin restoration would be treatment of choice.

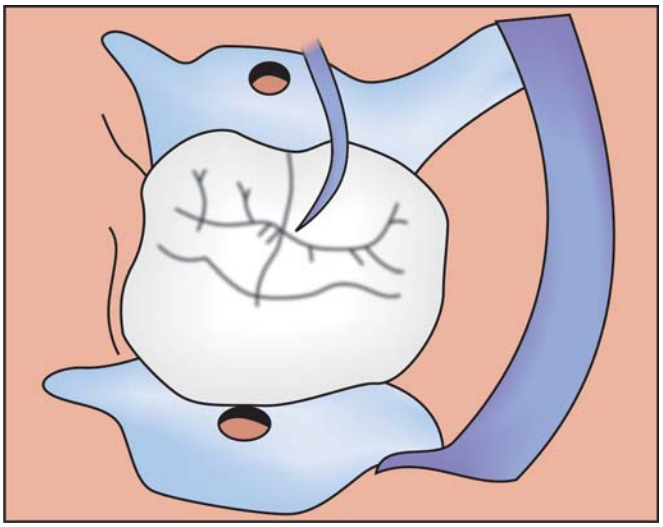


Fig. 17.3: Isolation and inspection

Step II: Tooth Cleaning and Preparation (Fig. 17.4)

- A prophylaxis is performed with pumice or non-fluoride paste-pumice paste and the tooth is rinsed with water thoroughly.

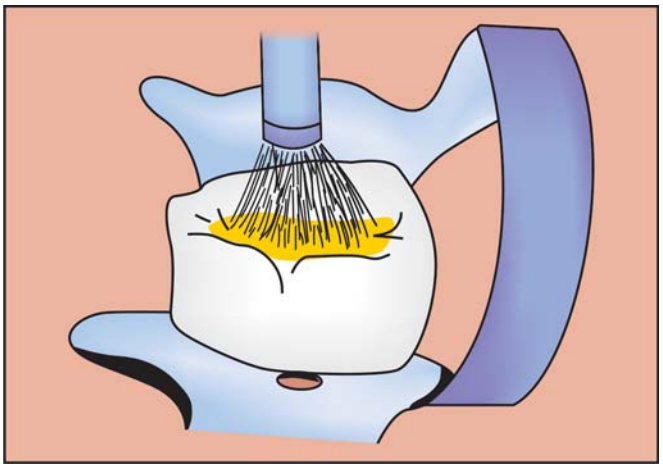
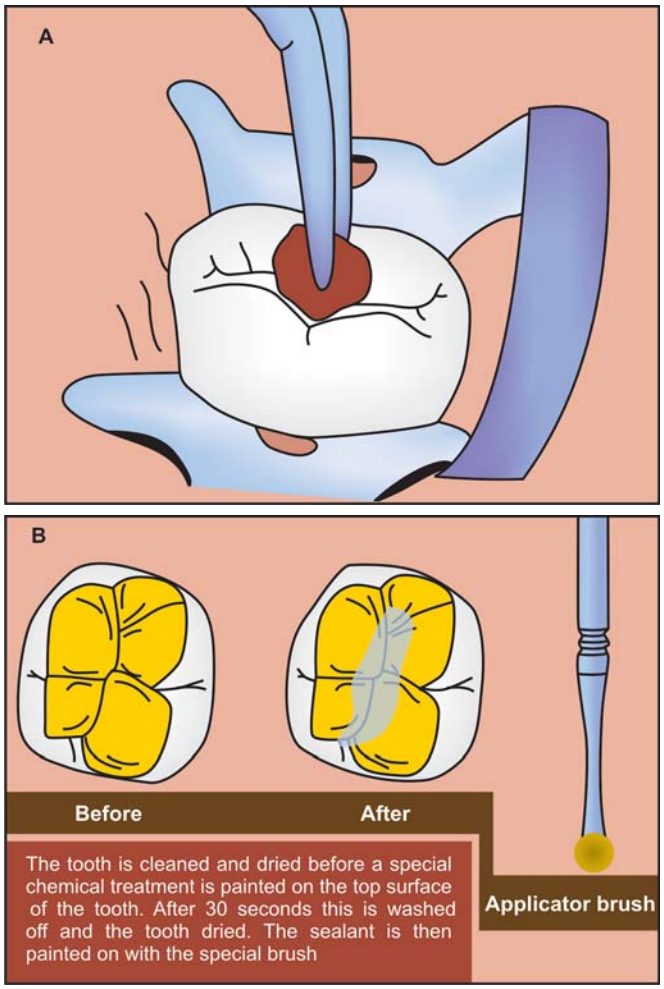


Fig. 17.4: Tooth cleaning and preparation

- Regardless of the means of prophylaxis only the inclined cuspal planes are cleaned.
- Little, if any debris is removed from fissure sites using an explorer tip.
- An effective alternative would be to eradicate the pits and fissures with a dental bur.

Step III: Etching (Fig. 17.5)

- Etching produces microscopic porosity with the depth of etch approximately 50 microns (0.05 mm).
- 30-40% buffered phosphoric acid solution is applied to occlusal enamel surface using a continuous application of fresh acid using a cotton pellet or application tips.
- Etching time-20-60 secs.
- It is essential that a wet surface be maintained by applying additional conditioner not allowing the solution to dry on surface.
- A gel is preferred over solution.
- Etchant should be placed only over the area that is to receive the sealant; generally the entire occlusal surface upto cusp tips (Fig. 17.6).
- Histologic perspective of etching reveals itself on the enamel surface as 3 different zones depending upon depth of penetration of etchant which is best illustrated (Fig. 17.7).



Figs 17.5A and B: Etching the tooth surface

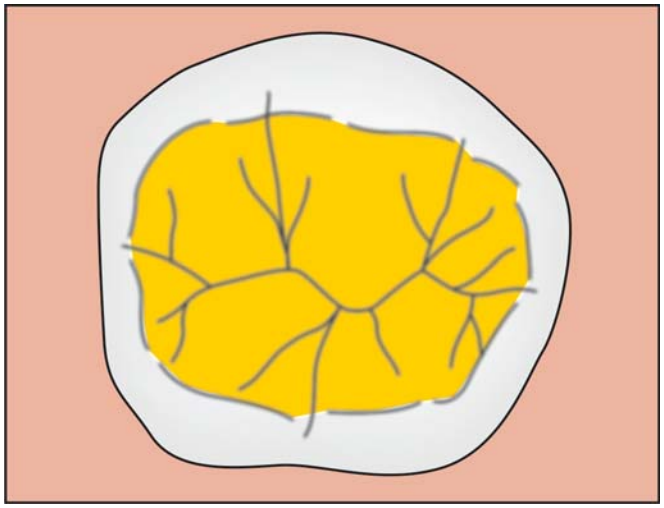


Fig. 17.6: Occlusal outline for etchant application

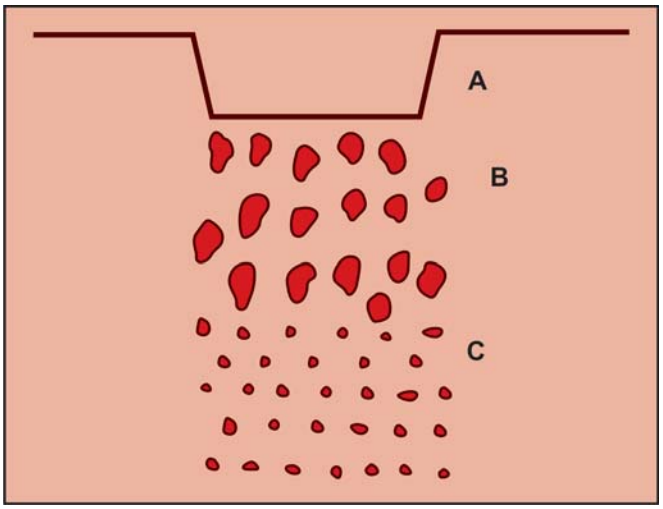


Fig. 17.7: Zones of etching. A. Etched zone, B. Qualitative zone, C. Quantitative zone

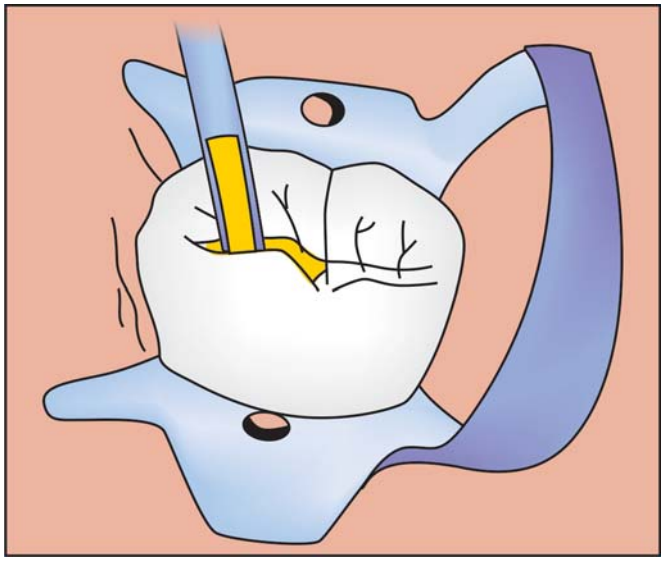


Fig. 17.8: Sealant application

Step IV: Rinse

- The enamel surface is thoroughly rinsed with water for 10-20 seconds and then air dried for 10 seconds, being careful not to use an oil contaminated air syringe.
- Do not wipe etched surface with cotton pellet.
- Look for frosty white appearance of enamel, if not re-etch for another 20 seconds.

Step V: Sealant Application (Fig. 17.8)

- The sealant is carried to the surface and applied smoothly by slowly depressing the lever. The sealant is allowed to flow ahead into crevices as the tip is advanced from one end of the tooth to the other. This method minimizes entrapment of air bubbles better than in brush on technique.
- Ensure that the sealant has sufficiently flowed into all grooves, if not using a explorer tip spread into narrow grooves that are not filled.
- When the sealant is adequately flown into all pits and fissures polymerize it using the light source.

Step VI: Evaluation (Figs 17.9A to C)

- Check for set.
- It should include an attempt to remove sealant with an explorer to determine if adequate bond strength is established.
- Also check for air bubbles if any.
- The use of this immediate test of retention has resulted in excellent future retention rates because potential failures are identified and rectified early.

Step VII: Occlusal Evaluation/Adjustment

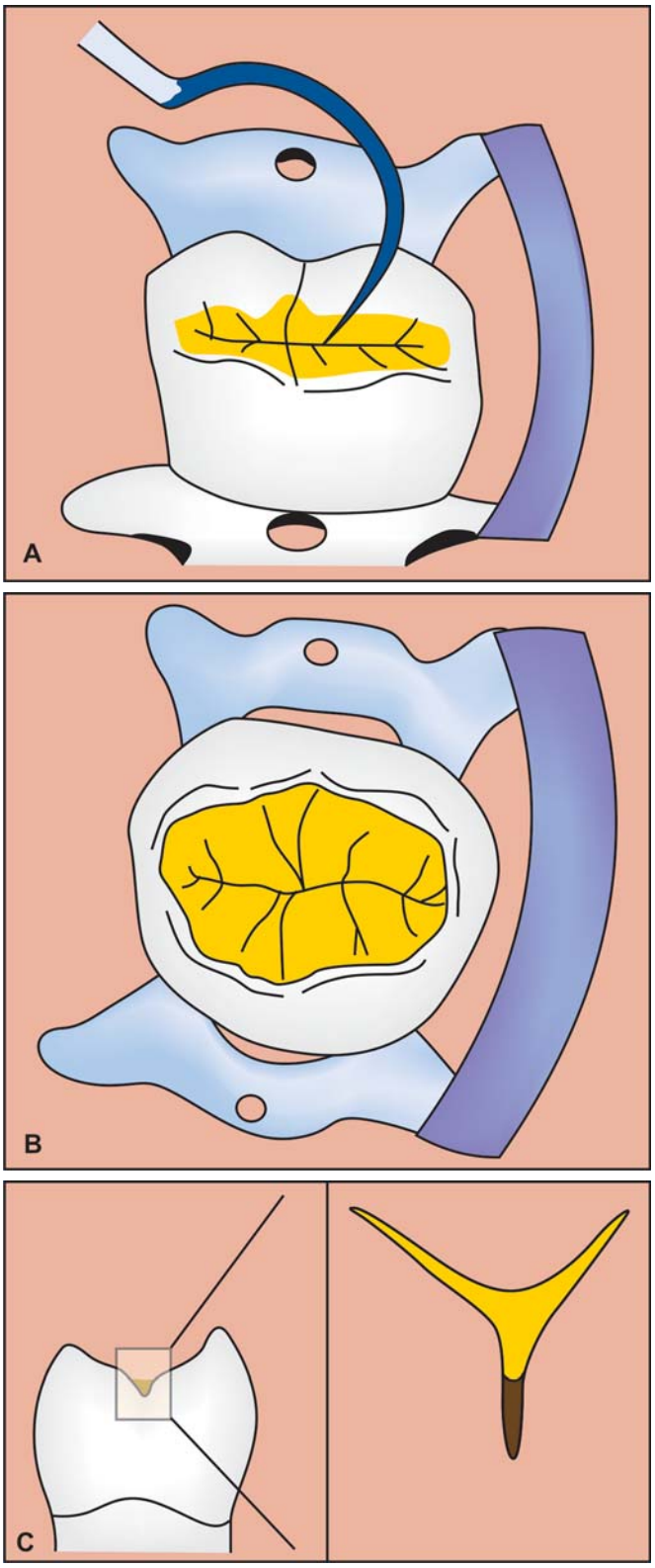
- Check occlusal interferences using articulating paper.
- A better option to reduce the chances of overfilling is by checking occlusal interferences before starting the sealant technique (i.e. before isolation) thereby while sealant application care can be taken not to involve or extend into areas of interference before curing; thereby minimizing the need for later adjustment.

Step VIII: Re-evaluation

- Sealant should be examined for loss once in every 6 months.
- Bitewing radiographs are important in the diagnosis of caries progression under the sealant if micro leakage has occurred since placement.

PREVENTIVE RESIN RESTORATIONS

The preventive resin restoration is a natural extension of the use of occlusal sealants. It integrates the preventive



Figs 17.9A to C: Evaluation. A. Using an explorer, B. Occlusal view of restored pits and fissures. C. Restored fissure cross sectional view

approach of the sealant therapy of caries susceptible pits and fissures with the therapeutic restoration of incipient caries with composite resin that occur on the same occlusal surface.

There are three types of preventive resin restorations based on the extent and depth of carious lesions as determined by exploratory preparation.

According to Simensons Classification (1978b, 1978c)

Type A : Comprises of suspicious pits and fissures where caries removal is limited to enamel.

Type B : It is defined as an incipient lesion in dentine that is small and confined.

Type C : It is characterized by the need for greater exploratory preparation in dentine and may require local anesthetic administration with placement of a suitable cavity liner over exposed dentinal surface.

Placement Technique

Type A Restoration (Fig. 17.10)

1. Clean the tooth surface.
2. Isolate optimally using rubber dam or cotton rolls.
3. Remove decalcified pits and fissures using bur.
4. Place etchant gel over entire occlusal surface for 20-60 seconds.
5. Wash thoroughly for 20 seconds with water and dry the tooth sufficiently.
6. Apply the sealant carefully and polymerize.
7. Occlusal adjustment is done wherever needed.

Type B Restoration (Fig. 17.11)

1. In such situation prior to application of resin, bonding agent is applied and cured on acid etched surface.
2. After polymerizing the bonding agent filled resin is packed in small increments and cured in multiple layers to account for polymerization shrinkage.
3. Care should be taken not to pack the entire cavity with filled composite; the superficial occlusal surface is restored by application of filled sealant and then polymerized.
4. Finally necessary occlusal adjustments are made.

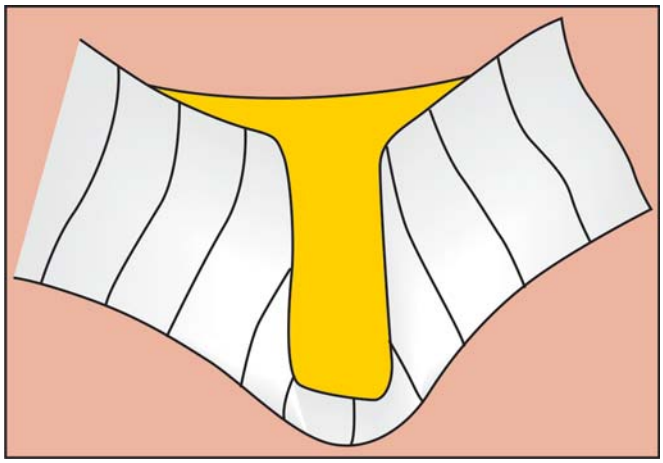


Fig. 17.10: Type 'A' Preventive resin restoration

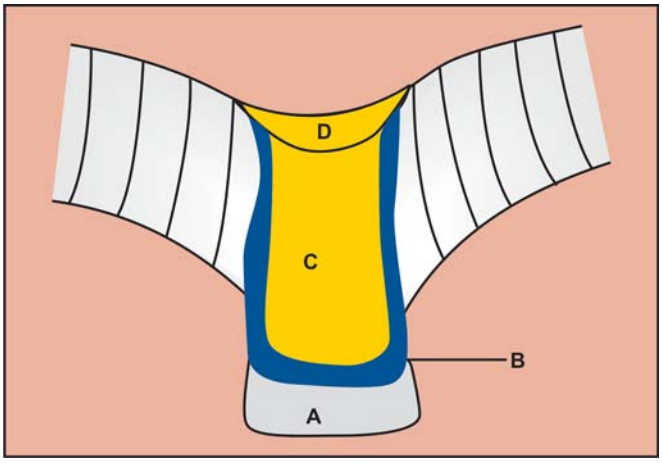


Fig. 17.11: Type 'B' Preventive resin restoration. A. Calcium hydroxide liner, B. Bonding agent, C. Filled composite resin, D. Sealant

Type C Restoration

1. Since this type of lesion is extensive in nature, it necessitates placement of cavity liner and then followed by steps similar to type B restoration.
2. Often it is done under local anesthesia to minimize patient discomfort and gain patient co-operation.

18

Fluorides in Pediatric Dentistry

INTRODUCTION

Fluoride plays a vital role in prevention of dental caries. It is considered to be the most effective and extensively used anticaries agent with increasing rate of success.

Chemistry

Fluorine (from Latin 'fluere', meaning to 'to flow'), is the chemical element in the periodic table that has the symbol 'F' and an atomic number 9. It is a highly reactive element of the halogen family, widely distributed in the earth's crust.

In nature, fluorine occurs abundantly in the form of Fluorite or Fluorspar (CaF_2); Fluorapatite [$Ca_{10}(PO_4)F_2$] and Cryolite (Na_3AlF_6).

Sources of Fluoride

- Water–Seawaters, springs, well water, lakes, etc.
- Foods–Fish, tea, rock salt, leafy plants, etc.

Metabolism of Fluorides

Absorption

Fluoride is absorbed into plasma from the gastrointestinal tract.

Blood plasma fluoride levels begin to rise in about 10 minutes after ingestion and reach maximum level within 1 hour.

Deposition

Absorbed fluoride gets deposited in the body in various tissues in different forms. It gets deposited in:

- Developing teeth
- Saliva
- Breast milk
- Fetal tissues
- Osseous tissues, etc.

Excretion

Fluoride passes through renal filtration and gets excreted in urine and to a lesser degree in sweat. The fluoride that remains unabsorbed is lost in feces.

FLUORIDES IN CARIES PREVENTION

Fluoride, an effective anticaries agent has various mechanisms of actions which occur simultaneously and has a cumulative effect.

A number of proposed mechanisms have been identified which are assumed to work simultaneously and are grouped as follows:

- Increase enamel resistance.
- Increased rate of posteruptive maturation.
- Remineralization of incipient lesions.
- Interference with plaque microorganisms.
- Modification in tooth morphology.

Increase Enamel Resistance

The enamel comprises of hydroxyapatite crystals that have certain voids in crystal structure rendering them highly reactive to chemical agents and thereby increasing its solubility.

Fluoride gets incorporated into these voids in exchange of hydroxyl ions to form fluorapatite crystals. Thereby rendering more compact and a stable structure and hence it increases enamel resistance to acid dissolution.

$$Ca_{10}(PO_4)_6\ (OH)_2 + 2F^- \rightarrow Ca_{10}(PO_4)_6\ F_2 + 2OH^-$$

Increased Rate of Posteruptive Maturation

Fluoride is said to hasten the rate of mineralization of hypomineralized areas. Since newly erupted teeth have more hypomineralized areas that are prone to dental caries, the effect of fluoride is optimum in mineralization of those caries prone surfaces.

It is accomplished by increasing the rate of mineralization and deposition of certain organic compounds that are more resistant to acid dissolution.

Remineralization of Incipient Lesion (Fig. 18.1)

Fluoride enhances the remineralization process by accelerating the growth of enamel crystals that have undergone demineralization.

Fluoride as an Inhibitor of Demineralization

Fluoride found in saliva adsorbs onto the surface of a tooth where demineralization has occurred. The presence

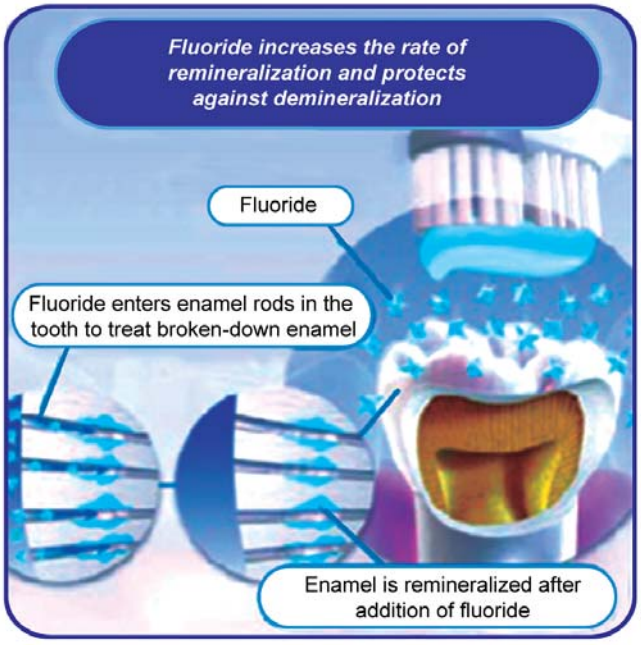


Fig. 18.1: Fluoride increases the rate of remineralization

of this fluoride in turn attracts minerals like calcium thus inhibiting demineralization and facilitating, remineralization of affected area.

Interference with Microorganisms

- Fluoride inhibits bacterial enzymatic processes involved in carbohydrate metabolism.
- In high concentration, it exhibits bactericidal action.
- In lower concentrations, it is bacteriostatic in nature.
- It destroys bacteria by cell wall destruction and also by depletion of carbohydrate source of bacteria by metabolizing the sugars in the plaque.

Modification in Tooth Morphology

There have been studies done to prove the effect of fluoride on morphology of teeth. It is assumed that fluoride present at stages of tooth development helps formation of a more causes resistant tooth slightly smaller with shallow fissures.

FLUORIDE DELIVERY METHODS

The above useful effects of fluoride in reduction of caries have led to its use in humans in various forms.

Fluoride has effects both topically and systematically when used. The concentration of fluoride varies according to the delivery method.

Systemic fluoride provides a low concentration of fluoride to the teeth over a long period. It circulates through the bloodstream and is incorporated into developing teeth. After teeth erupt fluoride contacts teeth directly through salivary secretions.

Topical fluorides are applied directly on tooth surfaces at a higher concentration but its effect ceases after a short period hence necessitating its reapplication within short span. Various systematic fluorides have a topical effect but their primary effect is systemic.

Need for Fluoride Supplements

It is a proved fact that maximum fluoride concentration is in first formed enamel near the incisal edge and gradually decreases towards the more recently formed cervical region. Thus in children enamel is much more susceptible to demineralization around cervical regions. This process can be inhibited by topical fluorides.

In older children this concentration is reversed. Hence the active role of fluoride in the caries process, thus recommends repeated application of fluoride at regular intervals.

METHODS OF FLUORIDE ADMINISTRATION

I. Systemic Fluoride

a. Dietary fluoride:
- Food stuffs
- Salt
- Sugar.

b. Fluoride supplements
- Fluoride drops
- Fluoride lozenges/tablets
- Sustained release fluorides
- Fluoride rinses.

c. Community approach
- Water fluoridation
- School water fluoridation
- Milk fluoridation.

II. Topical Fluorides

a. Professional application

- Fluoride solution
- Varnishes
- Foam
- Gels.

b. Self application

Water Fluoridation

Source of Fluoride

- Sodium fluoride
- Sodium silico fluoride
- Hydrofluorosilicic acids
- WHO recommends optimum level of fluoride in water as 0.7 to 1.2 ppm (1971)
- However, recent WHO recommendation states optimum fluoride level in drinking water as 0.5 to 1.0 ppm (1994).

Benefits

- Has both pre-and posteruptive cariostatic effects.
- Has both systemic and topical effect.
- Fluoride in saliva through this method remains elevated for extended period of time thereby protecting against demineralization and facilitating remineralization.
- Alters morphology of tooth by making pits and fissures shallow and self cleansable.
- Cost effective and targets large population easily.

School Water Fluoridation

- It is the adjustment of the fluoride concentration of a school's water supply for caries reduction and prevention.
- It has its own advantages and disadvantages.
- Less effective when compared to water fluoridation.

Dietary Fluoride Supplementation (Table 18.1)

Salt Fluoridation

- Incorporation of sodium/potassium fluorides on salt.

Advantages:

- Reduced risks of fluorosis.
- Safe
- Cost effective
- High acceptability and ease of availability and usage.

Disadvantage:

- No precise control over intake.

Table 18.1: Recommended dietary fluoride supplements

Age in years concentration of fluoride in drinking water in ppm			
	< 0.3 ppm	*0.3 to 0.6 ppm*	*> 0.6 ppm*
Birth to 6 months	None	None	None
6 months to 3 years	0.25 mg/day	None	None
3 to 6 years	0.50 mg/day	None	None
6 to 16 years	1.00 mg/day	None	None

Milk Fluoridation

- First advocated by Ziegler in 1956.
- An effective method targeted at children, infants and pregnant women as it is a staple food for them.

Disadvantages:

- Variation in intake and quantity of milk.
- Not cost effective.
- Absence of centralized milk supply universally.

Fluoride Supplements

- Fluoride supplements in the form of tablets, drops, Lozenges, etc. have a combined topical and systemic effects.
- However they need to be used with caution and used under professional advice.
- Its effect on both primary and permanent teeth has greatly reduced caries incidence.

Topical Fluorides

Professionally administered fluoride preparations (Fig. 18.2).

Fluoride varnishes: Two most commonly used varnishes are:

- Duraphat (NaF varnish)
- Fluorprotector (diflurosilane ethyl difluorohydroxy silane).

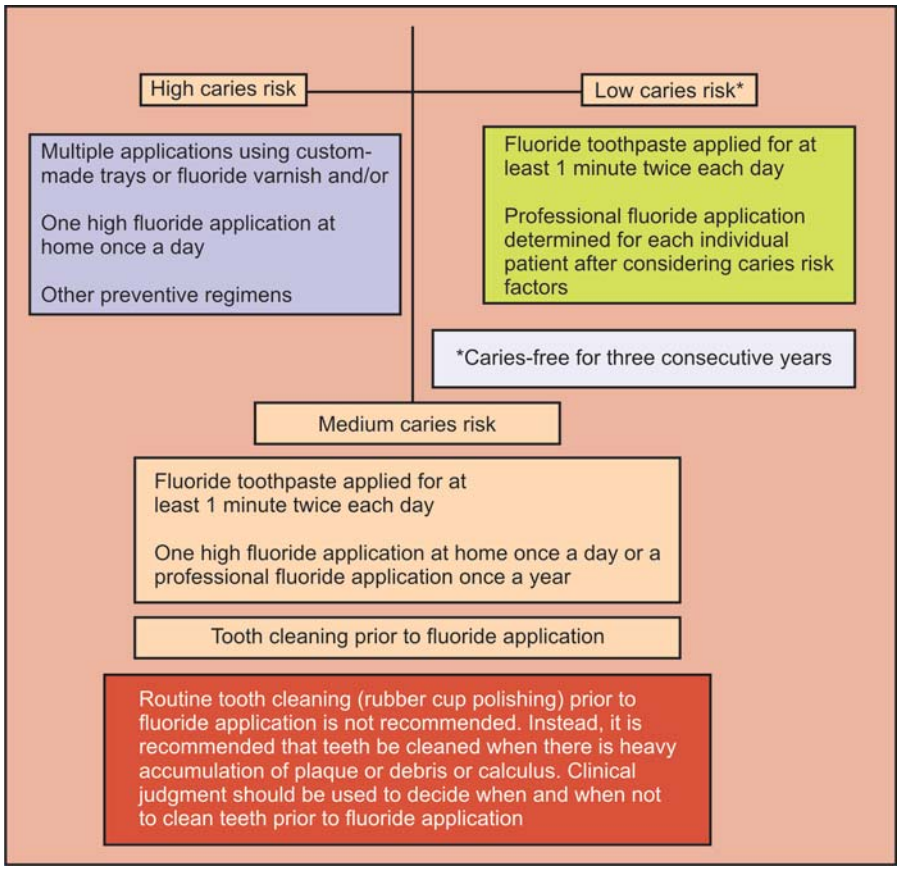


Fig. 18.2: Guidelines for fluoride applications

Mechanism of Action

On topical application of varnish, a reservoir of fluoride ions gets formed around enamel. There is a steady and slow release of fluoride reacting with hydroxyapatite crystals of enamel thereby steadily forming a layer of fluorapatite on enamel surface.

Method of Application (Fig. 18.3)

- Oral prophylaxis.
- Isolation (cotton rolls seldom used).
- Maintain a dry field.
- Application in lower arch first in order of lingual, occlusal and buccal surfaces using a single tufted small brush.
- Followed by varnish application in upper arch.
- After application; patient is advised to sit with mouth open for about 4-5 minutes and instructed not to rinse/drink/for one hour and not to consume solid food until next morning.

Fluoride Solution

Sodium Fluoride

Concentration: 2% NaF

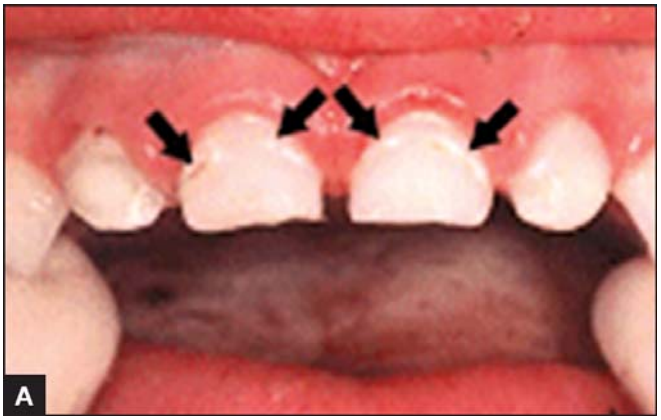
Method of preparation:

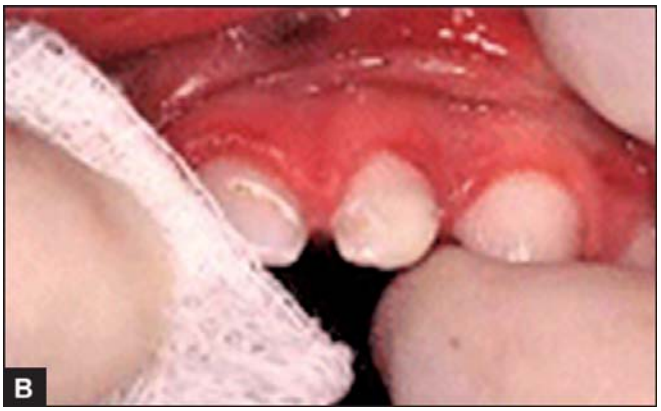
- 20 gm NaF dissolved in 1 litre distilled water in plastic bottle (if stored in glass bottle fluoride reacts with silica to form SiF_2).

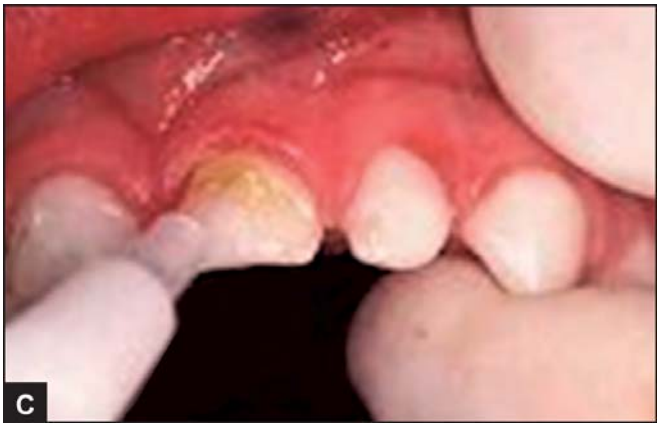
Application technique:

Knutson technique

- Oral prophylaxis followed by isolation with cotton rolls.
- Maintain a dry field.
- Two percent NaF is applied using cotton applicators on all exposed tooth surfaces in each quadrant.
- After completion patient is instructed to avoid eating, drinking or rinsing for 30 minutes.
- Second, third and fourth applications are done at weekly intervals.







Figs 18.3A to C: Application of fluoride varnish

- Full series of four treatments is recommended at 3, 7, 11 and 13 years of age.

Mechanism of action: Topically applied NaF reacts with hydroxyapatite to form CaF_2. Once a thick layer of CaF_2 forms, it interferes with further diffusion of fluoride to react with hydroxyapatite; in turn CaF_2 reacts with hydroxyapatite to form fluorapatite thereby making tooth structure more stable and less susceptible to acid dissolution.

Stannous Fluoride

Concentration-SnF_2 8%

Method of preparation:

- It has to be prepared freshly for every patient as it has no shelf life.
- Dissolve 0.8 gm of Stannous fluoride in 10 ml distilled water in a plastic container and immediately applied to the teeth.

Method of application:
Muhler's technique

- Oral prophylaxis followed by isolation.
- Maintain a dry field.
- Freshly prepared 8% SnF_2 solution is applied using cotton applicators.
- Teeth are kept moist with solution for 4 minutes with reapplication of solution once in every 15-30 seconds.
- Recommended once a year application.

Mechanism of action: Stannous fluoride when topically applied reacts with hydroxyapatite to form four different end products with unique functions of each.

i. Calcium fluoride reacts with hydroxyapatite to form fractions of fluorapatite.
ii. Tin hydroxyphosphate, another end product in responsible for metallic taste sensation after application.
iii. Calcium trifluorostannate is another end product.
iv. Tin trifluorophosphate is the main end product that enters the tooth structure more resistant to decay and more stable.

Acidulated Phosphate Fluoride

Concentration -> 1.23%

Method of Preparation:

"Brudevold's solution"

- Dissolve 20 gm of NaF in 1 liter of 0.1m phosphoric acid and 50% hydrofluoride acid is added to adjust pH at 3.0 and fluoride concentration at 1.23%.

Fig. 18.4: Gel foam trays

APF Gel

- To the above solution, methyl cellulose or hydroxyethyl cellulose (getting agent) is added and pH is adjusted 4-5.

Method of application:

- Oral prophylaxis and isolation using cotton rolls.
- Maintain dry field.
- Using cotton applicators APF solution is applied continuously and teeth are kept moist for four minutes.
- In case of APF gel, use foam U/L tray and fill them with APF gel (Fig. 18.4).
- Insert U/L trays simultaneously into patients mouth and have the patient bite down tightly for four minutes.
- Instruct the patient not to eat, drink or rinse for 30 minutes.
- Semiannual application of 1.23% APF gel/solution is recommended.

Mechanism of action: Initially when APF is applied on teeth there is dehydration and shrinkage in volume of hydroxyapatite crystals which further leads to form an intermediate product; Dicalcium phosphate dehydrate which is highly reactive with fluoride and thereby immediately starts forming fluorapatite crystals at a faster rate.

Other forms of topical fluoride applications include:

Iontophoresis

- A novel method that uses small electric currents to drive fluoride ions to react with hydroxyapatite crystals at a higher and faster rate.

Dental Materials Containing Fluoride

- With the advent of material science, various products have been developed with fluorides incorporated.
- These products are known to release low concentration of fluoride around the enamel when used and form of fluorapatite crystals.
- Fluoride containing dental materials are used as follows:
  - Fluoride containing varnishes and sealants.
  - Glass ionomer cements with fluorides are the recent innovations.

Self Applied Topical Fluorides

Fluoride Dentifrices

Dentifrices are generally a mixture of an abrasive/polishing agent, detergent, binders, flavoring agents and humidifiers. When Fluoride is included in the form of sodium fluoride or sodium monofluorophosphate between 1,000-1,500 ppm, it becomes a fluoridated dentifrices that holds a therapeutic or a preventive purpose.

There are various fluoridate dentifrices commercially available over the counter with ADA acceptance seal.

These dentifrices can be used regularly two or three times a day, as they provide a frequent source of low-concentration of fluoride that inhibits demineralization and promotes remineralization.

Precautions to be considered:

- Parental supervision is needed for preschool age children while brushing to avoid ingestion of excessive amount of paste.

- Only pea-size amount of dentifrice should be used by children less than or of 6 years age.

Recommendations for use of fluorides:

- Fluoride tooth paste is not recommended for children below 4 years.
- For children between 4 to 6 years, brushing once daily using fluoridated dentifrice is recommended.
- For 6-10 years children, use fluoridated dentifrice twice daily.
- For children above 10 years use fluoridated dentifrices upto three times a day.

Fluoride Mouth Rinses

It is not recommended for children above 6 years of age and those with orofacial musculature problems or other handicap.

Fluoride mouth rinses can be used daily or once in a week after brushing before bed depending upon fluoride concentration or as recommended by a dentist.

Sustained Release Fluorides

Recent advances in fluoride release/delivery system is the application of sustained release fluorides from either an intraoral device (impregnated into space maintainer, orthodontic appliances, crown, bridge, etc.) or into dental cements.

Fluoride diffuses out at a controlled steady rate and thereby maintains a steady low concentration of fluoride in oral environment leading to reduction of caries by remineralization of incipient carious lesions and also inhibiting demineralization.

Such products are of great use to patients who cannot properly maintain their oral health due to disabilities in physically or mentally.

FLUORIDE TOXICITY

Anything when used in excess will lead to toxicity, the same befits fluorides too. When there is overdosage or excessive administration acutely or chronically leads to a spectra of conditions/symptoms in the patient leading to what is termed as 'Fluoride toxicity'.

Fluoride toxicity may result either due to single ingestion of large amount of fluoride (acute) or long-term ingestion of a small quantity of fluoride that accumulates within tissues of the body (chronic toxicity).

Acute Fluoride Toxicity

The acute lethal dose for humans ranges from 2-10 gm with a mean of 5 gm of fluoride as a single ingestion.

Acute toxicity is a rate condition to occur, unless as in cases of suicidal attempts or accidental ingestion, etc.

Acute lethal dose for children is, however, very low as per their body weight. It is about 1-2 gm of fluoride.

Symptoms of Acute Fluoride Toxicity

- Vomiting, nausea, diarrhea.
- Pain in the abdomen and extremities.
- Excessive perspiration
- Thirst
- Muscle tremors
- Excessive salivation.
- Weak pulse
- Coma
- Cardiac arrhythmias
- Death.

Treatment

- Induce vomiting using either symptom of ipecac or mechanical stimulation tongue or throat.
- Administer liquids like warm water, milk, lime water, antacids, and aluminium hydroxide gels, egg orally which are potent fluoride binders and also protect upper gastrointestinal tract from fluoride burns.
- Seek immediate hospitalization and advocate gastric lavage.
- Also administer calcium gluconate with saline intravenously to prevent shock (only if muscle tremor occurs).

Chronic Fluoride Toxicity (Table 18.2)

Chronic fluoride toxicity occurs due to long-term ingestion of smaller amounts of fluorides which is usually a cumulative effect.

Table 18.2: Toxic effects of chronic excessive fluoride ingestion		
Effect	*Dosage*	*Duration*
Dental fluorosis	>2 times optimal	Until 5 years of age
Skeletal fluorosis	10-25 mg/day	10-20 years
Renal damage* (*in animals)	5-10 mg/kg	6-12 months

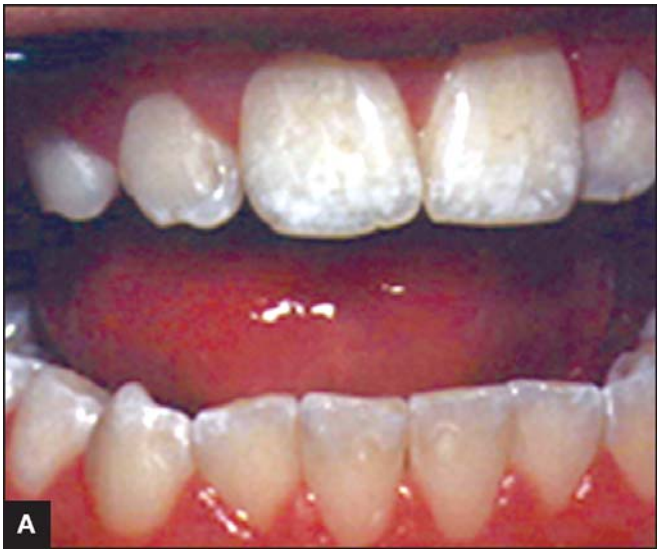
Effects are seen in hard tissues and kidneys. Common features of chronic fluoride toxicity:

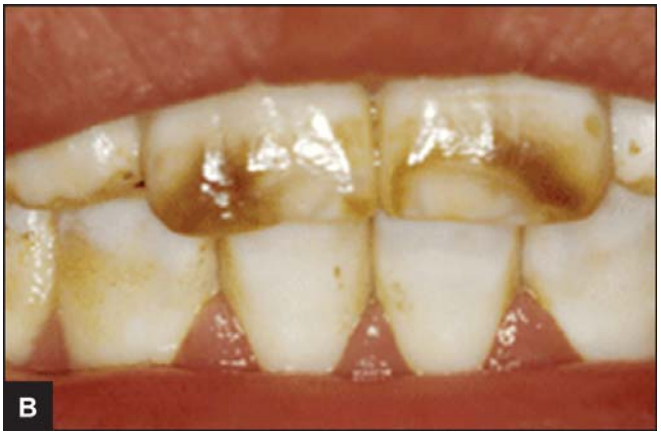
On Enamel

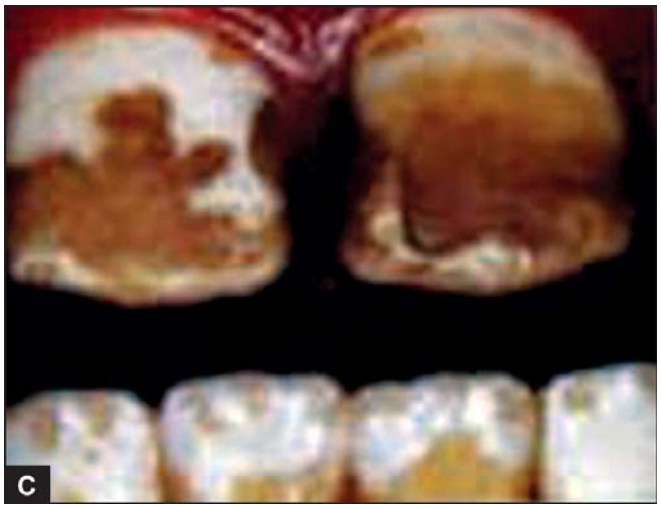
- If it occurs during tooth development, it leads to a condition called 'Fluorosis' (Table 18.3).

Table 18.3: Dean's fluorosis index and TF index

Criteria for Dean's Fluorosis Index	
Score	**Criteria**
Normal	The enamel represents the usual translucent semivitriform type of structure. The surface is smooth, glossy, and usually of a pale creamy white color.
Questionable	The enamel discloses slight aberrations from the translucency of normal enamel, ranging from a few white flecks to occasional white spots. This classification is utilized in those instances where a definite diagnosis of the mildest form of fluorosis is not warranted and a classification of "normal" is not justified.
Very mild	Small opaque, paper white areas scattered irregularly over the tooth but not involving as much as 25% of the tooth surface. Frequently included in this classification are teeth showing no more than about 1-2 mm of white opacity at the tip of the summit of the cusps of the bicuspids or second molars.
Mild	The white opaque areas in the enamel of the teeth are more extensive but do not involve as much as 50% of the tooth.
Moderate	All enamel surfaces of the teeth are affected, and the surfaces subject to attrition show wear. Brown stain is frequently a disfiguring feature.
Severe	Includes teeth formerly classified as "moderately severe and severe." All enamel surfaces are affected and hypoplasia is so marked that the general form of the tooth may be affected. The major diagnostic sign of this classification is discrete or confluent pitting. Brown stains are widespread and teeth often present a corroded-like appearance.
Clinical Criteria and Scoring for the TF (Thylstrup-Fejerskov) Index	
Score	**Criteria**
0	Normal translucency of enamel remains after prolonged air-drying.
1	Narrow white lines corresponding to the perikymata.
2	*Smooth surfaces:* More pronounced lines of opacity that follow the perikymata. Occasionally confluence of adjacent lines. *Occlusal surfaces:* Scattered areas of opacity <2 mm in diameter and pronounced opacity of cuspal ridges.
3	*Smooth surfaces:* Merging and irregular cloudy areas of opacity. Accentuated drawing of perikymata often visible between opacities. *Occlusal surfaces:* Confluent areas of marked opacity. Worn areas appear almost normal but usually circumscribed by a rim of opaque enamel.
4	*Smooth surfaces:* The entire surface exhibits marked opacity or appears chalky white. Parts of surface exposed to attrition appear less affected. *Occlusal surfaces:* Entire surface exhibits marked opacity. Attrition is often pronounced shortly after eruption.
5	*Smooth surfaces and occlusal surfaces:* Entire surface displays marked opacity with focal loss of outermost enamel (pits) <2 mm in diameter.
6	*Smooth surfaces:* Pits are regularly arranged in horizontal bands <2 mm in vertical extension. *Occlusal surfaces:* Confluent areas <3 mm in diameter exhibit loss of enamel. Marked attrition.
7	*Smooth surfaces:* Loss of outermost enamel in irregular areas involving <1/2 of entire surface. *Occlusal surfaces:* Changes in the morphology caused by merging pits and marked attrition.
8	*Smooth and occlusal surfaces:* Loss of outermost enamel involving >1/2 of surface.
9	*Smooth and occlusal surfaces:* Loss of main part of enamel with change in anatomic appearance of surface. Cervical rim of almost unaffected enamel is often noted.







Figs 18.5A to C: Clinical picture of dental fluorosis. A. Mild, B. Moderate, C. Severe

- Dental fluorosis is defined as a permanent hypomineralization of enamel, characterized by greater surface and sub surface porosity than in normal enamel that results from excess fluoride reaching the developing tooth during developmental stages. Excess fluoride available to the enamel during maturation disrupts mineralization and results in excessive retention of amelogenesis and other enamel proteins.
- Dental fluorosis is characterized by (Figs 18.5A to C)
- Increased porosity of subsurface enamel appearing as white opaque flecks irregularly.
- As the severity of fluorosis increases enamel becomes porous, pitted and discolored and more prove to fracture and wear.

Skeletal Effects

- Pains involving joints to hands, feet, knee and spine.

Knee and Spine

- Stiffness of spine.
- Rigidity of thoracic cage.
- Fragility of bones.

Renal Effects

- Affects GFR.
- Induces chronic renal failure, etc.

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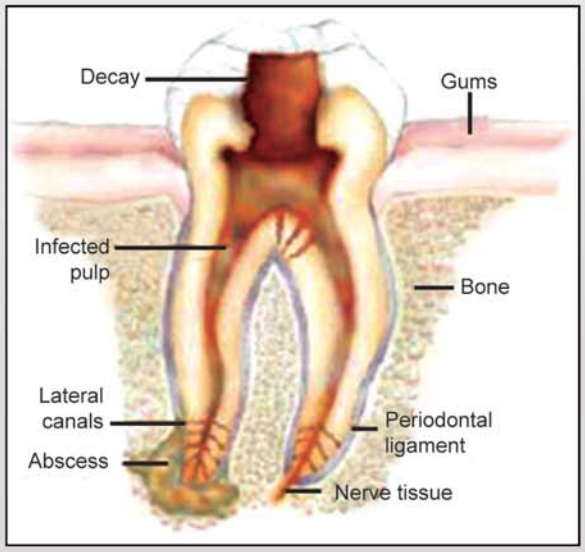


Pediatric Endodontics

INTRODUCTION

Despite advent of various preventive strategies like fluoridation, sealants and emphasis on prevention of caries, premature loss of primary and young permanent teeth continues to be common. The total prevention of caries is the ultimate goal of dentistry, but meanwhile procedures must be utilized to preserve the primary and young permanent teeth ravaged by caries.

OBJECTIVES OF PULPAL THERAPY

- Conservation of the tooth in a health state functioning as an integral component of dentition.
- Preservation of dental arch space and prevent consequent malocclusion due to premature tooth loss.
- Maintain esthetics, mastication.
- Prevent aberrant tongue habits
- Aid in speech.
- Prevent psychological effects associated with tooth loss.

However, there are certain restraints where the tooth cannot be saved by pulpal therapy such as:

- Medically compromised
- Non-restorability of teeth
- Rate of root resorption
- Life expectancy of a tooth.

Success of pulpal therapy lies in a systematic diagnosis and evaluation of the clinical situation and thereby providing a befitting treatment. The following outline provides a quick review of steps taken in endodontic diagnosis of involved primary and young permanent teeth.

1. Chief complaint
2. Health history
   Medical history
   Dental history
      History of trauma
      Past and present dental treatment
      Present signs and symptoms.
3. Diagnostic evaluations
4. Analysis of data obtained
5. Formulation of an appropriate diagnosis and treatment plans

DIAGNOSTIC EVALUATION

Subjective Examination

Obtain information by question and answer regarding history of presenting illness and symptoms.

- Location
- Intensity
- Duration
- Stimulus
- Relief
- Spontaneity

Tenative Diagnosis

After taking histories and identifying signs and symptoms, the practitioners may reach a tentative diagnosis. The objective examination will gather information necessary to confirm the diagnosis.

Objective Examination

Extraoral Examination

Check general appearance, skin tone, and facial asymmetry.

Note any swelling, redness, sinus tracts, tender or enlarged lymph nodes, or tenderness or discomfort upon palpation of TMJ.

Soft Tissue

Examine mucosa, gingival visually and digitally for discoloration, inflammation, ulceration, swelling and sinus tract formation.

Dentition

Examine teeth for discoloration, fracture, caries, large restorations or other abnormalities.

Clinical Tests

Most tests have inherent limitations. They require care on application and interpretation.

The objective is to discover which tooth is different from the patient's other teeth. Always best healthy control teeth first.

Clinical testing methods, at times can be misleading in children due to lack of emotional maturity; fear and lack of distinguishing pain from other symptoms, etc.

Periradicular Tests

- Percussion
- Palpation

Pulp Vitality Tests

- Cold test
- Heat test
- Electric pulp testing
- Test cavity

Radiographic Examination

Current radiographs are essential to examining for caries and periapical changes. Interpretation of radiographs is complicated in children.

Limitations

- Pathologic vital pulps are not visible.
- Necrotic pulps may not produce radiographic changes early.
- To be visible inflammation should spread to cortical bone.

The radiograph does not always give evidence of periapical pathosis when present, nor can the proximity of caries to the pulp always be accurately determined.

Radiographs provides details about

- Root resorption, if any
- Periapical pathosis
- Bone loss
- Relation to underlying permanent tooth bud outline of pulp chamber, etc.

Special Tests

Special investigations include:

- Laser Doppler flowmetry
- Pulse oximetry
- Dual wavelength spectrometry
- Transillumination
- Selective anesthesia
- Caries removal

TREATMENT OPTIONS

The treatment of pulpally involved teeth in the primary and young permanent teeth presents unique challenges. However various pulp therapies available are grouped as.

Primary Teeth

Vital Pulp Therapy for Primary Teeth Diagnosed with a Normal Pulp

a. Protective base
b. Indirect pulp therapy
c. Direct pulp capping
d. Pulpotomy

Nonvital Pulp Treatment for Primary Teeth Diagnosed with Necrotic Pulp

a. Pulpectomy
b. Mortal Pulpotomy

Young Permanent Teeth

Vital Pulp Therapy for Teeth Diagnosed with a Normal Pulp or Reversible Pulpitis

a. Protective base
b. Indirect pulp therapy
c. Direct pulp capping
d. Partial pulpotomy for carious exposures
e. Partial pulpotomy for traumatic exposures (Cvek's pulpotomy)
f. Apexogenesis.

Nonvital Pulp Treatment

a. Pulpectomy (Conventional root canal treatment)
b. Apexification (Franks' root end closure).

PULPAL MEDICAMENTS

Over decades of clinical research various pulpal medicaments have been found and clinically used with proved success rates with their own merits and demerits. Some of the pulpal medicaments commonly used are as follows:

Fig. 19.1A: Calcium hydroxide

Fig. 19.1B: Formocresol

Pulp Capping Agents

Calcium Hydroxide (Figs 19.1 A and B)

It was the first pulp capping agent used; introduced by Hermann in 1930.

It appears to serve as a protective barrier for pulp tissues, blocking patient dentine tubules and neutralizing the attack of inorganic acids and their leached products from certain cements and restorative fillings.

It is known to stimulate the formation of reparative dentine bridges at the junction of necrotic tissue and vital inflamed tissue.

It is initially bactericidal and later becomes bacteriostic in nature.

Three main forms of calcium hydroxide products available are:

Pulpdent:
52.5% calcium hydroxide suspended in aqueous solution of methyl cellulose.

Dycal:
Two paste form
Base: Titanium dioxide in glycol salicylate with pigments.
Catalyst: Calcium hydroxide and Zinc oxide in ethyl touledene sulfonamide.

Hydrex:
Two paste form, non-essential oil, hard setting form that contains calcium hydroxide, Barium sulfate, Tin oxide and selected resins.

Zinc Oxide Eugenol

Powder: Uncalcined Zinc Oxide.

Small amounts of zinc stearate, zinc acetate, rosin.

Liquid: Purified Eugenol.

Reinforced Zinc Oxide Eugenol

Powder: In addition to normal components 10-40% of finely divided natural/synthetic resin is added.

EBA Category:

Powder: In addition to zinc powder; Quartz alumina or rosin is added.

Liquid: Orthoethoxybenzoic acid is added along with eugenol.

Cavit:
Powder: Zinc oxide, Calcium sulfate, Zinc sulfate, glycol acetate, polyvinyl.

Acetate, polyvinyl chloride acetate, triethanolamine and red pigment.
Liquid: No eugenol only water.

Pulpotomy Medicaments

Formocresol

Formalin: 37% Formaldehyde solution.
Cresol: Cresylol, Tricresol.
It has been the medicament of choice since its innovation.

It is composed of 35% Tricresol and 19% Formaldehyde in a 15% water and glycerin solution.

This mixture is termed as "Buckley's formocresol"

Diluted form cresol is usually used in pulpotomies since it is equally effective as and less damaging than the traditional preparation.

Recommended way of diluting formocresol using Buckley's formocresol to make a 20% 1:5 concentrations is as follows:

Dilute 3 parts of glycerin with 1 part distilled sterile water; mix well.

Add 1 part of formocresol to 4 parts of diluents

Add 30 ml formocresol to 120 ml diluent to obtain 150 ml of dilute formocresol (20% strength 1:5 concentration).

Mechanism of action:
Formocresol when applied over infected pulp tissue it causes either partial or complete denaturation of the pulp tissue and fixes it.

It induces chronic inflammation in the pulp tissue.

Histologic features of Formocresol exposed pulp tissue reveals three distinct zones in 7-14 days:

- A broad acidophilic zone of fixation.
- A broad pale-staining zone with diminished cellular and fiber definition (atrophy) and
- A broad zone of inflammatory cells concentrated at pale-staining zone junction and diffusing apically into normal pulp tissue (Fig. 19.2).

Demerits:

- It has systemic distribution.
- It has mutagenic or carcinogenic potential.
- Increase pulpal irritation.
- Greater depth of penetration even into periapical area, etc.
- Rate of root resorption is greater than in normal.
- Causes dystrophic calcification in pulp canals.

Isobutyl Cyanoacrylates

- Used as an excellent capping agent
- Effective hemostatic agent
- Helps reparative dentine bridge formation
- Minimal inflammatory response.

Mineral Trioxide Aggregate (MTA) (Fig. 19.3)

When compared with calcium hydroxide, MTA produces significantly more dentinal bridging in shorter period of time with significantly less inflammation.

The procedure involves placing MTA over the exposure site and temporarily sealing to allow it to harden. The tooth is later re-entered and permanently

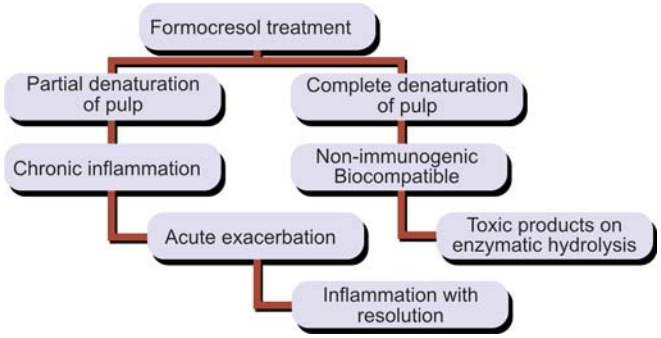


Fig. 19.2: Possible results of Formocresol treatment

Fig. 19.3: Mineral trioxide aggregate (MTA)

sealed over the set MTA with an etched, dentine bonding agent and composite resin.

The disadvantages are:

- Prolonged or late setting time (3-4 hrs)
- Second appointment needed.

Denatured Albumin

Newer materials include use of Collagen, Chondroitin sulfate, Sodium hyaluronate.

LASER

CO_2 laser can be used.

Glutaraldehyde

Concentration-2% aqueous Glutaraldehyde.

Properties:

- Rapid surface fixation by cross linkage.
- Limited depth of penetration.
- Excellent bactericidal agent.
- Causes less necrosis of pulp tissue.
- Causes less dystrophic calcification of pulp canals.
- Does not stimulate a significant immune response.
- Binding with protein tissue is irreversible.

N_2

Liquid

92% Eugenol
8% Rose oil.

Powder

72% Zinc oxide
12% Barium sulfate
6.3% Titanium oxide
4.7% Para formaldehyde
0.94% calcium hydroxide
0.16% Phenyl mercuric borate

Cresatin (Metacresyl acetate, etc.)

Ferric Sulfate (Fig. 19.4)

- It has been suggested as a substitute for formocresol.
- Has 92.7% success rate in primary teeth when compared with formocresol.

Properties:

- Astringent
- Forms Ferric ion complex that mechanically occlude capillaries
- Less inflammatory than formocresol.

Fig. 19.4: Ferric sulfate

Agents used for Devitalization Pulpotomy

Gysi priopaste:

- Tricresol 10 ml
- Cresol 20 ml
- Glycerin 4 ml
- Paraformaldehyde 20 ml
- Zinc oxide 60 gm

Easlick's paraformaldehyde paste:

- Paraformaldehyde 1 gm
- Procaine base 0.03 gm
- Powdered asbestos 0.05 gm
- Petroleum jelly 125 gm
- Carmine

Paraform devitalizing paste:

- Paraformaldehyde 1 gm
- Lignocaine 0.06 gm
- Propylene glycol 0.50 ml
- Carbowax 1500 1.30 gm
- Carmine

Root Canal Materials for Primary Teeth (Fig. 19.5)

Walkhoff paste:

- Parachlorophenol
- Camphor
- Menthol

KRI paste:

- Iodoform 80.8%
- Camphor 4.86%
- Parachlorophenol 2.025%
- Menthol

Maisto paste:

- Zinc oxide 14 gm
- Iodoform 42 gm
- Thymol 2 gm
- Chlorophenol camphor 3 cc
- Lanolin .50 gm

Vitapex:

- Calcium hydroxide
- Iodoform
- Oil additives.

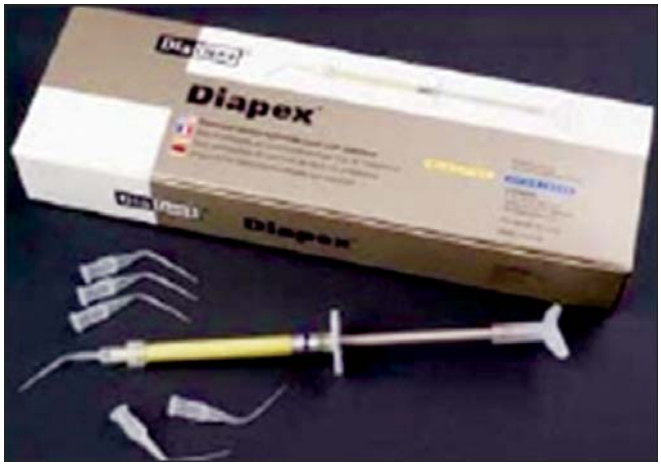


Fig. 19.5: Iodoform paste

PULPAL THERAPY IN PRIMARY TEETH

Vital Pulp Therapy for Primary Teeth Diagnosed with a Normal Pulp or Reversible Pulpitis

Protective Base

A protective base is a material placed on the pulpal surface of a cavity preparation, covering exposed dentin tubules, to act as a protective barrier between the restorative material or cement and the pulp.

Placement of a protective base such as calcium hydroxide or glass ionomer cement that possesses suitable physical properties and biocompatibility is at the dentist's discretion.

Indications:

In a tooth with normal pulp when dentin is exposed and all caries is removed during preparation for a restoration, a protective radio-opaque base may be placed between the permanent restoration and the dentin to minimize injury to the pulp and promote pulp tissue healing or minimize postoperative sensitivity.

Indirect Pulp Therapy (Fig. 19.6)

Indirect pulp therapy is a procedure performed in a tooth with a deep carious lesion adjacent to the pulp. The caries near the pulp is left in place to avoid pulp tissue exposure and is covered with a biocompatible material.

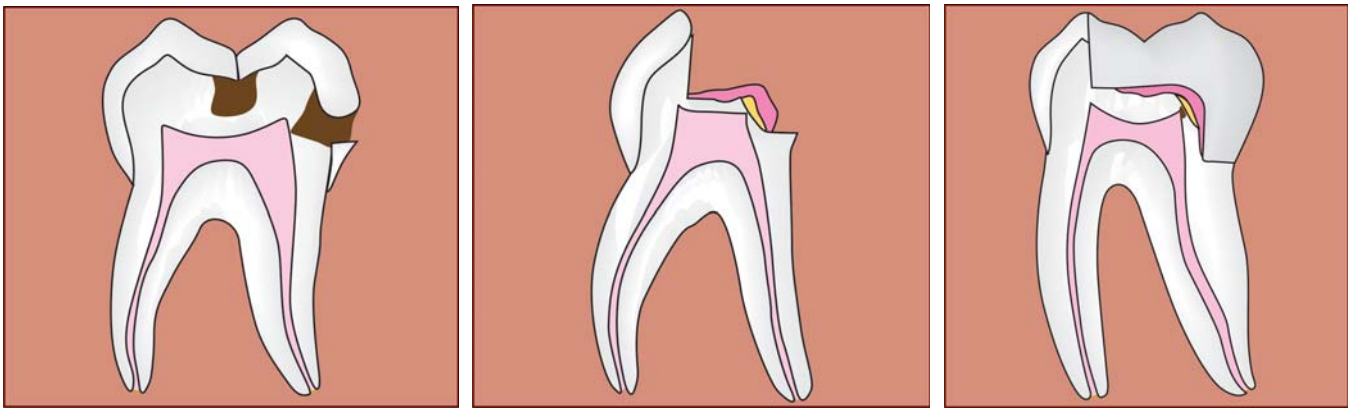


Fig. 19.6: Indirect pulp therapy

A radio-opaque base such as calcium hydroxide, zinc oxide eugenol or glass ionomer cement is placed over the remaining affected dentin to stimulate healing and repair. The tooth then is restored with a material that seals the tooth from microleakage.

Objectives of indirect pulp therapy:

- Reduction of hyperemia in pulp.
- Remineralization of carious or precarious dentine.
- Reduction of anaerobic bacteria.
- Formation of reparative dentine.
- Maintenance of pulpal vitality.
- Continued normal root closure.

Indications:

- Teeth with deep caries that are free of any symptoms of painful Pulpitis
- No history of spontaneous tooth pain, pain or percussion abnormal mobility, radiographic evidence of radicular disease, internal/external root resorption.

Technique:

- Administer L.A.
- Isolate with rubber dam.
- Use an F.G No.330 bur to open carious area, remove all unsupported enamel. The preparation is extended as dictated by carious process.
- Use no.4.6 or 8 carbide bur at low speed to remove carious dentin to within 1 mm of pulp chamber.
- Apply a protective base over the area of near exposure. There are three techniques of base placement follows:
  1. Technique I: A thin layer of calcium hydroxide is placed over area of near exposure. A thicker layer of zinc oxide eugenol is then applied.
  2. Technique II: A thin layer of zinc oxide eugenol paste is placed over the area of near exposure. A thicker layer of zinc oxide eugenol is then applied.
  3. Technique III: A dressing of calcium hydroxide paste with or without zinc oxide eugenol is placed over residual caries.
- Remove excessive temporary filling from the cavity margin and restore with silver amalgam or reinforced zinc oxide eugenol completely.
- Establish a recall time of about 6-9 weeks and re-entry is decided and base and calcium hydroxide is removed along with if at all any caries is left.
- Replace liner with dycal and restore the tooth with either silver amalgam or stainless steel crown.
- Indirect pulp therapy is otherwise termed as "Rest Treatment".

Direct Pulp Capping (Fig. 19.7)

Indications:

Small mechanical or traumatic exposure less than 1 mm which is surrounded by sound dentine.

In case of light red bleeding form site of pulp exposure that can be controlled by application of pressure using cotton pellets.

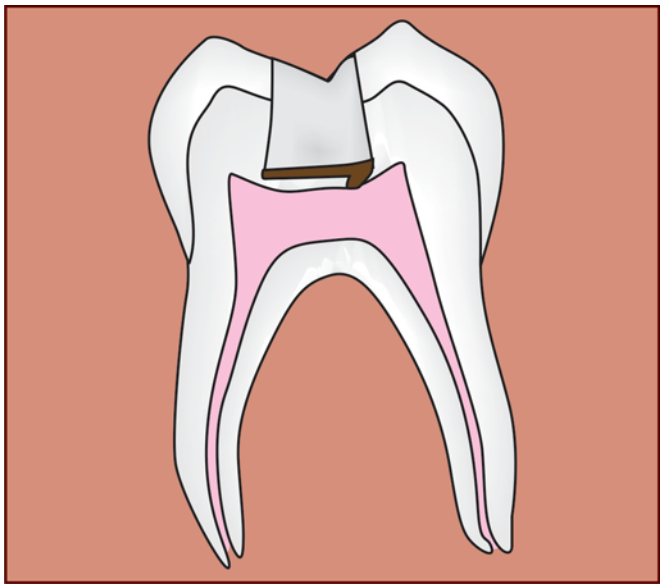


Fig. 19.7: Direct pulp capping

Contraindications:

- Carious pulp exposures.
- Any signs of pain; mobility.
- Radiographic evidence of thickened periodontal ligament/intraradicular radiolucency.
- Excess bleeding at exposure site.
- Presence of purulent exudates/sinus tract formation.

Objectives:

- Maintain tooth's vitality.
- No post treatment signs or symptoms of sensitivity, pain or swelling should be evident.
- No radiographic signs of pathologic external or internal root resorption or furcation/apical radiolucency
- No harm to succedaneous tooth.

Technique:

- Administer LA
- Isolate with rubber dam.
- Carefully remove the deep carious dentin over the exposure site to keep to a minimum the pushing of dentinal debris into pulp chamber.
- Flush out dentinal debris and control bleeding at exposure site.
- The pulp capping agent is applied over the exposure site.
- The rest of the cavity is restored with silver amalgam or composite resin or stainless steel crown that seals the tooth from microleakage.

Pulpotomy

Finn (1995) defined pulpotomy as the complete removal of the coronal portion of dental pulp, followed by placement of a suitable dressing or medicament that will promote healing and preserve the vitality of the tooth.

Refer Table 19.1 for classification of pulpotomy.

Table 19.1: Classification of pulpotomy

I. Vital Pulpotomy technique		
1. Devitalization Pulpotomy		
Single sitting :	1.	Formocresol
	2.	Electro surgery
	3.	Laser
Two sitting:	1.	Gysi trio paste
	2.	Easlick's formaldehyde
	3.	Paraform devitalizing paste
2. Preservation		
1. Glutaraldehyde		
2. Ferric sulfate		
3. Regeneration		
1. Bone morphogenetic protein		
II. Nonvital Pulpotomy Technique (Mortal Pulpotomy)		
1. Beechwood cresol		
2. Formocresol		

Indications

- Carious pulp exposures of teeth when their retention is more advantageous than extraction.
- When inflammation is confined to coronal poster of pulp.
- Vital tooth with health periodontal support.
- Restorable teeth.
- Tooth that possess atleast 2/3rd of its root length.
- Hemorrhage from the amputation site is pale red and easy to control.

Contraindications

- Evidence of internal/external resorption.
- Evidence of interradicular bone loss.
- Presence of abscess, fistula in relation to teeth.
- Radiographic evidence of pulpal calcification.
- Life expectancy of tooth is very short
- Non-restorable teeth.
- Marked tenderness to percussion.
- Any sign of spontaneous pain especially at nights.

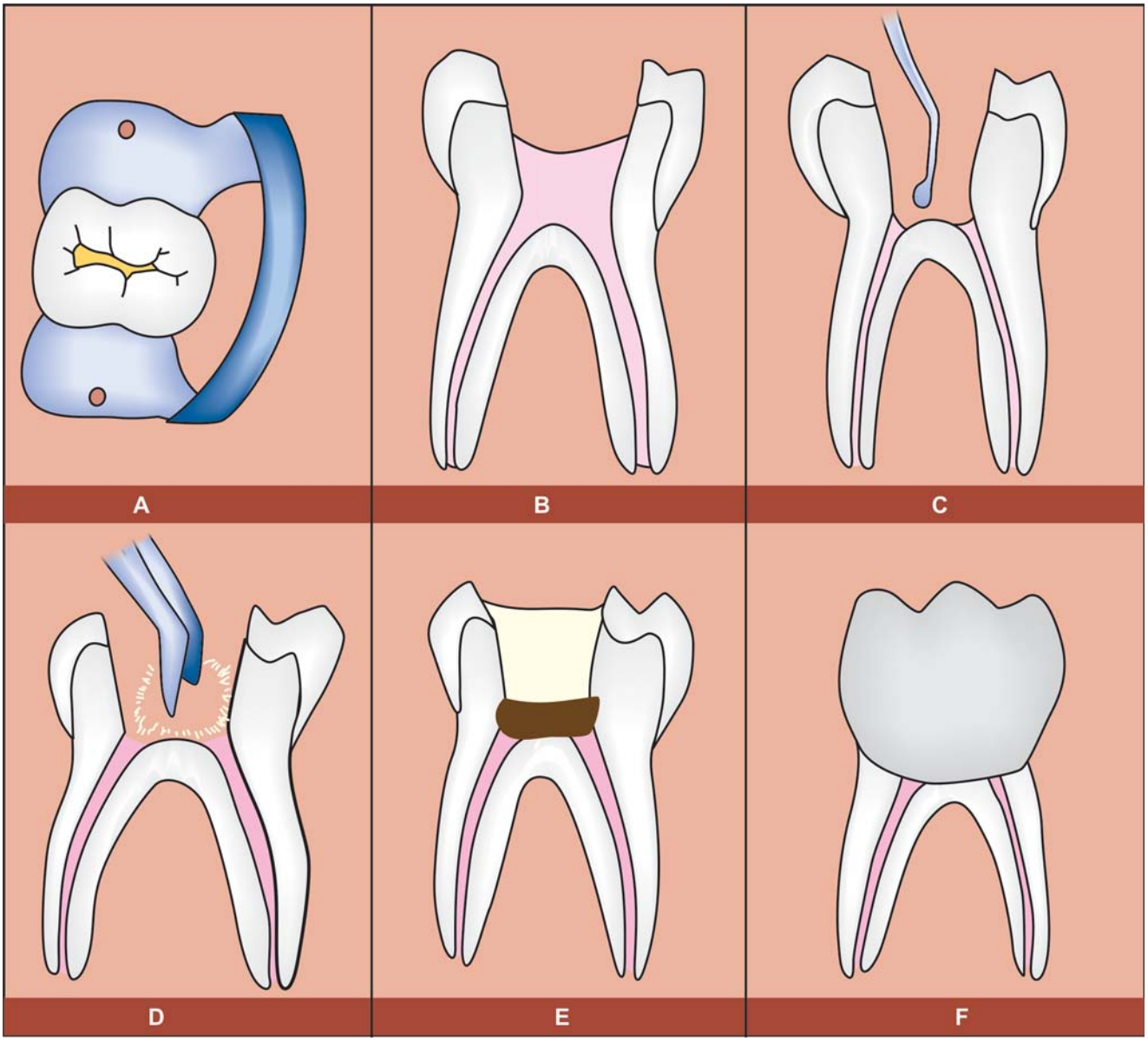
Objectives

- Radicular pulp should remain healthy without adverse clinical signs or symptoms such as sensitivity, pain or swelling.
- There should be no postoperative radiographic evidence of pathologic external/internal root resorption.
- There should be no harm to the succedaneous tooth.

Devitalization

Formocresol Pulpotomy—Single Sitting (Fig. 19.8)

- Obtain profound anesthesia.
- Selection of appropriate rubber dam clamp application.
- Access opening with high speed handpiece with appropriate fissure bur.



Figs 19.8 A to F: Formocresol pulpotomy

- Prior to entering pulp chamber remove all carious lesion with slow speed bur.
- Deroof the chamber with a fissure bur in a high speed handpiece.
- Amputate the coronal pulp using sterile round bur or sterile sharp spoon excavator.
- Obtain hemostasis by exerting pressure with sterile cotton pellets.
- Treat radicular pulp stumps by applying a barely moistened cotton pellet of formocresol for 5 minutes.
- Place a dressing of zinc oxide eugenol with or without a drop of formocresol over radicular stumps.
- The tooth is then restored with stainless steel crown.

Formocresol Pulpotomy—Two Stage/Sitting

First visit:

- Preparation of cavity until excavation of deep carious dentine under LA and isolation.
- Pulp exposure site enlarged with sterile round bur.
- Incorporate Paraformaldehyde paste into cotton pellet and place over exposure site and seal the tooth with IRM for 1-2 weeks.

Second visit:

- Re-enter the tooth and amputate coronal pulp tissue with sterile spoon excavator/round bur.
- No LA is needed.
- Tooth is then restored with an antiseptic dressing and stainless steel crown.

Preservation Pulpotomy

Similar to single stage formocresol pulpotomy only difference is instead of formocresol either glutaraldehyde or ferric sulfate is used and restored with stainless steel crown.

Nonvital pulp treatment for primary teeth. Diagnosed with irreversible pulpitis or necrotic pulp.

Mortal Pulpotomy

It is a two stage/visit procedure that uses beechwood cresol and formocresol.

First visit:

- Removal of necrotic coronal pulp.
- Irrigation with saline and dry with sterile cotton pellets
- Infected radicular pulp is treated with beechwood cresol moistened sterile cotton pellet and temporarily sealing the cavity for 1-2 weeks.

Second visit:

- Remove temporary dressing
- Pulp chamber is filled with antiseptic paste and restored with stainless steel crown.

Pulpectomy (Figs 19.9 and 19.10)

Indications

Primary teeth with irreversible pulpitis or necrosis.

Tooth treatment planned for pulpotomy is which the radicular pulp exhibits signs of pulpal necrosis.

No evidence of pathologic conditions with root resorption not more than 2/3rd of roots length.

Contraindications

- Radiographic evidence of severe bone loss intra-radicular/periapical radiolucency, pathologic internal/external root resorption.
- Sinus tract formation
- Life expectancy of primary tooth is short.

When a medical condition of the patient outweighs extraction rather than attempts to save the tooth.

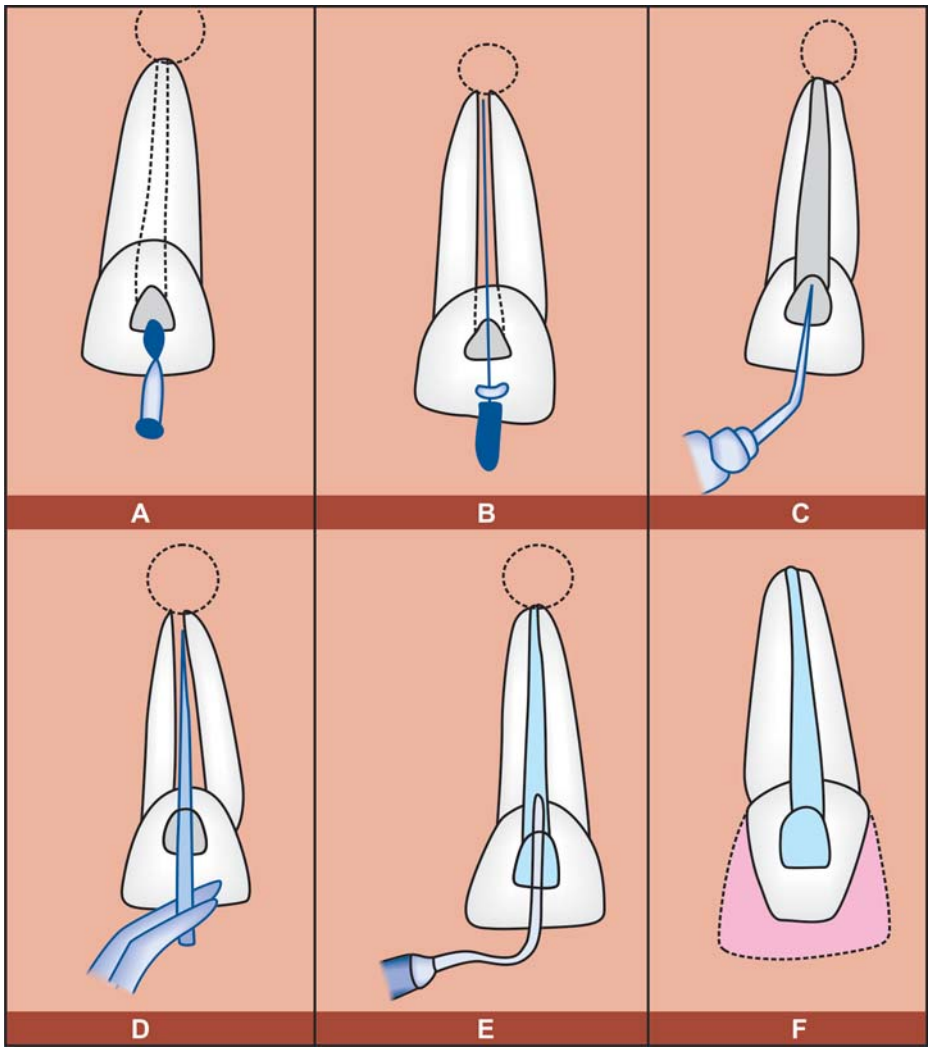
Objectives

Following treatment, the radiographic infections process should resolve in 6 months as evidenced by bone deposition in the pretreatments radiolucent areas, and pretreatment clinical signs and symptoms should resolve within 2 weeks.

There should be radiographic evidence of successful felling without gross overextension or underfilling.

The treatment should permit resorption of primary tooth root structures and filling materials at the appropriate time to permit normal eruption of the succedaneous tooth.

There should be no pathologic root resorption or furcal/apical radioluscency.



Figs 19.9 A to F: Pulpectomy in primary anteriors

Procedure

- Administer LA and isolate tooth with rubber dam.
- Prepare cavity as dictated by caries.
- Check that all coronal pulp is removed.
- After opening the pulp chamber, evaluate the hemorrhage and control it.
- With an endodontic file, remove diseased pulp tissue from all canals.
- Irrigate the canals and dry the canal and working length is determined radiographically.
- Obturate the canals with zinc oxide eugenol cement on Iodoform paste, using an amalgam plugger applying constant pressure.
- Obtain a periapical radiograph to be certain that canals are filled with zinc oxide eugenol.
- Restore the tooth with a stainless steel crown.
- Recall the patient for periodic visits to evaluate the success of the procedure.

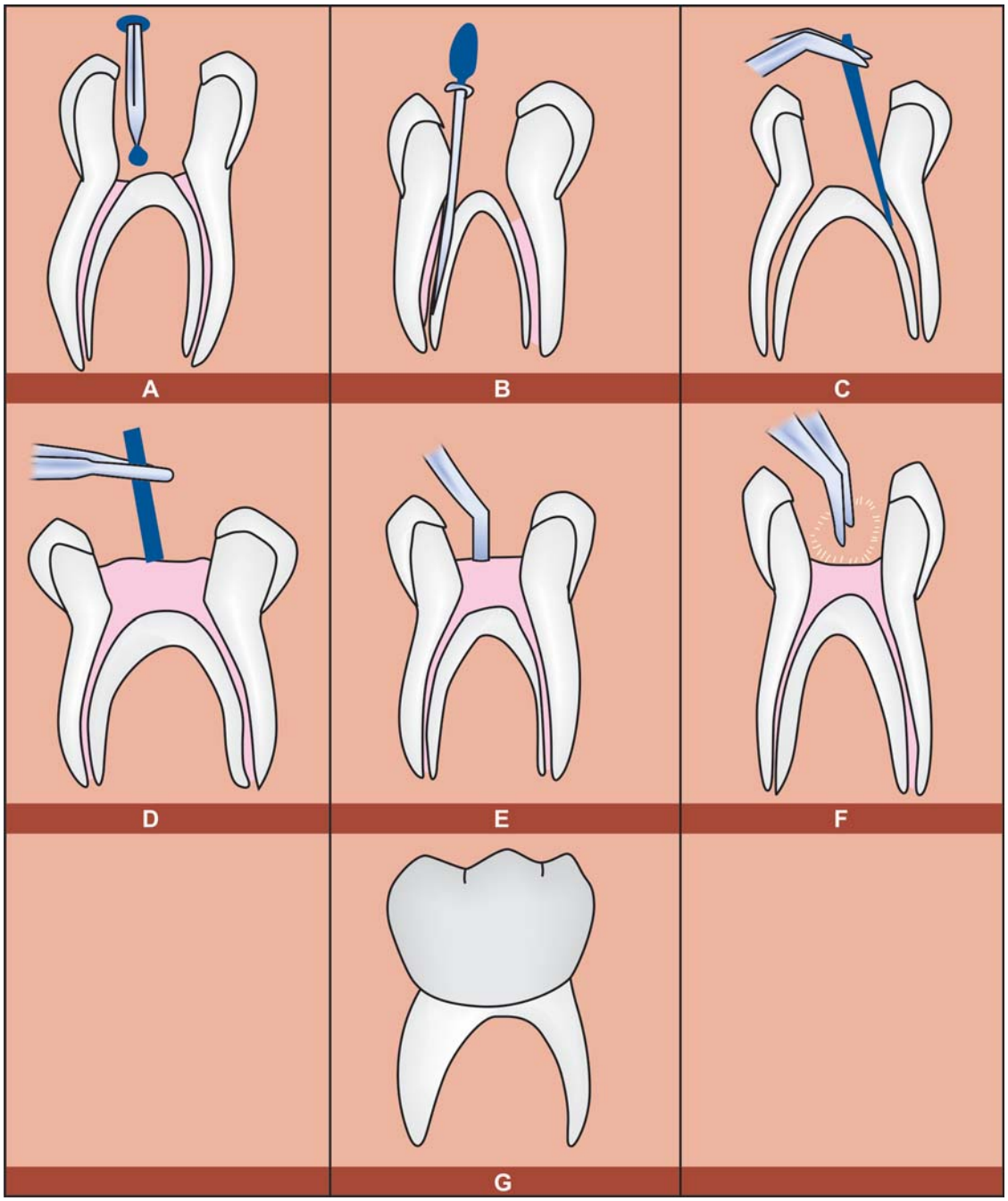


Fig. 19.10: Pulpectomy in primary molars

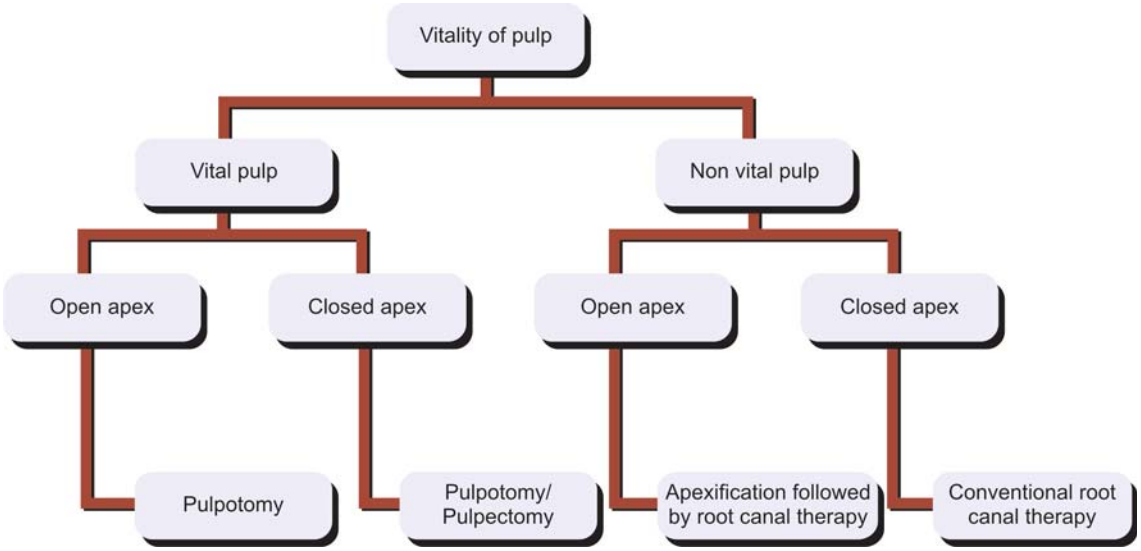


Fig. 19.11: Clinical decision of pulpal therapy in young permanent teeth

PULPAL THERAPY IN YOUNG PERMANENT TEETH (FIG. 19.11)

Vital Pulp Therapy for Teeth Diagnosed with a Normal Pulp or Reversible Pulpitis

a. Protective base.
b. Indirect pulp therapy.
c. Direct pulp capping
   - The above procedures are similar to as in primary teeth.

d. Partial pulpotomy for carious exposures:

The partial pulpotomy for carious exposures is a procedure in which the inflamed pulp tissue beneath an exposure is removed to a depth of 1 to 3 minor; in some cases, deeper to reach the healthy pulp tissue.

Pulpal bleeding must be controlled, and the site should be covered with calcium hydroxide or MTA. A restoration that seals the tooth from microleakage is placed.

Objectives:

- The remaining pulp should continue to be vital after partial pulpotomy.
- There should be no adverse clinical signs or symptoms such as sensitivity pain or swelling.
- There should be no radiographic sign of internal/ external resorption abnormal canal calcification, periapical radiolucency postoperatively.
- Teeth with immature roots should continue normal root development and apexogenesis.

e. Partial pulpotomy for traumatic exposures (Cvek's pulpotomy)

In traumatically exposed pulps, only tissue judged to be inflamed is removed Cvek has shown that with pulp exposures resulting from traumatic injuries, regardless of the size of the exposure or the amount of lapse time, pulpal changes are characterized by a proliferative response with inflammation extending only a few millimeters into the pulp. When the hyperplastic inflamed tissue is removed healthy pulp tissue is encountered.

Following amputation of coronal pulp in above methods; bleeding over exposure site is controlled using sterile cotton pellets and calcium hydroxide dressing is placed over exposure site and zinc oxide eugenol or any other suitable base is applied and finally restored with either silver amalgam or composite resin.

After pulpotomy, the patient should be recalled periodically to evaluate the success of the treatment.

f. Formocresol pulpotomy on young permanent teeth.
It is similar to as in primary teeth with similar rates of success.

Nonvital Pulp Treatment in Young Permanent Teeth

Pulpectomy

Pulpectomy in permanent teeth with closed apex is conventional root canal treatment for exposed, infected and/or necrotic teeth to eliminate pulpal and periradicular infection. In all cases, the entire roof of the pulp chamber is removed to gain proper access to the canals and eliminate all coronal pulp tissue.

Following debridement and shaping of the root canal system, obturation of the root canal is accomplished with a biologically acceptable, nonrestorable filling material. Obturation as close as possible to the cementodentinal junction should be accomplished with gutta percha or other filling material.

Apexogenesis and Apexification

Problems encountered with young permanent teeth:
The pulpal/root morphology of young permanent teeth has a significant role in deciding the treatment modality.

In young permanent teeth, presence of an open apex indicates absence of a hard tissue stop at the apex hence it is difficult to obdurate the canal within the apex and there is greater risk of extrusion of obturating material into periapex

Apicectomy also cannot be performed due to the fragility of developing roots.

Hence root canal treatment of these teeth requires a root end closure technique to form a barrier at the apex of the tooth, against which the tooth can be obturated successfully without extruding into periapical area.

Apexogenesis

Apexogenesis is the physiologic process of root development in teeth with inflamed coronal pulp and radicular pulp is relatively vital.

Apexogenesis is thereby a resultant physiologic phenomenon of pulpotomy procedure in total young permanent teeth with an open apex.

Procedure:

- From a sterile endodontic kit; Use not bur to gain access to the pulp chamber.
- Remove the pulpal tissue to the estimated level of the gingival crest of the bone, using a large sharp spoon excavator without causing undue trauma to remaining pulpal tissue.
- Rinse out all residual and dentinal debus.
- Usually the hemorrhage is fairly controlled, if not place sterile moist cotton pellets.
- Use calcium hydroxide applicator and place a mixture of calcium hydroxide over the pulp stump. Follow with a mixture of polycarboxylate cement.

Restore the tooth with composite resin; follow up with periodic revisits, including radiographs.

Apexification (Fig. 19.12)

- Also termed as "**Frank's procedure of root end closure**".
- It is indicated in non-vital young permanent teeth with open apex.

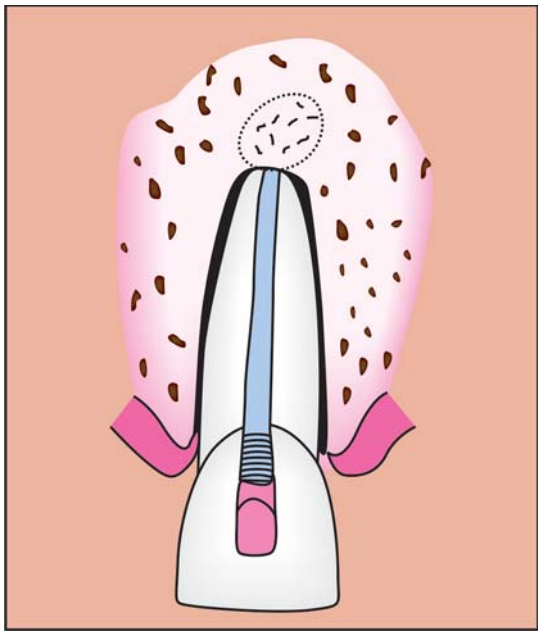
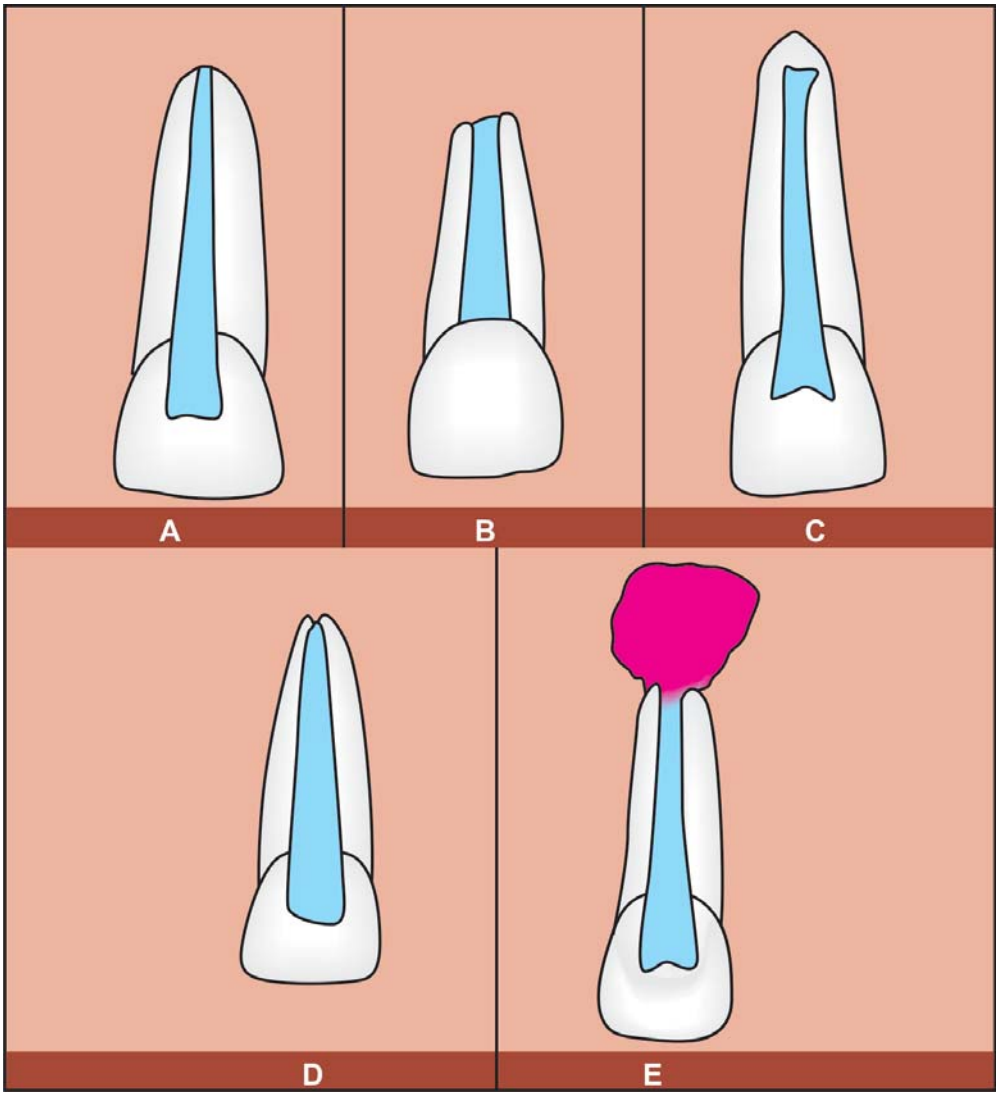


Fig. 19.12: Apexification



Figs 19.13A to E: Radiographic evidence of five alternatives to root end closure (Weine 1976). A. Resistance point without apparent apical closure. B. Calcified bridge at the apex. C. Apical closure without canal changes. D. Normal apical closure. E. Pathosis

Objectives:

- To induce root end –closure at the apices of immature roots, as evidenced by periodic radiographic evaluation.
- Post treatment clinical signs or symptoms such as sensitivity, pain or swelling should not be evident.
- There should be no radiographic evidence of abnormal calcification internal/external resorption or any periapical pathosis.

Procedure:

- Achieve profound anesthesia.
- After applying rubber dam gain access through lingual portion of the tooth.
- Using large reamers and files remove the debris from the coronal half of pulp and establish the file length radiographically.
- Clean the canal, irrigate it and dry it with sterile paper points.

- Seal a pellet of camphorated monochlorophenol (CMCP) within the pulp chamber with a provisional restoration.
- Recall the patient in 1-3 weeks, re-enter the tooth and clean the canals.
- Mix calcium hydroxide and CMCP and fill the canal completely using pluggers and obtain a radiograph to check the accuracy of the root canal filling.
- On a 6 month recall obtain radiographs to check for apical closure.
- When apical closure is achieved the root canal filling is completed as in routine procedure.

Outcome of apexification (Fig. 19.13).

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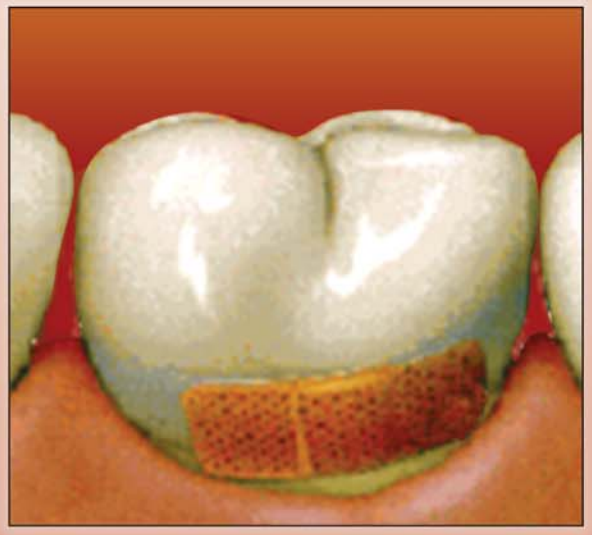


Trauma and its Management

INTRODUCTION

Orodental trauma in children is a cause of much pain and distress. Dental trauma in childhood and adolescence is common. Facial trauma that results in fractured, displaced or lost teeth can have significant negative, functional, esthetic and psychological effects on children.

Dentists and physicians should collaborate to educate the public about prevention and treatment of oral traumatic injuries.

EPIDEMIOLOGY

A reliable assessment of the distribution of orodental injury in the population is extremely difficult. There are two main reasons, firstly the data derived from treatment centers records only those patients who perceive a need for treatment and attend the clinic. Secondly, any epidemiological study only records injury which has a visible effect on the teeth.

INCIDENCE

The greatest incidence of trauma to the primary dentition occurs at 2 to 3 years of age, when motor coordination is developing. The most common injuries to permanent teeth occur secondary to falls, followed by traffic accidents, violence and sports.

According to a study boys are more prone to facial injuries than the girls do and patients with overjet are more common to get easily injured.

DISTRIBUTION OF INJURIES

The teeth most commonly damaged are the upper central incisors. The differential susceptibility of individual incisor teeth is 73% for upper centrals, 18% for lower centrals, 6% for lower laterals and 3% for upper laterals. Other teeth such as upper and lower canines represent only a very small proportion of damaged teeth. Upper central incisors are most commonly affected because they are, in general, the most prominent teeth and also relationship with increased overjet.

ETIOLOGY

1. Accidental injuries due to:
   - Falls, collisions, accidents, sports, violence, etc.
2. Non-accidental injury:
   - Physical child abuse.

Factors which influence the outcome or type of injury are a combination of:

- Energy impact
- Resilience of impacting object
- Shape of impacting object
- Angle of direction of impacting force.

CLASSIFICATION OF DENTOALVEOLAR INJURIES

Table 20.1 summarizes the classification of dentoalveolar injuries based on World Health Organization (WHO) system.

When a definite type of injury is referred to by a classification, it should be recognized by everyone. There are two types of classification.

Numerical Classification

It gives a number code for each type of injury. It has no real standardization and therefore inappropriate for general use and difficult to memorize and very confusing.

Descriptive Classification

This is more satisfactory as the location, type and extent of injury is given. This classification is based on WHO system and endodontic treatment needs.

Some of the classifications of traumatic injuries by various authors are summarized as follows:

Table 20.2 summarizes Ellis and Davey classification (1960).

Table 20.3 summarizes modification of Ellis classification by McDonald, Avery (1983).

Table 20.4 summarizes Andreason's classification.

Table 20.5 summarizes Garcia-Godoy's classification for traumatic injuries.

Table 20.6 summarizes Pulver's classification of traumatic injuries.

Table 20.1: WHO classification of nature of dentoalveolar injuries	
Injuries to the hard dental tissues and the pulp	
Enamel infarction	Incomplete fracture (crack) of enamel without loss of tooth substance
Enamel fracture	Loss of tooth substance confined to enamel
Enameldentine fracture	Loss of tooth substance confined to enamel and dentine not involving the pulp
Complicated crown-fracture	Fracture of enamel and dentine exposing the pulp
Uncomplicated crown root fracture	Fracture of enamel, dentine, and cementum but not involving the pulp
Complicated crown-root fracture	Fracture of enamel, dentine, and cementum and exposing the pulp
Root fracture	Fracture involving dentine, cementum, and pulp. Can be sub classified into: Apical Middle and Coronal(gingival) third's
Injuries to the periodontal tissues	
Concussion	No abnormal loosening or displacement but marked reaction to percussion
Subluxation	Abnormal loosening but no displacement
Extrusive luxation	Partial displacement of tooth from socket
Lateral luxation	Displacement other than axially with communition or fracture of alveolar socket
Intrusive luxation	Displacement into alveolar bone with communition or fracture of alveolar socket
Avulsion	Complete displacement of tooth from socket
Injuries to supporting bone	
Communition of mandibular or maxillary alveolar socket wall	Crushing and compression of alveolar socket. Found in intrusive and lateral luxation injuries
Fracture of mandibular or maxillary alveolar socket wall	Fracture confined to facial or lingual/palatal socket wall
Fracture of mandibular or maxillary alveolar process	Fracture of the alveolar process which may/or may not involve the tooth sockets
Fracture of mandible or maxilla	May/or may not involve the alveolar socket
Injuries to gingiva or oral mucosa	
Laceration of gingiva or oral mucosa	Wound in the mucosa resulting from a tear
Contusion of gingiva or oral mucosa	Bruising not accompanied by a break in the mucosa, usually causing sub mucosal hemorrhage
Abrasion of gingiva or oral mucosa	Superficial wound produced by rubbing or scraping the mucosal surface

Table 20.2: Ellis and Davey classification	
Class 1	Simple fracture of the crown involving little or no dentine
Class 2	Extensive fracture of crown involving considerable amount of dentine but not the dental pulp
Class 3	Extensive fracture of crown involving considerable dentine, and exposing the pulp
Class 4	The traumatized tooth that becomes non-vital with or without loss of crown structure
Class 5	Teeth lost as a result of trauma
Class 6	Fracture of the root with or without loss of crown structure
Class 7	Displacement of the tooth without fracture of crown or root
Class 8	Fracture of the crown enmasse and its replacement
Class 9	Injuries to primary teeth

Table 20.3: McDonald and Avery modification of Ellis classification of fracture	
Class 1	Simple fracture of the crown involving little or no dentine
Class 2	Extensive fracture of the crown involving considerable dentine; but not involving dental pulp
Class 3	Extensive fracture of the crown with an exposure of the dental pulp
Class 4	Loss of the crown

Table 20.4: Andreason's classification of traumatic injuries to teeth		
Injury	*Code*	*Description*
Injuries to hard dental tissues and pulp		
Enamel infarction	N 502.50	Incomplete fracture(crack) of enamel without loss of tooth substance
Enamel fracture	502.50	Loss of tooth substance confined to enamel
Enameldentine fracture	502.51	Loss of tooth substance confined to enamel and dentine not involving the pulp
Complicated crown fracture	N502.52	Fracture of enamel and dentine exposing the pulp
Uncomplicated crown-root fracture,	N502.54	Fracture of enamel, dentine, and cementum but not involving the pulp
Complicated crown-root fracture	502.54	Fracture of enamel, dentine, and cementum and exposing the pulp
Root fracture	N502.53	Fracture involving dentine, cementum, and pulp. Can be sub classified into: Apical Middle and Coronal(gingival) third's
Injuries to the periodontal tissues		
Concussion	N503.20	No abnormal loosening or displacement but marked reaction to percussion
Subluxation	N503.20	Abnormal loosening but no displacement
Extrusive luxation	N503.20	Partial displacement of tooth from socket
Lateral luxation	N503.20	Displacement other than axially with communition or fracture of alveolar socket
Intrusive luxation	N503.21	Displacement into alveolar bone with communition or fracture of alveolar socket
Avulsion	N503.22	Complete displacement of tooth from socket

Table 20.5: Garcia-Godoy's classification for traumatic injuries	
Class 0	Enamel crack
Class 1	Enamel fracture
Class 2	Enamel dentine fracture without pulpal exposure
Class 3	Enamel dentine fracture with pulp exposure
Class 4	Enamel dentine cementum fracture without pulpal exposure
Class 5	Enamel dentine cementum fracture with pulpal exposure
Class 6	Root fracture
Class 7	Concussion
Class 8	Luxation
Class 9	Lateral displacement
Class 10	Intrusion
Class 11	Extrusion
Class 12	Avulsion

Table 20.6: Pulver's classification of traumatic injuries	
Class I	
Division I	No external fracture, no displacement
Division II	Displacement but no fracture
Division III	Fracture of enamel only, no displacement
Division IV	Fracture and displacement
Class II	
Division I	Fracture of enamel and dentine only, no displacement
Division II	Displacement and fracture of enamel and dentine only
Class III	
Division I	Fracture with exposure of pulp, no displacement
Division II	Displacement and fracture with exposure of pulp
Class IV	
Division I	Fracture of root
Class V	
Division I	Intrusion
Division II	Partial avulsion
Division III	Complete avulsion

Descriptive Classification of Traumatic Injuries to Dentition (SHY Wei 1989)

Injuries of the Tooth

Crown:

- Crack or craze, horizontal or vertical without loss of tooth structure.
- Fracture of crown
  - In enamel only
  - Enamel and dentine
  - Enamel, dentine and pulp.
- Can be horizontal, vertical or oblique.
- Fracture of crown and root involving cementum with or without pulpal involvement.

Root:

- Apical, middle or cervical third
- Horizontal, vertical or oblique.

Whole tooth:

- Concussion
- Subluxation
- Displacement.

Injuries to supporting bone:

- Fracture of alveolar socket due to tooth intrusion
- Fracture of socket wall
- Fracture of alveolar process
- Fracture of maxilla
- Fracture of mandible.

INVESTIGATION OF ORODENTAL INJURIES

Any orodental injury is to be considered an emergency-prompt assessment and appropriate treatment are needed to ensure the best outcome.

A systematic approach is needed towards history taking, examination, diagnosis and treatment planning.

Table 20.7 summarizes the systematic assessment of acute traumatic injuries to teeth.

History

Medical History

Essential general medical questions are asked to the parent/guardian/patient. A pre-printed medical history

Table 20.7: Model sheet for assessment of acute traumatic injuries

Patient Name:	**Sex:**	**Date of birth:**
Referred by:	**Date :**	**Time:**

MEDICAL HISTORY

Allergies: Date of last tetanus inoculation:
Date and time of injury: Time lapsed since injury:
Where injury occurred?
How injury occurred?

Check if present and describe:

Non-dental injuries
Loss of consciousness
Altered orientation or mental status
Hemorrhage from nose/ears
Headache/nausea/vomiting
Neck pain
Spontaneous dental pain
Pain on mastication
Reaction to thermal changes
Previous dental trauma
Other complaints

EXTRAORAL EXAMINATION

Check if present and describe:

Facial fractures
Lacerations
Contusions
Swelling
Abrasion
Hemorrhage/drainage
Foreign bodies
TMJ deviation/asymmetry

OTHER FINDINGS /COMMENTS:

INTRAORAL EXAMINATION

Check if injured and describe:

Lips
Frenae
Buccal mucosa
Gingivae
Palate
Tongue
Floor of the mouth

DIAGRAM OF THE INJURY

Occlusion

Molar classification	R:	L:
Canine classification	R:	L:
Overbite (%)		
Overjet(mm)		
Cross bite	Y	N
Midline deviation	Y	N
Interferences	Y	N

DENTAL INJURIES

Avulsion	Extraoral time:	Storage medium:
Infarction		
Crown fracture		
Pulp exposure	Size:	Appearance:
Color		
Mobility (mm)		
Percussion		
Luxation	Direction:	Extent:
Pulp testing	Electrical:	Thermal:
Caries/previous restorations		

Contd...

Table 20.7: *Contd...*

Patient Name:	**Sex:**	**Date of birth:**
Referred by:	**Date :**	**Time:**

RADIOGRAPHS

- Pulp size
- Root development
- Root fracture
- Periodontal ligament space
- Periapical pathology
- Alveolar fracture
- Foreign body
- Developmental anomaly

SUMMARY

Check if performed and describe:

- Soft tissue management
- Medication
- Pulp therapy
- Repositioning
- Stabilization
- Restoration
- Extraction
- Prescription
- Referral
- Other

INSTRUCTIONS AND DISPOSITION

Check if discussed:

- Diet
- Hygiene
- Pain
- Swelling
- Infection
- Prescription
- Complications:
  - Damage to developing teeth
  - Abnormal position or ankylosis
  - Tooth loss
  - Pulp damage to injured teeth
  - Other
- Follow-up
- Other

sheet is useful, especially as it reduces the risk of omitting particular items.

Dental, Social and Family History

The child's dental experience will be a useful guide to their ability to cooperate when treatment is carried out. Social and family history will be given as to the parent's attitude to future treatment also incidence of any familial disorders and diseases, etc.

Trauma History

There are six specific questions:

- When did the injury take place?
- Where?
- How?
- Has treatment been provided elsewhere?
- Has there been previous trauma?
- Have all tooth fragments or avulsed tooth been accounted for?

Other important details to be noted are:

- Any other body part affected by trauma?
  - If yes? Where and which part and its status.
- Any chance of unconsciousness after the injury or bleeding or liquid running from ear, nose.

Examination

A thorough and detailed examination of the face, jaws, teeth, soft tissues is carried out, together with any special

tests and investigation, so that the extent of injuries may be established and a full diagnosis made. Pre-printed proformas provide a permanent record and serve as an useful aid.

Extraoral Examination

A general appraisal of the patient should be made and any cuts, abrasions, swelling or bruises noted. The bony borders of maxilla, mandible should be palpated, unless the extent of any edema makes this too uncomfortable.

In addition TMJ should be palpated during opening and closing of mouth, check for deviations in opening and closing.

Extraoral wounds, particularly of the lips should be palpated gently to detect the presence of any tooth fragments or foreign bodies.

Intraoral Examination

It includes a thorough soft tissue and hard tissue examination to note for any bruises, laceration, fracture, bleeding, dislocation, tenderness, swellings, etc. and charted out.

Special Investigations

It includes the following:

Assessment of vitality:

- Electrical pulp testing
- Thermal pulp testing
  - Heat and Cold
- Pulse oximetry
- Doppler flowmetry, etc.

Radiographic Examination

Use of extra and intraoral radiographs are indispensable in the diagnosis of orodental trauma. Some of the common extraoral views are:

- Panoramic tomography
- Lateral soft view
- Intraoral views
  - IOPA
  - Occlusal, etc.

The practitioner arrives at a diagnosis after going through history, examination and investigation and formulates an immediate and effective treatment plan to and attends upon the patient.

DESCRIPTION OF TRAUMA TO DENTAL STRUCTURES

General Reaction of Tooth to Trauma

- Pulpal hyperemia
- Internal hemorrhage
- Calcific degeneration
- Internal/external resorption
- Pulpal necrosis
- Ankylosis
- Damage to successional tooth (In case of primary teeth).

From the study of Andreasen and Raun (1971), the most important factor in determining whether damage to successional tooth will result or not seems to be the age of child, the highest incidence of damage takes place between 0 and 2 years to the developing successional tooth due to its close proximity to the primary tooth (Fig. 20.1).

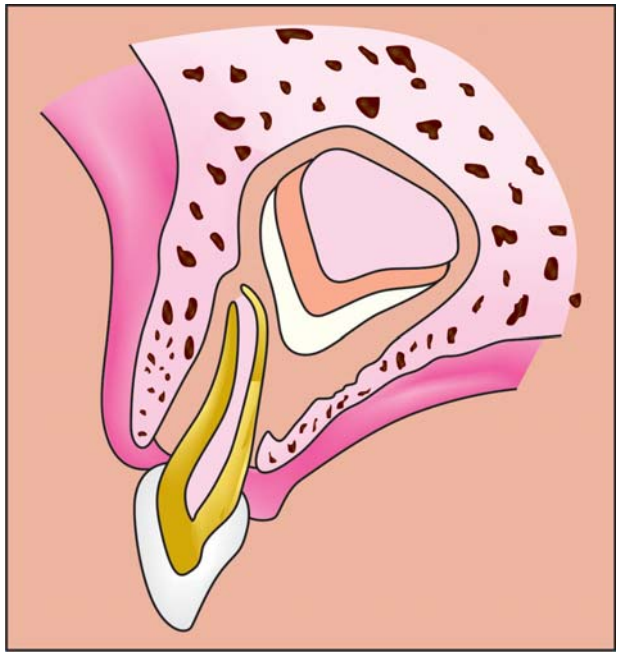


Fig. 20.1: Schematic representation of the proximity of the developing tooth bud in relation to primary teeth

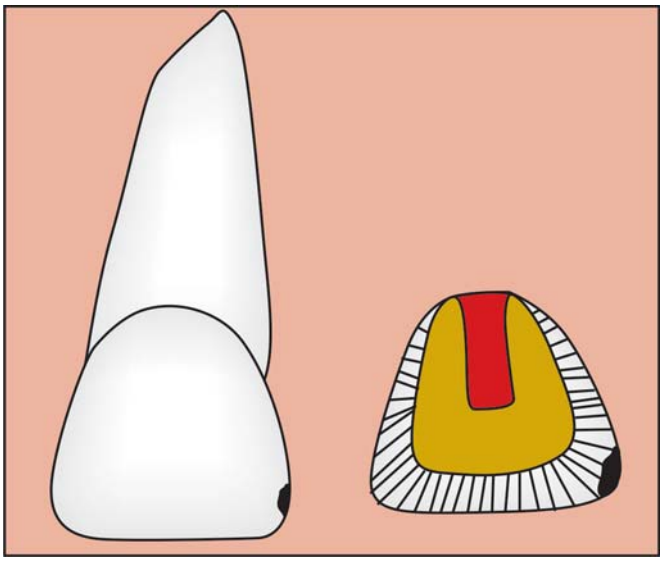


Fig. 20.2: Infarction

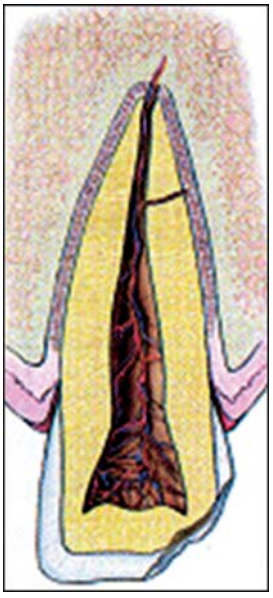


Fig. 20.3: Uncomplicated crown fracture

Dental Trauma

Infarction (Fig. 20.2)

Definition: Incomplete fracture (crack) of enamel without loss of the tooth structure.

Diagnosis: Normal gross anatomic and radiographic appearance, craze lines apparent especially with trans-illumination.

Treatment objectives: To maintain structural integrity and pulp vitality.

Treatment: Smoothen the edges and restore with composite/GIC.

Crown Fracture Uncomplicated (Fig. 20.3)

Definition: An enamel fracture or an enamel dentine fracture that does not involve the pulp.

Diagnosis: Clinical and/or radiographic findings reveal loss of tooth structure confined to enamel or to both enamel and dentine.

Treatment objectives: To main pulpal vitality and restore esthetics and function.

Treatment: Lost tooth structure restored with acid etched composite, if there is large fracture, restore lost tooth structure with open faced stainless steel crown.

Crown Fracture Complicated (Fig. 20.4)

Definition: Enamel-dentine fracture with pulp exposure.

Diagnosis: Clinical and radiographic findings reveal a loss of tooth structure with pulp exposure.

Treatment objective

- To maintain pulpal vitality.
- Restore normal esthetics and function.
- **Primary teeth:**
  - Decisions often are based on life expectancy of the traumatized primary tooth and vitality of the pulpal tissue.
- **Permanent teeth:**
- Decisions often are based on root maturity.

Treatment

- **Primary teeth**
  - Pulpal treatment alternatives are
    - Pulpotomy
    - Pulpectomy
    - Extraction
- **Permanent teeth:**
  - Direct pulp capping
  - Partial pulpotomy
  - Pulpectomy

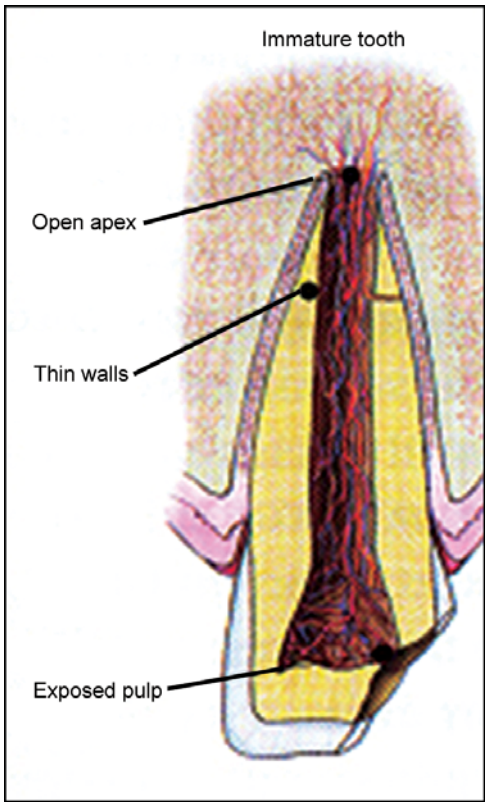


Fig. 20.4: Complicated crown fracture

Crown/root Fracture (Fig. 20.5)

Definition: An enamel, dentine and cementum fracture with or without pulp exposure.

Diagnosis: Clinical findings usually reveal mobility in coronal fragment attached to gingival with or without pulpal exposure.

Radiographic findings: Radiolucent oblique line that comprises crown and root in a vertical direction in primary teeth and in a direction usually perpendicular to central beam in permanent teeth.

Treatment Objectives

- To maintain pulp vitality.
- To restore normal esthetics and function.
- **Primary teeth:** When primary tooth cannot or should not be restored, the entire tooth should not be restored; the entire tooth should be removed unless retrieval of apical fragments may result in damage to the succadenous tooth.

Fig. 20.5: Crown/root fracture

- **Permanent teeth:**
  - Emergency treatment objective is to stabilize the coronal fragment.
    - Definitive treatment alternatives are to remove the coronal fragment followed by a supragingival restoration or necessary gingivectomy, osteotomy or surgical or orthodontic extrusion to prepare for restoration.
    - If pulp is exposed, pulpal treatment alternatives are pulp capping, pulpotomy or root canal treatment.
    - **General prognosis:** Fractures extending significantly below the gingival margin may not be restorable.

Root Fracture (Figs 20.6A to D)

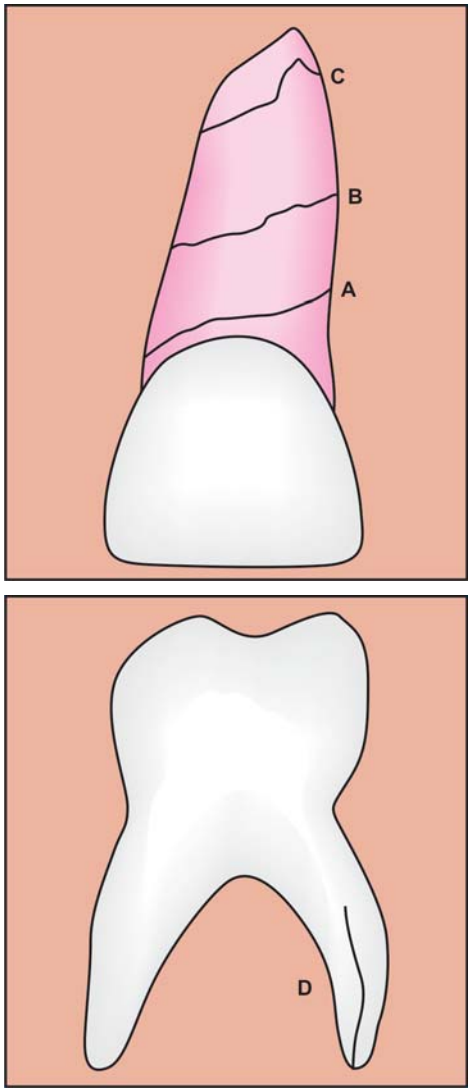
Definition: A dentine and cementum fracture involving the pulp.

Diagnosis

- Clinical findings reveal mobile coronal fragment attached to gingival that may be displaced.

Radiographic Findings

- 1 or more radiolucent lines that separate the tooth fragments in horizontal fractures.
- A root fracture in primary tooth may be obscured by a succedaneous tooth.



Figs 20.6Ato D: Root fracture. A. Coronal third, B. Middle third, C. Apical third and D. Vertical

Treatment Objectives

- To reposition the tooth as soon as possible.
- To stabilize the coronal tooth fragment in its anatomically correct position to optimize healing of the periodontal tissues and vascular supply.
- Maintain esthetic and functional integrity.

Treatment

- **Primary teeth**
  - Treatment alternatives include extraction of coronal fragment without insisting on removing apical fragment or observation.
  - Space maintenance after extraction.
- **Permanent teeth:**
  - Reposition and stabilize the coronal fragment by provisional splinting.
  - Flexible splinting assists in periodontal healing.

Ideal Characteristics of the Splint

- Easily fabricated in the mouth without additional trauma.
- Passive unless orthodontic forces are intended.
- Allows physiologic mobility (except for root fractures).
- Non-irritating to soft tissues.
- Does not interfere with occlusion.
- Allow endodontic access.
- Easily cleansed.
- Easily removed.

Instructions to Patients Having a Splint Placed

- Avoid biting on splinted teeth.
- Maintain meticulous oral hygiene.
- Call immediately if splint breaks/loosens.
- Use chlorhexidine/antibiotics as prescribed.

Acid Etched Bonded Splints (Fig. 20.7)

Technique:

- Infra occlude the involved tooth if needed, to prevent stress during occlusion.
- Adapt an orthodontic wire along facial surfaces extending up to 2-3 sound teeth either side (e.g. canine-to-canine if central incisor is involved).
- Etch the region of enamel in the area where the wire comes to contact using phosphoric acid.
- Apply bonding agent and cure.
- Apply the reinforced composite resin at the required area and place the stainless steel wire and cure simultaneously.

General prognosis:

- In permanent teeth, the location of the root fracture has not been shown to affect the pulpal survival after injury.

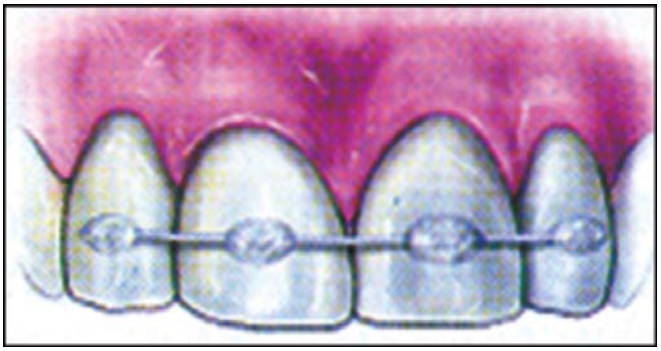


Fig. 20.7: Acid etched bonded splint

- Root fractures occurring in either cervical or apical third can be treated successfully but middle third has poor prognosis and should be extracted and replaced by prosthesis.

Concussion

Definition

- Injury to the tooth supporting structures without abnormal loosening or displacement of the teeth.

Diagnosis

- **Clinical findings reveal**
  - Tooth is tender to pressure and percussion without mobility, displacement or sulcular bleeding.
- **Radiographic findings**
  - No radiographic abnormalities detected.

Treatment Objectives

- To optimize healing of periodontal ligament and maintain pulp vitality.

General prognosis

- **Primary teeth**
  - Unless associated infection exists.
  - No pulpal therapy is indicated.
- **Permanent teeth:**
  - Although there is a minimal risk for pulpal necrosis, mature permanent teeth with closed apices may undergo pulpal necrosis due to associated injuries to the blood vessels at the apex and therefore must be followed carefully.

Subluxation

Definition: Injury to tooth supporting structures with abnormal loosening but without tooth displacement.

Diagnosis

- Clinical findings reveal a mobile tooth without displacement that may or may not have sulcular bleeding.
- Radiographic abnormalities are not expected.

Treatment objectives

- To optimize healing of the periodontal ligament and neurovascular supply.
- **Primary teeth:**
  - The tooth should be followed for pathology.
- **Permanent teeth:**
  - Stabilize the tooth and relieve any occlusal interference. For comfort, a flexible splint can be used.
  - Splint for no more than 2 weeks.

General prognosis

- Prognosis is usually favorable. The primary tooth should return to normal within 2 weeks. Matured permanent teeth with closed apices may undergo pulpal necrosis due to associated injuries to the blood vessels at the apex and, therefore, must be followed carefully.

Lateral Luxation

Definition: Displacement of the tooth in a direction other than axially. The periodontal ligament is torn or contusion or fracture of the supporting alveolar bone occurs.

Diagnosis: Clinical findings reveal that a tooth is displaced laterally and may be lucked firmly into this new position. The tooth usually is not mobile or tender to touch.

Radiographic findings reveal an increase in periodontal ligament space and displacement of apex toward or though the labial bone plate.

Treatment objectives:

- **Primary teeth:**
  - To allow reposition or actively reposition and splint for 1-2 weeks as indicated to allow for healing,

except when the injury is severe or the tooth is nearing exfoliation.

- **Permanent teeth:**
  - To reposition as soon as possible and then to stabilize the tooth in its anatomically correct position to optimize healing of the periodontal ligament and neurovascular supply, while maintaining esthetic and functional integrity.
  - Repositioning of the tooth is done with little force and digital pressure. The tooth may need to be extruded to free apical lock in the cortical bone plate. Splinting an additional 2 to 4 weeks may be needed with breakdown of marginal bone.

General prognosis

- Primary teeth requiring repositioning have an increased risk of developing pulp necrosis compared to teeth that are left to spontaneously reposition.
- In mature permanent teeth with closed apices, there is considerable risk for pulp necrosis and progressive root resorption.

Intrusion (Fig. 20.8)

Definition: Apical displacement of tooth into the alveolar bone. The tooth is driven into the socket, compressing the periodontal ligament and commonly causes a crushing fracture of alveolar socket.

Diagnosis

- **Clinical findings reveal**
  - Tooth appears to be shortened or in severe cases, it may appear missing.
  - Tooth's apex usually is displaced labially toward or through the labial bone plate in primary teeth and driven into alveolar process in permanent teeth.

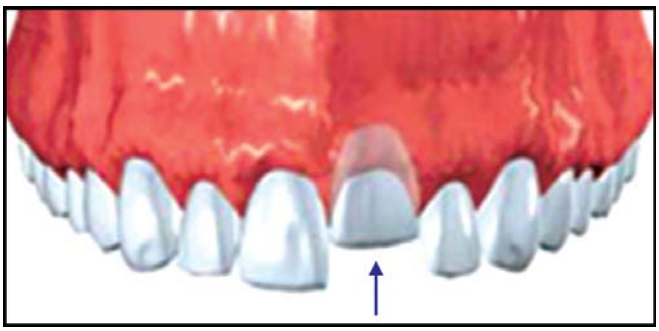


Fig. 20.8: Intrusive luxation

- **Radiographic findings reveal that:**
  - Tooth appears displaced apically and periodontal ligament space is not continuous.
  - Determination of the relationship of an intruded primary tooth with the follicle of the succedaneous tooth is mandatory.
  - If the apex is displaced labially, the apical top can be seen radiographically with the tooth appearing shorter than it's contralateral.
  - If the apex is displaced palatally towards the permanent tooth germ, the apical tip cannot be seen radiographically and the tooth appears elongated.
  - An extraoral lateral radiograph also can be used to detect displacement of the apex toward or through the labial bone plate.
  - An intruded young permanent tooth may mimic an erupting tooth.

Treatment Objectives

- **Primary teeth**
  - To allow spontaneous re-eruption except when displaced into the developing successor.
  - Extraction is indicated when the apex is displaced toward the permanent tooth germ.
- **Permanent teeth**
  - To reposition passively or actively and stabilize the tooth in its anatomically correct position to optimize the healing of periodontal ligament and neurovascular supply while maintaining esthetic and functional integrity.
  - In teeth with immature root formation, the objective is to allow for spontaneous eruption.
  - In matured teeth, the goal is to reposition the tooth with orthodontic or surgical extrusion and initiate endodontic treatment within the first 3 weeks of the traumatic incidence.

- **General Prognosis:**
  - In primary teeth, 90% of intruded teeth will re-erupt spontaneously in 2 to 6 months. Even in cases of complete intrusion and displacement of primary teeth through labial bone plate, a retrospective study showed the re-eruption and survival of most teeth for more than 36 months.

- Ankylosis may occur, however, if the periodontal ligament of the affected tooth was severely damaged, thereby delaying or altering the eruption of the permanent successor.
- In mature permanent teeth with closed apices, there is considerable risk for pulp necrosis and progressive root resorption.

Extrusion (Fig. 20.9)

Definition: Partial displacement of the tooth axially from the socket. The periodontal ligament is usually torn.

Diagnosis:

- Clinical findings reveal
  - Tooth appears elongated and is mobile.
- Radiological findings revel
  - Increased periodontal ligament space apically.

Treatment objectives

- **Primary teeth**
  - To re-position and allow healing, except when there are indications for an extraction.
  - If the treatment decision is to reposition and stabilize, splint for 1 to 2 weeks.
- **Permanent teeth:**
  - To reposition as soon as possible and then to stabilize the tooth in its anatomically correct position to optimize healing of the periodontal ligament and neurovascular supply while maintaining esthetic and functional integrity.
  - Repositioning may be accomplished with slow and steady apical pressure to gradually displace coagulum formed between root apex and floor of the socket.
  - Splint for up to 3 weeks.

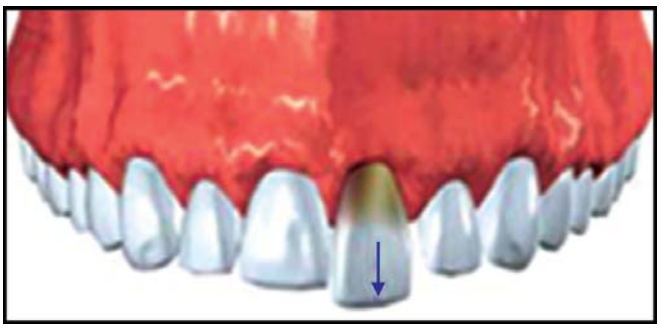


Fig. 20.9: Extrusive luxation

General prognosis

- There is lack of clinical studies evaluating repositioning of extruded primary teeth.
- In mature permanent teeth with closed apices, there is considerable risk for pulp necrosis. These teeth must be followed carefully, if necessary root canal treatment is advocated.

Avulsions (Figs 20.10 to 20.12)

Definition: Complete displacement of tooth out of socket. The periodontal ligament is severed and fracture of the alveolus may occur.

Diagnosis

- Clinical and radiographic findings reveal that the tooth is not present in the socket or tooth already has been replanted.
- Radiographic assessment will verify that the tooth is not intruded when the tooth was not found.

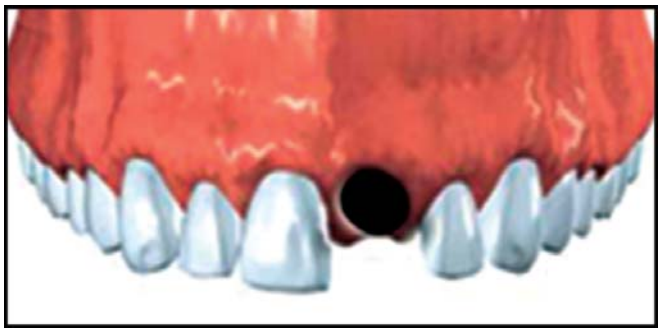


Fig. 20.10: Avulsion

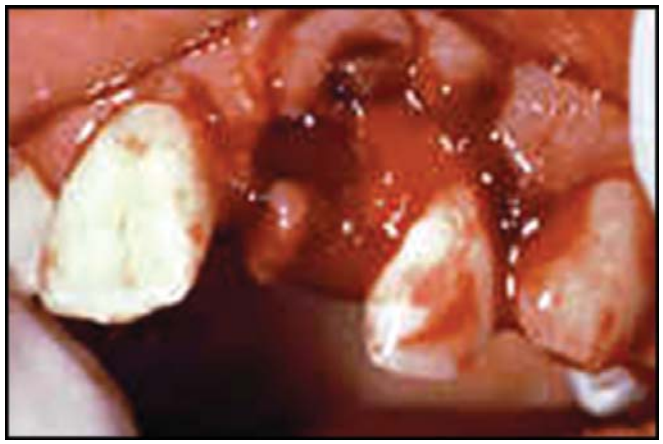
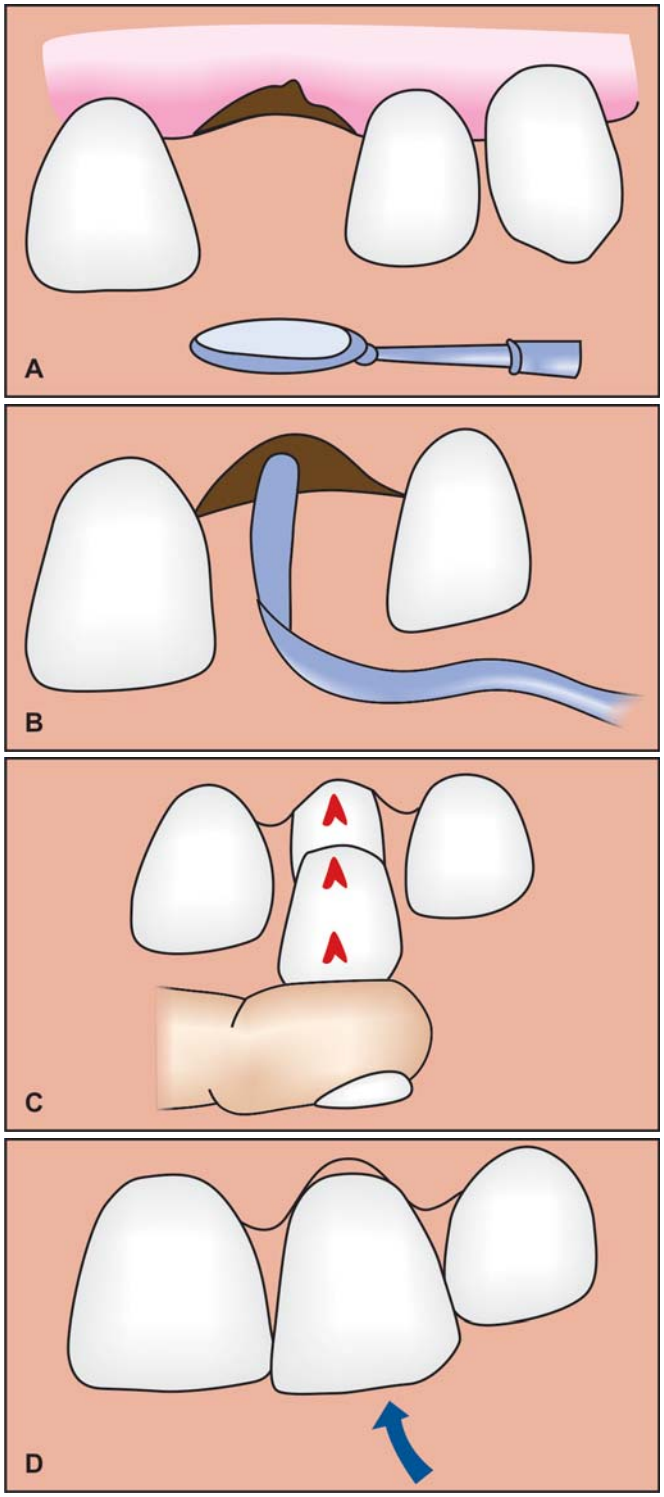


Fig. 20.11: Avulsion injury



Figs 20.12A to D: Reimplantation of the avulsed tooth. A. Inspection of the socket, B. Debridement and curettage of the socket after administration of LA, C. Re-insert the tooth with light finger pressure and D. Re-inserted tooth may appear longer than its normal counter part, proceed to splinting

Treatment Objectives

- **Primary teeth**
  - To prevent further injury to the developing successor. Avulsed primary teeth should not be replanted because of the potential for subsequent damage to developing permanent tooth germs, and pulpal necrosis is a frequent event.
- **Permanent teeth**
  - To replant as soon as possible and then to stabilize the replanted tooth in its anatomically correct location to optimize healing of the periodontal ligament and neurovascular supply while maintaining esthetics and functional integrity except when replanting is contraindicated by:
    - Child's stage of dental development (risk of ankylosis where considerable alveolar growth has to take place).
    - Compromising medical condition.
    - Compromised integrity of the avulsed tooth or supporting tissues.
  - Flexible splinting for 1 week is indicated (Fig. 20.13).
  - Tetanus prophylaxis and antibiotic coverage should be considered.
- **General prognosis**
  - Prognosis in the permanent dentition is primarily depending upon formation of root development and extraoral dry time.
  - The tooth has best prognosis if replanted immediately. If the tooth cannot be replanted within 5 minutes, it should be restored in a

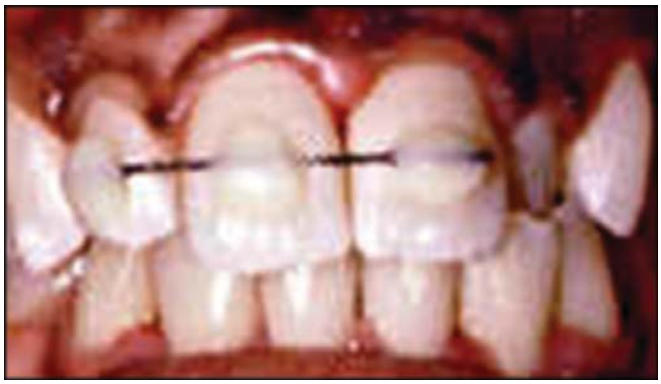


Fig. 20.13: Flexible splinting following reinsertion of avulsed tooth

medium that will help maintain vitality of periodontal ligament fibers.

- Transportation media for avulsed teeth include (in order of preference):
  - Viaspan
  - Hank's balanced salt solution (Tissue culture medium)
  - Cold milk
  - Saliva (buccal vestibule)
  - Physiologic saline
  - Tender coconut water
  - Water
  - Risk of ankylosis increases significantly with an extraoral dry time of 15 minutes.
- **Additional considerations:** There are possible contraindications to tooth replantation. Examples are:
  - Immunocompromised conditions
  - Severe congenital anomalies
  - Severe uncontrolled seizure disorder
  - Severe mental disability
  - Severe uncontrolled diabetes
  - Lack of alveolar integrity.

Resorption

Root resorption is a serious and a destructive complication which may follow trauma to primary and permanent teeth. Primary teeth which develop pathological resorptive lesions are not eligible for conservative treatment and should be extracted. Permanent teeth on other hand may often be successfully treated provided tissue destruction has not advanced to unrestorable state.

Two general types of pathological root resorption are recognized. They are:

- Inflammatory root resorption
- Replacement root resorption.

Inflammatory Root Resorption

Following trauma injured internal and external root surfaces are rapidly colonized by multinuclear giant cells; if these cells are continuously stimulated by microbial interactions from infected canal/periodontal pocket it results into inflammatory root resorption.

Inflammatory root resorption is classified based upon its site of origin as

- External root resorption (Fig. 20.14)
- Cervical root resorption (Fig. 20.15)
- Internal root resorption (Fig. 20.16).

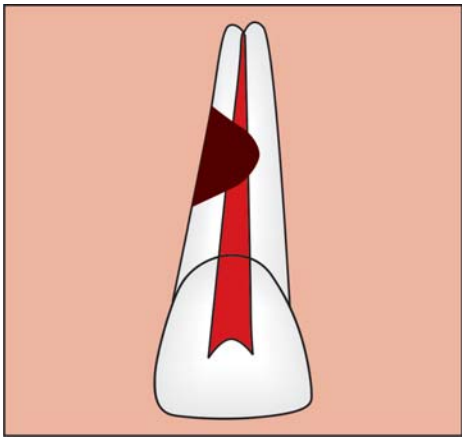


Fig. 20.14: External root resorption

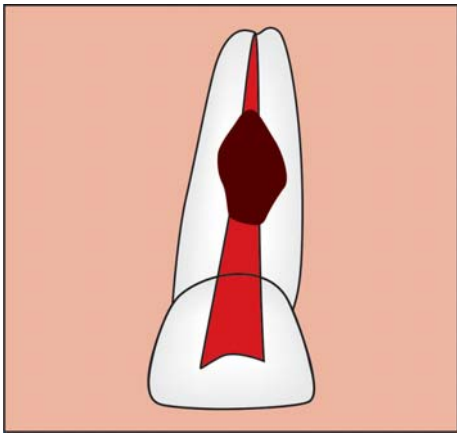


Fig. 20.15: Cervical root resorption

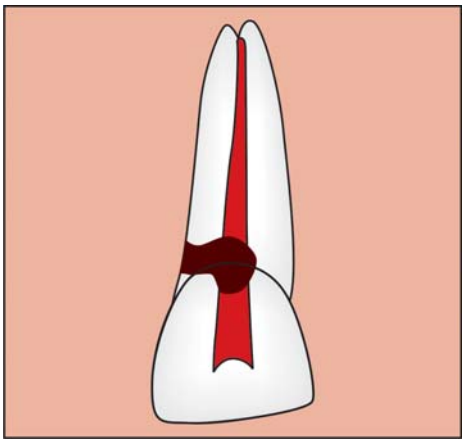


Fig. 20.16: Internal root resorption

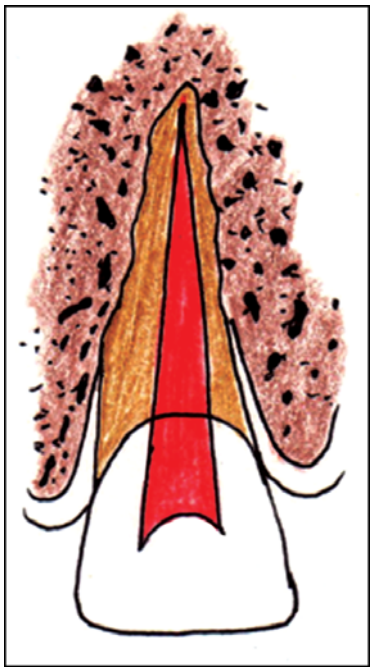


Fig. 20.17: Replacement resorption/ankylosis

Replacement Resorption (Fig. 20.17)

It is a distinct form of root resorption that follows serious luxation on avulsion injury that has caused damage to the investing periodontal ligament.

If more than 20% of the periodontal ligament is damaged or lost and the tooth is subsequently reimplanted, bone cells are able to grow into contact with root surface at a faster rate than the remaining periodontal fibroblasts are able to recolonize the root surface and intervene between tooth and bone.

The consequence is that the root gets involved in remodeling of bone in which it is implanted and is gradually replaced by bone over the course of following years.

In young children where the bone remodeling is high, the root may be entirely lost within 3-4 years. In adolescents, it may be 10 years or more before the tooth is lost.

Splinting

Trauma may loosen a tooth either by damaging the periodontal ligament or fracturing the root. Splinting immobilizes the tooth in the correct anatomical position so that further trauma is prevented and healing can occur. Different injuries require different splinting regimens. A functional splint involves one, and a rigid splint two abutment teeth either side of the injured tooth.

Periodontal Ligament Injuries

This injury heals within 10 days and is complete within a month. The splinting period should be as short as possible. The splint should allow some functional movement to prevent replacement root resorption. As a general rule avulsion injuries require 7-10 days and luxation injuries 2-3 weeks of functional splinting.

Root Fractures

These require three to four weeks of functional splinting. Coronal third fractures and individual cases may require longer splinting. Excessive mobility leads to the fracture site becoming filled with granulation tissue.

- **Dentoalveolar fractures:** These require three to four weeks of rigid splinting.

Types of Splinting

- Composite resin/acrylic and wire splint
- Approximal composite/ acrylic resin splint
- Orthodontic brackets and wire
- Foil/cement
- Laboratory splints
  - Acrylic
  - Thermoplastic.

Provision of Mouth Guards in Sports

Dental injuries in children should be considered as serious, since injuries to teeth and jaws that are not fully developed can lead to growth being adversely affected, causing lifelong problems. Studies have shown a dramatic reduction in the number of dental injuries when a mouth guard is worn. All persons especially children and adolescents involved in contact sports, should be encouraged to wear a mouth guard.

Criteria for Mouth Guard Construction

The Federation Dentaire Internationale has listed the following criteria for constructing an effective mouth guard (FDI 1990). The mouth guard should be:

- Made of a resilient material that can be easily washed, cleaned and readily be disinfected.

- Have adequate retention to remain in position during sporting activity, and allow for a normal occlusal relation ship to give maximum protection.
- Absorb and disperse the energy of a shock by covering the entire maxillary dental arch; excluding interferences; reproducing the occlusal relationship.
- Allow mouth breathing.
- Protect the soft tissues.

The FDI also recommends that mouth guards should, preferably, be made by dentists from full arch impressions of teeth.

Mouth Guard Design

The accepted design is based on that suggested by Turner (1977).

- The mouth guard is normally fitted to the maxillary arch, except in class III malocclusion patients.
- It should be a close fitting and should cover the occlusal surfaces of all the teeth except where it is anticipated that the exfoliation of primary teeth and further eruption of teeth will occur.
- The flanges of the mouth guard should extend beyond the level of attached gingivae but fall short of the muco-buccal fold.
- The flange should be no greater than 2 mm thick over the labial mucosa to avoid stretching of lips, which may otherwise split on impact.
- The buccal edge of the flange should be smooth and rounded and carefully relieved around frenal and muscle attachments.
- The palatal aspect of the mouth guard should extend approximately 5 mm on to the palate and should be tapered to a smooth, thin, rounded edge to avoid interference with speech and breathing or stimulation of the 'gagging' reflex.
- The occlusal thickness of the guard should not exceed the width of the freeway space.
- There should be cusp indentations for the opposing teeth to facilitate and even comfortable occlusion with the opposing arch.

Types of Mouth Guards

Mouth guards can be divided into two main types:

1. Mouth formed, and
2. Custom made.

Both the types are constructed from preformed thermoplastic shells of either polyvinyl-polyethylene (PVAc-PE) copolymer or polyvinylchloride (PVC)

Care of Mouth Guards

Bacteriological studies have led to the recommendation that mouth guards should be:

- Washed with soap and water immediately after use
- Dried thoroughly and stored in a perforated container
- Rinsed in a mouth wash or a mild antiseptic immediately before repeat use.

Special Considerations in Mouth Guard Design

- Partially dentate individuals should not wear removable prostheses while participating in sports, in order to prevent injury and aspiration of fragments if the appliance were to fracture.
- Occlusal rims may be constructed on a thermoplastic base to replace the missing teeth.
- Individuals undergoing fixed orthodontic treatment can have a mouth guard constructed provided that the brackets and arch wires are covered with wax prior to taking an impression.
- Care should be taken not to place the lips or soft tissues under tension by making the mouth guard too thick.

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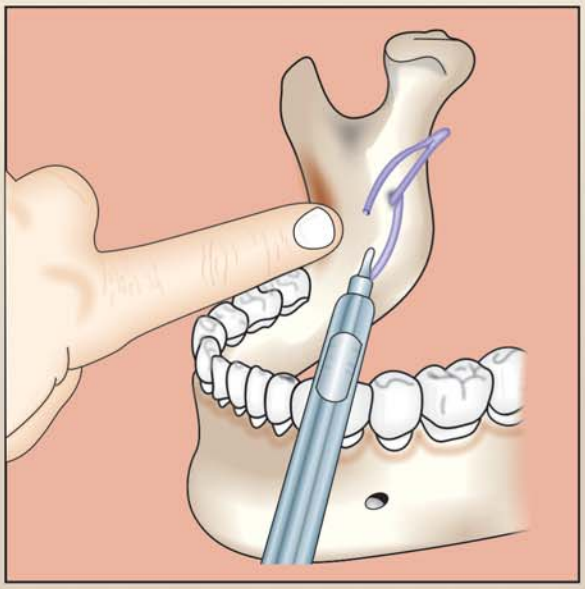

Oral Surgical Considerations in Children

INTRODUCTION

Management of pain and anxiety are interrelated factors that hold a greater role in treating a pediatric patient. The control of pain while performing dental procedures for the child is one of the most fundamentals and important component supporting, sound principles of behavior management.

Pain control is achieved by means of skilled and sensible local anesthetic delivery technique. The successful administration of the anesthetic agent is based on the concept of psychological preparation and a skillful administration of the agent and a sound knowledge of anatomical landmarks and innervations of teeth is also inevitable.

In pediatric dentistry, the dental professional should be aware of proper dosage to minimize the chance of toxicity and the prolonged duration of anesthesia, which can lead to accidental lip or tongue trauma. Familiarity with the patient's medical history is essential to decrease the risk of aggravating a medical condition while rendering dental care.

LOCAL ANESTHESIA

Malamed (1980) defines local anesthesia as "**a loss of sensation in a circumscribed area of the body caused by a depression of excitation in nerve endings or an inhibition of the conduction process in peripheral nerves".**

Many local anesthetic agents are available to facilitate management of pain in dental patient. There are 2 general types of local anesthetic chemical formulations.

1. **Esters**
   - Procaine
   - Benzocaine.
2. **Amides**
   - Lidocaine
   - Mepivacaine
   - Prilocaine
   - Articaine.

Duration of Action and Potency

The duration of action of local anesthetic is directly proportional to protein binding characteristics agents that are highly protein bound (etidocaine, Bupivacaine) have the longest duration of action, whereas those with lower protein binding capacities (Lidocaine, Mepivacaine) have shorter durations of action.

Mechanism of Action (Fig. 21.1)

Local anesthetic agents are weak bases; they are commonly combined with a strong acid (HCl) to improve water solubility, tissue diffusibility and stability in solution.

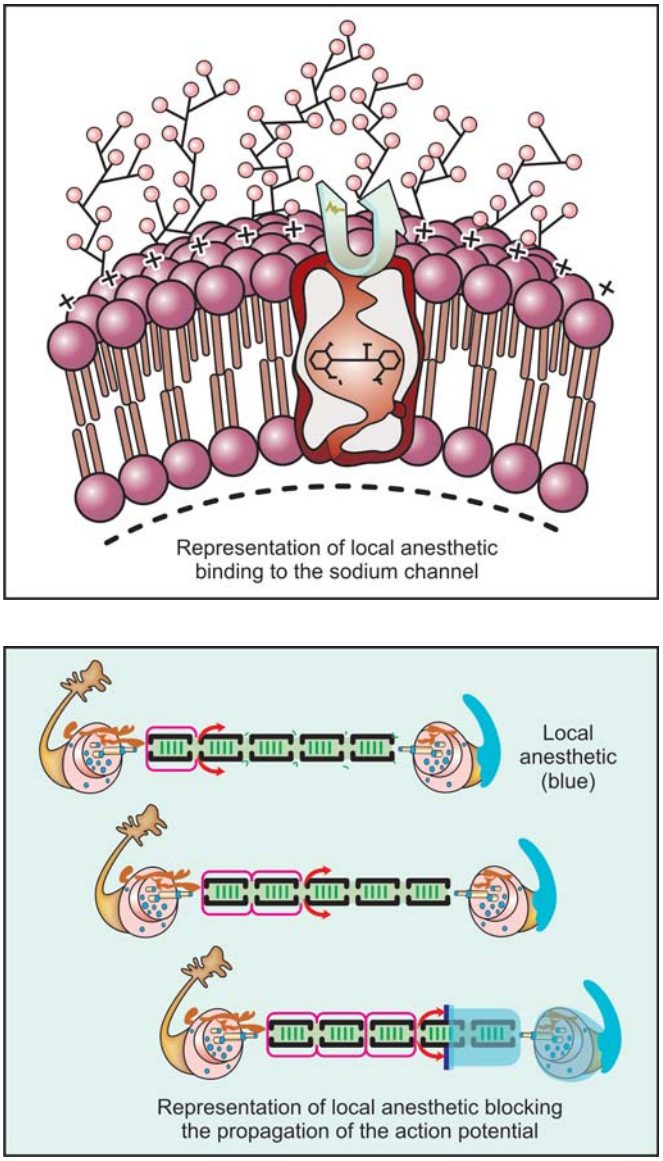

Fig. 21.1: Mechanism of action of local anesthetic agent

When acid is injected into the tissue, it interacts with tissue buffers, forming free base, or unionized from which permits diffusion of the anesthetic agent across the nerve membrane; where it dissociates into ionized form.

It in turn interferes with the conduction of action potential along the peripheral nerve fibers by impairing the functions of sodium ion channels.

Composition of Local Anesthetic Agent (Fig. 21.2)

- Local anesthetic agent — Lidocaine, Prilocaine, etc.
- Vasoconstrictor — epinephrine/norepinephrine
- Reducing agents — prevents oxidation of the vasoconstrictor, e.g. Sodium metabisulphite
- Preservatives — Preserves the stability of local anesthetic agent, e.g. Caprylhydrocuprienotoxin, Methylparaben.
- Fungicide — A small quantity of Thymol
- Vehicle/Solvent — Modified Ringer's solution That comprises of sodium chloride that maintains isotonicity and water that acts as solvent.

Fig. 21.2: Local anesthetic agent

Topical Anesthetics

The application of topical anesthetic may help to minimize the discomfort caused during administration of local anesthesia. Topical anesthetic is effective on surface tissues (2-3 mm in depth) to reduce painful needle penetrations of the oral mucosa when using Intraoral topical anesthetics.

1. The area of application should be dried.
2. The anesthetic should be applied over a limited area.
3. The anesthetic should be applied for sufficient time.

A variety of topical anesthetic agents are available in gel, liquid, ointment, patch and aerosol forms.

The topical anesthetics available are

- 20% benzocaine
  - Has a rapid onset of action
- 50% Lidocaine (Solutions, ointment)
- 10% Lidocaine (spray).

AAPD Recommendations

1. Topical anesthetic may be used prior to the injection of a local anesthetic to reduce discomfort associated with needle penetration.
2. The pharmacological properties of the topical agent should be understood.
3. A metered spray is suggested if an aerosol preparation is selected.
4. Systematic anesthetic must be considered when calculating the total amount of anesthetic administered (topical Lidocaine is absorbed systematically and can combine with an injected amide local anesthetic to increase the risk of overdosage).

Topical Anesthetic that will Anesthetize Skin

EMLA Cream

- 5% eutectic mixture of the anesthetic agents' Prilocaine and Lidocaine.
- It was the first topical anesthetic to be shown to produce effective surface anesthesia of intact skin.
- Useful adjunct in providing topical anesthesia for pain free venepuncture.

AAPD Guidelines on Selection of Syringes at Needles

The American Dental Association (ADA) has established standards for aspirating syringes for use in the administration of local anesthesia. Needle selection should allow for profound local anesthesia and adequate

aspiration. Larger gauge needles provide for less deflection as needle passes through soft tissues and for more reliable aspiration.

The depth of insertion varies not only by injection technique, but also by the age and size of the patient. Dental needles are available in 3 lengths:

- Long (32 mm)
- Short (20 mm)
- Ultra short (10 mm)

Needle gauges range from size 23 to 30.

Needles should not be bent or inserted to their hub for injections to avoid needle breakage.

AAPD Guidelines on Injectable Local Anesthetic Agents

Local amide anesthetics available for dental usage include (Tables 21.1 and 21.2):

- Lidocaine
- Mepivacaine
- Articaine
- Prilocaine
- Bupivacaine

Absolute contraindications for local anesthetics include a documented local anesthetic allergy. True allergy to an amide group is exceedingly rare. Allergy

Table 21.1: Injectable local anesthetics

	Duration in min 3, 17						
	Maxillary infiltration		*Mandibular block*		*Maximum dosage*		*Maximum total*
Anesthetic	*Pulp*	*Soft tissue*	*Pulp*	*Soft tissue*	*mm/kg*	*mg/lb*	*dosage (mg)*
Lidocaine					(–) 4.4	2.0	300
2% plain	5		5-10				
2%+1:50,000 epinephrine	60	170	85	190			
2%+1:100,000 epinephrine	60	170	85	190			
Mepivacaine					4.4	2.0	300
3% plain	25	90	40	165			
2%+1:100,000 epinephrine	60	170	85	190			
2%+1:20,000 levonordefrin	50	130	75	185			
Articaine					7.0	3.2	500
4%+1:100,000 epinephrine	60	190	90	230			
Prilocaine					6.0	2.7	400
4% plain	20	105	55	190			
4%+1:200,000 epinephrine	40	140	60	220			
Bupivacaine					1.3	0.6	90
0.5%+1:200,000 epinephrine	40	340	240	440			

Table 21.2: Dosage per dental cartridge

Anesthetic	*mg/1.8 ml cartridge*	*Vasoconstrictor/1.8 ml cartridge*
Lidocaine		
2% plain	36	N/A
2%+1:50,000 epinephrine	36	36 µg or 0.036 mg
2%+1:100,000 epinephrine	36	18 µg or 0.018 mg
Mepivacaine		
3% plain	54	N/A
2%+1:100,000 epinephrine	36	18 µg or 0.018 mg
2%+1:20,000 levonordefrin	36	90 µg or 0.090 mg
Articaine		
4%+1:100,000 epinephrine	72	18 µg or 0.018 mg
Prilocaine		
4% plain	72	N/A
4%+1:200,000 epinephrine	72	9 µg or 0.009 mg
Bupivacaine		
0.5%+1:200,000 epinephrine	9	9 µg or 0.009 mg

to one amide does not rule out the used of another amide but allergy of one ester rules out the use of another ester.

A bisulfate preservative is used in local anesthetics with epinephrine. For patients having an allergy to bisulfate, use a local anesthetic without a vasoconstrictor.

A long acting local anesthetic (Bupivacaine) is not recommended for children or physically or mentally disabled patient due to its prolonged effect, which increases the risk of soft tissue injury.

Prilocaine may be contraindicated in patient with methemoglobinemia, sickle cell anemia or symptoms of hypoxia or in patients receiving acetaminophen or phenacetin, since both medications elevate methemoglobin levels.

ADA Recommendations

1. Selection of LA agents should be based upon:
   a. Patient's medical history and mental/development status.
   b. Anticipated duration of dental procedures.
   c. Need for hemorrhage control.
   d. Planned administration of other agents.
   e. Practitioner's knowledge of the anesthetic agent.
2. Use of vasoconstrictors in LA is recommended to decrease the risk of toxicity of the anesthetic agent.
3. The established maximum dosage for any anesthetic should not be exceeded.

Alternative Modes to Achieve Local Anesthesia

Use of controlled released devices such as Intraoral patches comprising of Lidocaine which are under clinical trial.

Jet Injectors

Belong to a category between topical and local anesthesia. These devices allow anesthesia of surface and to a depth of over 1 cm without the use of needle. They deliver a jet of solution through the tissue under high pressure.

Electroanalgesia or TENS (Transcutaneous Electrical Nerve Stimulation)

Electroanalgesia or TENS has been very effective in providing anesthesia for restorative procedures in children aged between 3 and 12 years. The technique has also been used to provide pain control during the extraction of primary teeth.

INNERVATIONS OF PRIMARY TEETH

Effective local anesthesia for the maxillary and mandibular teeth and their related structures first requires familiarity with the manner in which these areas are innervated.

Superior Alveolar Branches of Maxillary Nerve

- External division of posterior superior alveolar nerve innervates the buccal mucosa and mucous membrane of the cheek.
- Middle superior alveolar nerve when present (a branch of infraorbital nerve) innervates the root ends and supporting structures of the primary first and second molars.
- Anterior superior alveolar branch raises short distance posterior to the external orifice of the infraorbital canal. It innervates anterior wall of maxillary sinus and maxillary primary incisors and canine root ends and periodontal structures.

Anterior Palatine and Nasopalatine Branches of the Maxillary Nerve

- Anterior palatine nerve supplies sensory fibers to the tissues of the hard palate.
- Nasopalatine branches innervate quadrant and hard tissues of the anterior quadrant from midline laterally to the maxillary canine tooth.

Inferior Alveolar Nerve (Fig. 21.3)

- The only means to accomplish adequate pulpal anesthesia for all mandibular teeth is through the use of nerve block procedure.
- Anatomic change is elicited through normal growth and development process. As an example, the location of mandibular foramen in the child is inferior

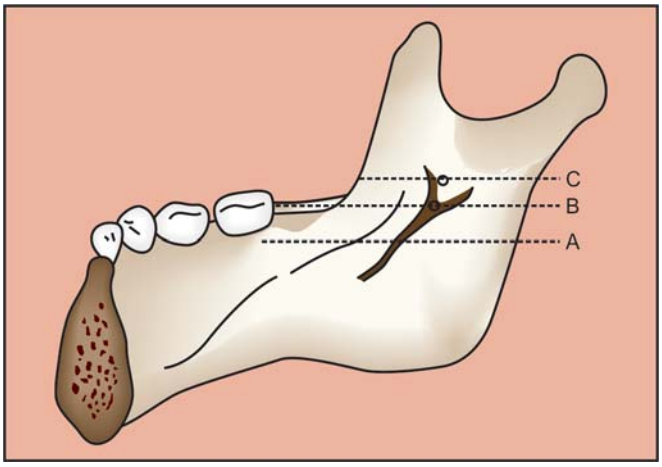

Fig. 21.3: Position of mandibular foramen to locate the inferior alveolar nerve. A. Position below occlusal plane at 3 years of age, B. Position at the level of occlusal plane at 6 years of age and C. Position above the level of occlusal plane in patients over 12 years of age

to its site in an older individual and also the mandibular ramus is narrower anteroposterior in child than in adult. The factors cited require adjustments in the steps of the block procedure.

- The line of delivery of syringe will be in a slightly downwards direction to accommodate the more inferior location of the foramen.

Mental and Incisive Nerves

- Emerges as the terminal branches of inferior alveolar nerve.
- Mental nerve exits from mental foramen and innervates the skin and mucosa of the lower lip and chin as well as the gingival tissues labial to the mandibular incisors.
- Incisive branch furnishes sensory fibers to mandibular incisors and canine.

Long Buccal Nerve

- Sensory fibers are distributed to the buccinator muscle, buccal mucosa and buccal gingivae over mandibular molars and second premolar.

Lingual Nerve

- Sensory innervation to the floor of the mouth and gingival of the lingual surface of the body of the mandible.

General Techniques

Use of Assistant

- Assistant should be ready at all times to restrain hands; position the child control movements.
- Assistant can help block view and keep the patient distracted.

Use Topical Anesthetic

- Use appropriate topical anesthetic
- Allow atleast 1 minute for application.

Patient Position

- Position the patient seated in a dental chair reclined about 30° to the vertical for extractions under local anesthesia.
- When removing upper teeth, the operator stands in front of the patient, with a straight back, except that the patient's mouth is at a height just below operator's elbow.
- A right handed operator removes lower left teeth from a similar position in front of the patient except that the patient's mouth is at a height just below the operator's elbow.
- When removing teeth from lower right the right handed operator stands behind the patient with the chair as low as possible to allow good vision.

Body Control

- Operator should be in control of patient's head and hands.

The Non-working Hand

- It has important roles to play.
- It retracts soft tissues to allow visibility and access.
- It protects the tissues if the instrument slips.
- It provides resistance to the extraction force on the mandible to prevent dislocation
- It provides 'feel' to the operator during the extraction and gives information about resistance to removal.

Syringe Management and Etiquette

- Hide it; do not wave it in front of the child.

- Block patient's view with retracting hand
- Be confident.

Euphemisms

- Topical anesthetic → Tooth jelly
- Local anesthetic → Sleepy juice/medicine
- Needle prick → Mosquito bite, pinch
- Numbness → Tooth will take a nap and feel fat and fuzzy.

Distraction

- Reduces or relieves patient anxiety and discomfort. A behavior management technique.

Order of Extraction

When performing multiple extractions in all quadrants of the mouth, the order of extraction is as follows:

- Symptomatic teeth are extracted before 'balancing extraction' on the opposite side.
- Lower teeth are extracted before upper teeth.
- If there are symptomatic teeth in all quadrants right handed operators should begin with lower right extractions. This minimizes the number of changes of the position of the surgeon, which will reduce anesthetic time.

Specific Injection Sites for Children

Inferior alveolar block (Fig. 21.3):

- Injection site is lower and more posterior and do not need to penetrate tissue as far as in adults.
- The mandibular foramen is located below the occlusal plane in children less than 3 years and those above 3 years it is seen at the level of occlusal plane and in older individuals it is seen above the level of occlusal plane. This variation is due to the relative growth and development of mandible, thereby anatomic position of mandibular foramen varies according to age in relation to anatomic landmarks.

Infiltrations (Fig. 21.4):

- Infiltrations work in mandibular anterior although block may be best for posterior extractions.
- Infiltrations are best and effective in maxillary primary teeth and permanent premolars.

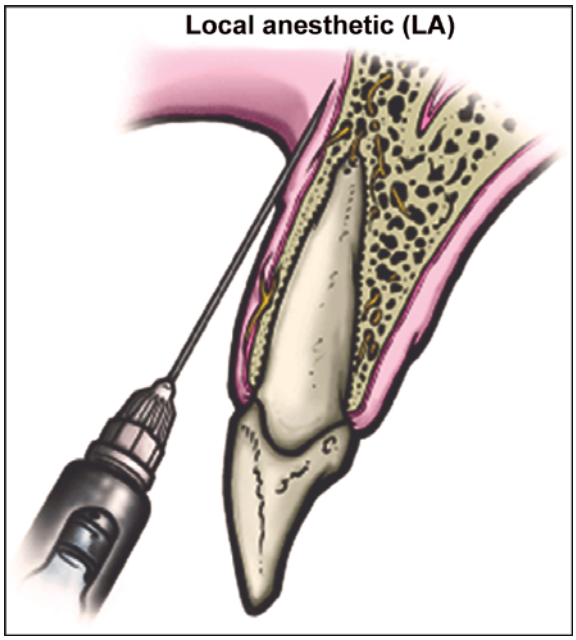

Fig. 21.4: Infiltration anesthesia

- Apices of maxillary primary anterior teeth are at depth of mucobuccal fold; inject at depth of mucobuccal fold using short or extra short needle.
- Inject over the apices of maxillary primary molars.

Interdental papilla of sulcus:

- To achieve palatal anesthesia. Inject as you go through papilla from facial to lingual aspect; should see blanching as you inject.
- Inject in sulcus starting in distal interproximal to mesial interproximal to achieve palatal anesthesia.
  Wait until anesthetic action has set in and check for any symptoms of pain if present and then proceed for extraction.

Use of Forceps and Force Applied

Upper primary anteriors:

- Apply forceps beaks to the root and then using clockwise and anticlockwise rotations along long axis.
- In older children some additional buccal expansion may be required.

Upper primary molars:

- Initial palatal movement to expand the socket and tooth in then subjected to continuous buccally directed force, which results in delivery.

Lower primary anteriors:

- Similar to upper anteriors.

Lower primary molars:

- These teeth are removed by buccolingual expansion of the socket.
- After application of the force beats at the bifurcation of the root, a small lingual movement followed by a continuous buccal force delivers the tooth.

Key Points to be noted during and after Extraction

- Avoid excessive force application to avoid extraction/displacement of underlying developing permanent tooth.
- After extractions ensure that the tooth has completely been extracted and no tooth/root fragments left inside the socket.
- Check the extracted socket for tooth/root remnants.
- Composes the socket after the extraction and approximate the bony walls of the socket bucco-lingually.
- Place a sterile gauze pressure pack to prevent hemorrhage and facilities clot formation.

Postextraction Instructions

- Advice patient/parent not to spit saliva for 24 hours and to swallow saliva.
- Advice patient not to remove the cotton pack until 30 minutes after extraction.
- Do not consume any hard hot food stuffs and patient should be on cold and soft diet.
- Instruct the parent/patient about persisting numbness of lips and tongue for transient period and advice not to chew or bite the tongue of lips.
- Instruct the patient to take necessary medication as directed.

MINOR ORAL SURGICAL PROCEDURES PEDIATRIC DENTISTRY

Soft Tissue Abnormalities

Labial Frenum

- A prominent midline frenum in the maxilla may be present in association with diastema. It is nothing but a band of fibroblastic tissue with high attachments.
- Frenectomy is advocated in above case.

Indications:

- Gingival recession.
- Persistent midline diastema, etc.

Technique:

- Place excisions, perpendicular to the frenum in the mucobuccal fold and extend around the frenum in both directions until the incision is carried to the bone.
- The tissue delineated is excised, the surface of the exposed bone in the interdental space should be curetted or gently burred to remove residual fibrous attachments.
- Primary closure of the labial part of incision is achieved by suturing and the defect in attached gingival is covered with either a periodontal dressing or ribbon gauze soaked in whitehead's varnish, which is held in place by sutures.
- The pack is removed 7-10 days after surgery.

Lingual Frenum

- A prominent lingual frenum should be excised if it is interfering with speech or oral hygiene. This is simply performed under local anesthesia.
- The frenum is held by a pair of hemostatic forceps, a triangular section of tissue is removed, and the wound ends sutured.

Mucocele (Fig. 21.5)

- Mucocele is an extravasation type cyst caused by trauma to minor salivary glands with mucous/fluid spreading into adjacent tissues and has a fibrous lining.

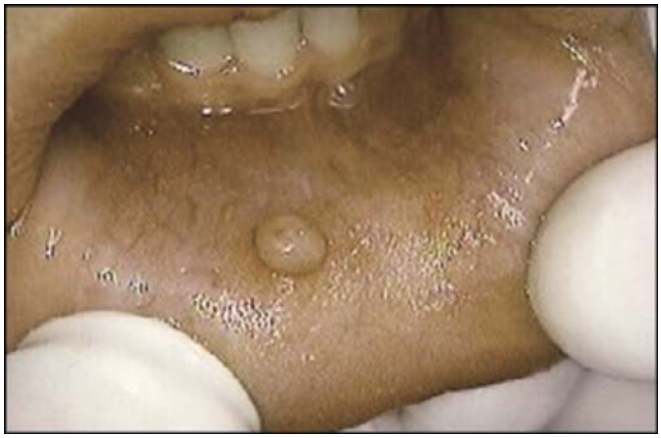

Fig. 21.5: Mucocele

- Mucocele is commonly seen in lower lip area due to accidental biting of lower lip or any injury.

Excision

Common approaches are:

- Place an elliptical incision around the lesion and thoroughly excise the entire lesion.
- Place a blunt dissection superficially over the lesion and the lesion is excised entirely.
- Tissue around the lesion is trimmed in little excess to prevent recurrence; in case of deep lesions suturing is essential to eliminate dead space.

Ranula

- Ranula is another type of retention cyst that is found is the floor of the mouth located in sublingual space between mylohyoid muscle and lingual mucosa.
- It is formed by retention of fluid in the submaxillary gland or their ducts.

Treatment:

- This lesion is often more extensive and complete cure occasionally involves the sublingual gland. The best method is marsupilization.

Eruption Cyst

- Associated with natal teeth and also in relation with deciduous teeth and permanent teeth.
- Appear as clear or blood tinged fluid filled sac on the alveolar crest.
- It interferes with feeding or may be asymptomatic.
- It is usually transient in existence and usually resolves as soon as the involved tooth erupts. If not it should be deroofed and drained. If infected the offending natal tooth is also extracted.

Hard Tissue Abnormalities

Extraction of Supernumerary Teeth

Technique:

- Localization of the tooth using SLOB technique radiologically. Anesthetize the area.
- Place crevicular incisions in the involved area either buccally or palatally.
- Raise the flap and remove the bone covering the supernumerary teeth and once enough clearance is available deliver the tooth from the space.
- Trim the walls of bony cavity.
- Irrigate well with saline and reapproximate the flaps and suture.

Extraction of Natal Teeth

- Natal teeth are often asymptomatic, however they may cause discomfort during feeding or may cause trauma of soft tissues opposing the natal teeth.

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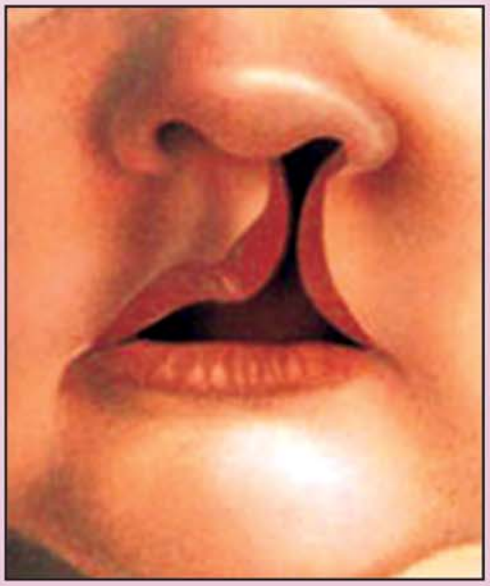


Comprehensive Management of Cleft Lip and Palate

INTRODUCTION

Birth of a child is considered a happiest moment of parent's life and a magical experience where god entitles a mother power of creation. The birth of a child with a cleft represents a crisis for parents who may experience a range of emotions such as shock, numbness, grief and anger. It takes time for the reality of the situation to sink in. The joy of child's birth may soon be overshadowed.

Cleft lip and palate is one of the most common congenital malformations. A child born with cleft lip/palate or other craniofacial anomalies has multiple and complex problems, including:

- early feeding and nutritional concerns
- middle ear disease
- Hearing deficiency
- Speech disturbances
- Dentofacial/Orthodontic abnormality
- Psychosocial problems, etc.

INCIDENCE

Cleft lip and palate are among the most common of birth defects; approximately one newborn in 700-800 live births has cleft lip/palate or both. Cleft lip and palate is more frequent in females.

ETIOLOGY

In spite of the vast literature on the etiology of cleft lip + palate anomaly, the knowledge on the subject is still insufficient and a confused one.

The anomaly is said to be of multifactor inheritance and mostly occurs due to an interaction of both genetic and environmental factors.

Following are some of the suggested etiologies of cleft lip and palate:

Genetics

- Single gene mutations
- Chromosomal aberrations.
- Familial inheritance.

Environmental

- Teratogens like thalidomide, phenytoin, etc.
- Trauma during pregnancy affecting the fetus
- Nutritional disturbances
- Infectious diseases
- Defective vascularity.

Unknown Causes

- Mechanical obstruction by tongue.

EMBRYOLOGY OF CLEFT LIP AND PALATE

The critical period of fetal development, when genetic and environmental factors can influence clefts is from fourth to ninth week of pregnancy. The lip and primary palate develop at 4-6 weeks of gestation, while secondary palate develops at around 9 weeks.

The overall development of palate involves the formation of primary palate followed by formation of secondary palate. At approximately 30-37 days gestational age, the primary palate forms by growth and fusion of medial nasal, lateral nasal and maxillary processes (Fig. 22.1).

The maxillary process, derived from the proximal half of the first arch, grows to meet and fuse with the nasal processes that have grown and moved in association with olfactory placode. General opinion holds that meso-dermal penetration underlies the formation of the

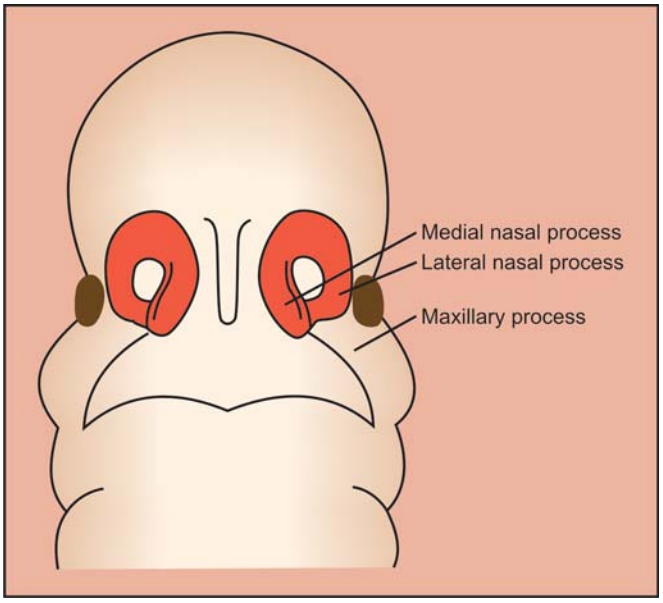


Fig. 22.1: Embryology

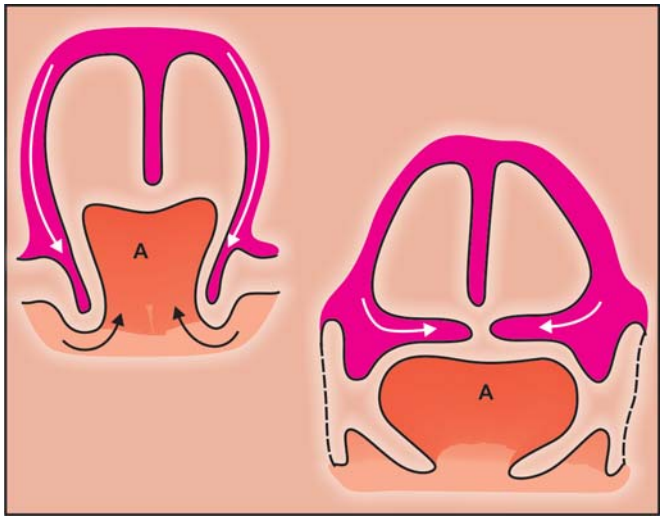


Fig. 22.2: Formation of palate. A. Position of tongue. Arrow indicates the direction of growth

primary palate. Mesodermal re-inforcement along lines of fusion is important as epithelial breakdown and clefting is though to result from lack of re-inforcement.

The secondary palate arises from 2 palatal shelves, which initially are in a vertical position because of the interposed tongue with the extension of the head at 7 weeks GA and mandibular growth, the tongue is withdrawn and the palatal shelves can swing into a more horizontal and midline position for fusion and formation of hard and soft palate (Fig. 22.2). The cleft of the hard and soft palate is said to occur because of the intervening tongue, which impedes the elevation of the palatal shelves.

CLASSIFICATION OF CLEFT LIP/PALATE (FIG. 22.3)

Various classification schemes have been devised in the last 7 decades, but few have received widespread clinical acceptance. They are:

Davis and Ritchie Classification

Each of the following subgroup is further subdivided into the extent of the cleft (1/3, ½ etc.):

Group I: Clefts anterior to the alveolus
- Unilateral
- Median
- Bilateral cleft lip

Group II: Post-alveolar clefts
- Cleft palate alone
- Soft palate alone
- Soft and hard palate or
- Submucous cleft.

Group III: Alveolar clefts, unilateral, bilateral/median.

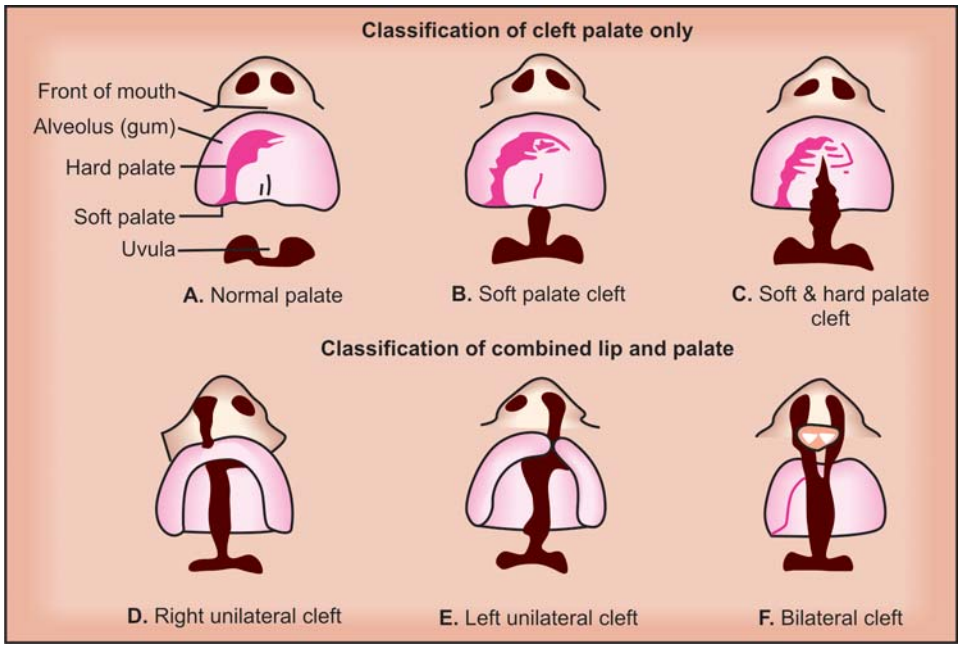


Fig. 22.3: Classification of cleft lip and palate

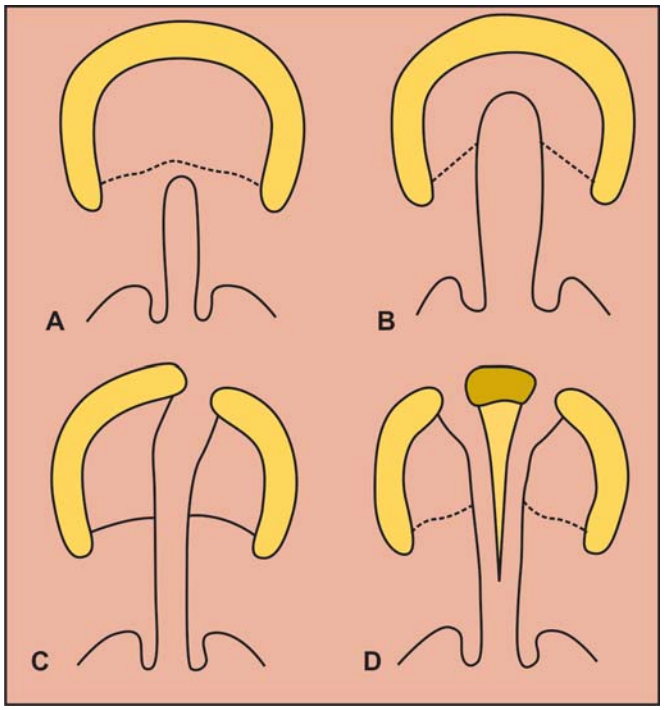


Fig. 22.4: Veau's classification (refer text for description)

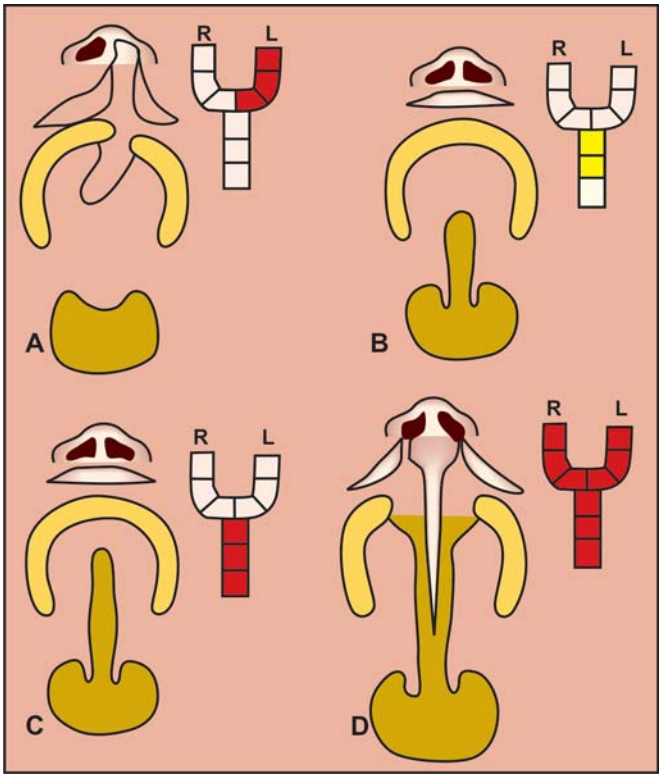


Fig. 22.5: Kernahan and Stark classification

Veau Classification (Fig. 22.4)

Group I (A) : Defects of the soft palate alone.
Group II (B) : Defects involving hard palate and soft palate.
Group III (C) : Defects involving soft palate an alveolus alone with lip.
Group IV (D) : Complete bilateral clefts.

Kernahan and Stark Classification (Fig. 22.5)

This classification highlights the anatomic and embryonic importance of incisive foramen formed during 4-7 weeks GA. The secondary palate forms the root of the mouth from the incisive foramen to the uvula during 7-12 weeles GA.

This system provides a symbolic/graphic classification scheme using a Y-configuration which can be divided into 9 areas.

- Areas 1 and 4 : Lip
- Areas 2 and 5 : Alveolus
- Areas 3 and 6 : Palate between the alveolus and incisive foramen
- Areas 7 and 8 : Hard palate
- Area 9 : Soft palate.

Harkins and Associates (1962)

Harkins and associates (1962) at the instigation of the American Cleft Palate Association presented a classification of facial clefts based on the same embryologic principles used by Kernahan and Stark. A modified version follows:

I. Cleft of primary palate.
A. Cleft lip:
1. Unilateral - right, left
   a. Extent- 1/3, 2/3, complete
2. Bilateral: right, left
   a. Extent-1/3, 2/3, complete
3. Median
   a. Extent-1/3, 2/3, complete
4. Prolabium: Small, medium, large
5. Congenital scar: Right, left, median.
   a. Extent-1/3, 2/3, complete

B. Cleft of alveolar process
1. Unilateral - Right, left
   a. Extent- 1/3, 2/3, complete
2. Bilateral: Right, left
   a. Extent-1/3, 2/3, complete
3. Median
   a. Extent-1/3, 2/3, complete
4. Submucous: Right, left, and median
5. Absent incisor tooth

II. Cleft of palate:

A. Soft palate
1. Posteroanterior : 1/3, 2/3, complete
2. Width-maximum (mm)
3. Palatal shortness: None, slight, moderate, marked.
4. Submucous cleft.
   a. Extent-1/3, 2/3, complete

B. Hard palate
1. Posteroanterior : 1/3, 2/3, complete
2. Width-maximum (mm)
3. Vomer attachment : Right/left/absent
4. Submucous cleft : 1/3, 2/3, complete

III. Mandibular process clefts:

A. Lip : 1/3, 2/3, complete
B. Mandible : 1/3, 2/3, complete
C. Lip pits: Congenital lip sinuses.

IV. Naso-ocular:Extending from the nasal towards the medical canthal.

V. Oro-ocular:Extending from the angle of the mouth towards palpebral tissue.

VI. Oro-aural: Extending from the angle of the mouth towards the auricle.

Spina (1974)

Spina (1974) modified and simplified above classification as follows:

Group I : Preincisive foramen clefts
Clefts of the lip with or without an alveolar cleft
A. Unilateral
B. Bilateral
C. Median

Group II : Transincisive foramen clefts (cleft of lip, alveolar, palate)
A. Unilateral
B. Bilateral

Group III : Post-incisive foramen clefts

Group IV : Rare: Facial clefts

International Confederation of Plastic and Reconstructive Surgery Classification

This system used an embryonic criterion to divide clefts into 4 groups with further subdivision to denote unilateral or bilateral cases.

Group I : Defects of the lip/alveolar

Group II : Clefts of the secondary palate (Hard palate, soft palate or both).

Group III : Any combination of cleft's involving the primary and secondary palates.

COMPLICATIONS AND PROBLEMS ASSOCIATED WITH CLEFTS

1. Aesthetic: Facial disfigurement
2. Dental complication:
   - Congenitally missing teeth.
   - Presence of supernumerary teeth
   - Anomalies of tooth morphology
   - Poor periodontal support
   - Orthodontic complications
     - Rotations
     - Posterior/anterior cross bites
     - Protruding premaxilla
     - Deep bite
     - Spacing
     - Crowding
3. Hearing and speech disturbances
4. Recurrent nasal infections
5. Feeding difficulties leading to malnourishment
6. Psychological stress.

COMMON SYNDROMES ASSOCIATED WITH CLEFT LIP AND PALATE

Though CLP may occur as a separate entity, it is also an occurrence along with various syndromes.

Autosomal Dominant Inheritance

- Clefting-Ankyloblepharon filiform adentum syndrome.
- Ectodactyly, Ectodermal dysplasia, clefting syndrome.
- Popliteal pterygium syndrome
- Van der Woude syndrome

Autosomal Recessive Inheritance

- Appelt syndrome
- Bixler syndrome
- Bownen-Armstrong syndrome
- Juberg-Haward syndrome
- Robert syndrome.

Environment

- Fetal hydantoin syndrome
- Fetal trimethadione syndrome
- Clefting ectropions syndrome.

Unknown Genesis

- Pilloto syndrome
- Yong syndrome.

MANAGEMENT OF CLEFT LIP AND PALATE

The management of CLP patient is no one man show and has led to the concept of a multidisciplinary cleft palate team. Ideal treatment facilities can be provided with combined efforts of a cleft team in which "parent" also holds an important position in treating a cleft child. The cleft team comprises of

- A Pediatrician
- A Pedodontist
- An Orthodontist
- Oral and maxillofacial surgeon
- Prosthodontist
- Audiologist and speech therapist
- Psychiatrist
- Parent
- ENT surgeon
- Plastic surgeon
- Geneticist

ROLE OF PEDODONTIST IN MANAGEMENT OF CLP

A role of Pedodontist in treating a cleft child is emphasized in following areas of concerns starting from:

- Feeding and nutritional advice
- Dental management
- Psychological reinforcement of parent and patient.

Nutrition and Feeding Cleft Child

A cleft child faces difficulty to suckle and encounters problems of nasal regurgitation and malnourishment, etc.

Advice Proper Feeding Techniques

Breastfeeding

Each baby is unique, so different techniques will suit different babies even if they have same kind of cleft. However, some general tips on breastfeeding are:

- Ensure if baby requires a prosthetic feeding appliance and in fabricated appropriately.
- Babies require longer feeding time.
- Lot of air is suckled through cleft and results in aerophogia which requires frequent burping after every ½ oz of feed.
- Mothers must be advised to observe the infant for chocking, cyanosis and abdominal distension during feeding.
- Infants must be held at 30-45° position.

Bottle Feeding (Fig. 22.6)

- Advise the mother to hold the baby at 30-45° angulation or in an upright position to prevent aspiration and to aid in swallowing.
- Use of appropriate clean teats with adequate slit the most comfortable in cross slit cut the end of the teat.

Following are the various types of teats that are designed for cleft child:

Mead Johnson cleft palate nurser (Fig 22.7)

- A very soft bottle that can be squeezed to help the flow of milk to which any standard teat in mixed.
- It reduces amount of effort required for baby and amount of air swallowed during feeding.

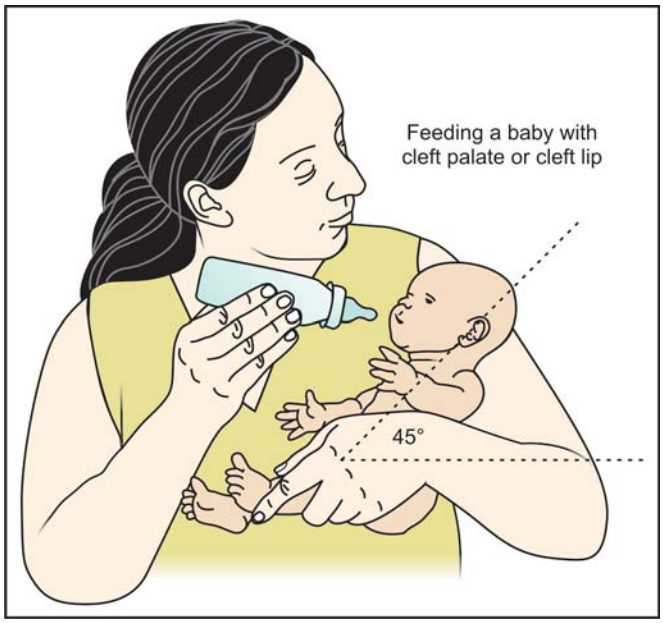


Fig. 22.6: Bottle feeding a child with cleft

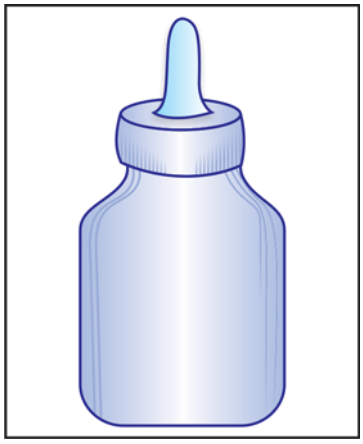


Fig. 22.7: Mead Johnson cleft palate nurser

- Easily re sterilized /reused by normal methods.
- Inexpensive.

Haberman feeder (Fig. 22.8)

- Normal bottle with a soft variable flow teat and pump action valve that requires no active suction.
- Milk flows into teat through a valve and cannot flow back into the bottle.
- Flow of milk is controlled by rotating the nipple in infant's mouth.

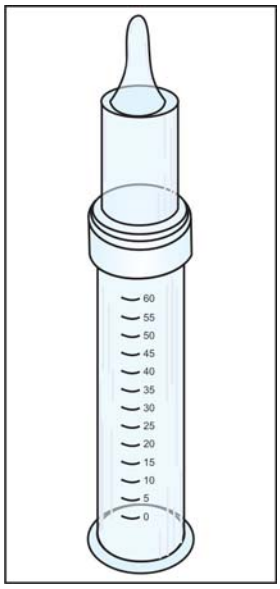


Fig. 22.8: Haberman feeder

- The flow level can be set at zero, moderate or maximum to Control the milk flow.
- Only disadvantage is being very costly.

Special cleft palate teat:

- The rubber flange tends to rub against the edges of the cleft covers it preventing aspiration and making it possible for vacuum to be created in mouth.
- Its disadvantages:
  - Improper usage can lead to fibrosis.
  - Increases risk of infection.
  - Difficult to clean the teat.

Other commonly used teats/ bottle are:

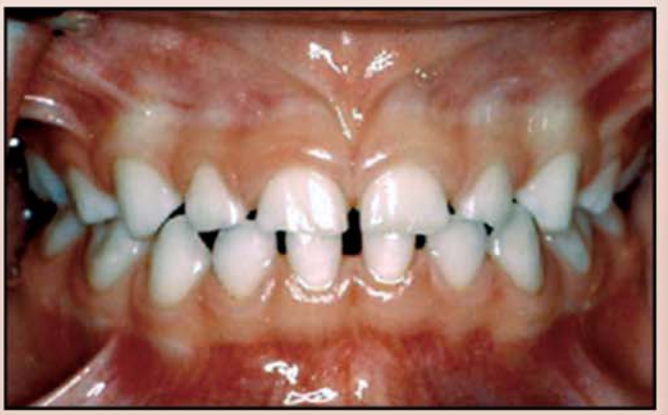
- Queensland and cleft PALS bottle.
- Pigeon cleft teat.
- Chu-chu easy feed teat (nitrosamine free)
- Chu-chu cross cut feed teat
- Nuk nipple.

Dental Management

- A thorough examination, investigation and diagnosis of dental anomalies and disease is necessary.
- Advice safe diet to reduce dietary risk for caries and diet planning should be.

- Advocate needed prophylactic measures to maintain oral hygiene through relevant brushing techniques.
- Obtain proper impressions to fabricate an obturator to enable easy feeding of the child/infant.
- Educate both parent/patient to maintain clean gum pads in infants after every feed and brushing in older children.
- Advocate appropriate preventive measures like:
  - Fluoride application
  - Sealant therapy
- Early intervention of carious teeth should be ensured.
- Ensure frequent referral to prosthodontist and an orthodontist for managing the needs of the patient at regular intervals to meet the demands of patient's craniofacial growth.
- Reinforce the patient/parent to cope up and ward off psychological stress and social stigma and prevent depression.
- Monitor changes in speech/heavily abilities of the child and needed reinforcements.

23



Gingiva and Periodontium in Health and Disease in Children

INTRODUCTION

The Gingiva is that part of the oral mucosa, which covers the alveolar processes of the jaws and surrounds the cervical portions of the teeth. Periodontal ligament is the connective tissue that surrounds the root and connects it to the bone and continuous with connective tissue of gingiva and communicates with the marrow spaces through vascular channels in the bone.

Gingival and periodontal diseases comprise the most widespread infections of human kind and many of these first manifest themselves in childhood. As early as 1938, MacCall stated that there was a lack of appreciation of the fact that the foundation of virtually all periodontal diseases are laid in childhood, citing Wordsworth **"The child is the father of the man".**

Periodontal diseases may have their inception during childhood and reach destructive stages while those affected are still in their teens. Tremendous strides have been made in fields like molecular biology, genetics, immunology and microbiology, which have influenced our understanding of all aspects of periodontology and has also, emphasized the relevance of gingival and periodontal diseases in children and adolescents.

NORMAL GINGIVA AND PERIODONTIUM

The components of the gingival and periodontal structures differ from children, adolescents and those seen in adulthood owing to the significant changes that take place during growth and development.

As cited by Baer and Benjamin (1974) **"The periodontium during childhood and puberty is in constant state of change owing to the exfoliation and eruption of teeth. This makes a general description of the normal periodontium difficult because it varies with the age of the patient."**

Division of Gingiva

It is divided into three different zones as follows (Fig. 23.1).

Papillary Gingiva

It is also termed as either interdental gingival or interdental papilla. Its shape is determined by the contact relationship between the teeth, the width of proximal tooth surfaces and course of CEJ. When normal spaces are evident between the teeth, the papilla has a **"saddle"** shape and remains healthy because of increased keratinization this feature may appear both in adults and children, both in the primary dentition in which primate spaces are common and during the eruption of permanent teeth.

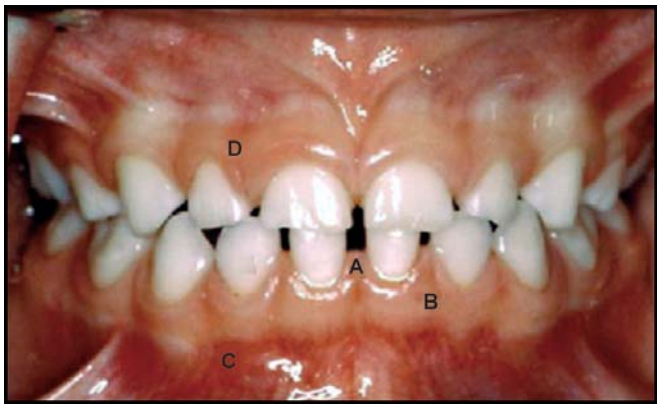


Fig. 23.1: Zones of gingival tissues. A. Papillary gingiva. B. Marginal gingiva. C. Alveolar mucosa. D. Interdental clefts

In both cases when the spaces close during normal development the interdental papilla with col formation will completely occupy the interdental space.

Marginal Gingiva

The free or marginal gingiva has a dull surface and firm in consistency. It comprises of gingival tissue at the vestibular and lingual/palatal aspects of the teeth. It is flaccid and can be easily retracted by a blunt instrument or air syringe. In adults, gingival tends to slope coronally to a round or a thin edge in primary dentition, owing to the normal cervical construction and presence of immature collagen fibers, increased vascularity, the marginal gingiva appears to be thick, rounded and rolled.

Attached Gingiva

The coronal demarcation of the attached gingiva is the free gingival groove and apical demarcation is the mucogingival junction where it becomes continuous with the alveolar mucosa. Width of attached gingiva is significantly modified by the eruption and exfoliation of

teeth. Width of attached gingiva is greater in adults than in children. This widening is not linear since the attached gingiva in a newly erupted permanent tooth is significantly narrower than that of its deciduous predecessor (Table 23.1). Presence of interdental cleft and the retrocuspid papilla are the two unique anatomic features found in children. Interdental clefts are seen lying apical to interdental areas while the retrocuspid papilla is present approximately 1 mm below the free gingival groove on the attached gingiva lingual to mandibular canine, which apparently decreases in prevalence with age.

Table 23.1: Width of attached gingiva in relation to central incisors in children (mm)

		Maxilla		*Mandible*	
Age (years)	*Type of tooth*	*Mean*	*SD*	*Mean*	*SD*
3-5	Deciduous	3.31	0.70	2.16	0.48
5	Permanent	0.76	0.82	0.45	0.70
13-15	Permanent	2.59	0.87	1.54	0.63

Gingival characteristics are tabulated as follows in Table 23.2.

Alveolar Mucosa

It is red in color, easily movable and different from the remaining tissues because of its thin epithelium and absence of keratin. It increases in width with age and eruption of the teeth.

Alveolar Bone

Alveolar bone has fewer trabecular, larger marrow spaces, is less calcified, and has a thinner lamina dura and wider periodontal membranes when compared with adults.

GINGIVAL DISEASES IN CHILDREN

A spectrum of factors are held responsible for gingival disease in children like Oral microbes, Systemic conditions, Hormonal changes, Drugs, Neoplasm, Nutritional factors and even normal exfoliation and eruption of teeth are included.

Classification of Gingival Diseases in Children

Gingival Diseases

Plaque induced gingivitis

1. Gingivitis associated with dental plaque
   - A. Without local contributing factors only
   - B. With local contributing factors
2. Gingival diseases modified by systemic factors
   - A. Associated with endocrine system
     1. Puberty associated gingivitis
     2. Menstrual cycle associated gingivitis
     3. Diabetes mellitus gingivitis
   - B. Associated with blood dyscrasias
     1. Leukemia associated gingivitis
     2. Other
     3. Gingival diseases modified by medications
        - A. Drug influenced gingival diseases
          1. Drug influenced gingival enlargement
          2. Drug influenced gingivitis
     4. Gingival diseases modified by malnutrition
        - A. Ascorbic acid deficiency gingivitis
        - B. Other

Non-plaque induced gingival lesions

1. Gingival diseases of specific bacterial origin
   - A. *Neisseria gonorrhoeae*

Table 23.2: Characteristic features of gingiva

Characteristic	*Features in children*	*Governing factor*
Color	pink or red	-Normal change with age -Degree of vascularity epithelial keratinization -Pigmentation -Thickness of epithelium
Stippling	-absent in infants and -Starts to be evident at age 2-3 yrs sometimes	-Relates to round protuberances and depressions on gingival surface due to papillary layers of gingiva

   B. *Treponema pallidum*
   C. Streptococcal species
   D. Fuso-spirochetal species
2. Gingival diseases of viral origin
   A. Herpes virus infection
      1. Primary herpetic gingivostomatitis
      2. Recurrent oral herpes
      3. Varicella zoster
   B. Other
3. Gingival diseases of fungal origin
   A. Candida species
   B. Linear gingival erythema
   C. Histoplasmosis
4. Gingival diseases of genetic/hereditary origin
   A. Hereditary gingival fibromatosis
   B. Other
5. Gingival manifestations of systemic condition
   A. Mucocutaneous lesions
   B. Allergic reactions
      1. Dental restorative materials
      2. Reactions attributed to
         i. Dentifrices
         ii. Mouth rinses
         iii. Food and additives
6. Traumatic lesions
7. Foreign body reactions

Abscess

- Gingival abscess
- Pericoronitis
- Gingival disease.

Plaque Induced Gingival Diseases

In children, the prevalence of plaque induced gingivitis increases with age until it reaches a zenith at puberty. Plaque induced gingivitis begins at the gingival margin and can spread throughout the remaining gingival unit.

Clinical signs of gingival inflammation involving changes to gingival contour, color and consistency are associated with a stable periodontium which exhibits no loss of periodontal attachment or alveolar bone.

In children, gingivitis is not as intense as that found in young adults with similar amounts of plaque. This age related difference in the development and severity of gingivitis may be associated with the quantity or quality of dental plaque, the response of the immune system, or morphological differences in the periodontium between children and adults such as:

- Thicker junctional epithelium
- Increased vascularity in gingival connective tissue
- Increased keratinization

More specifically, the dental plaque of children usually contains lower concentrations of putative pathogens.

Common clinical findings include

- Erythema
- Edema, loss of stippling
- Bleeding
- Sensitivity
- Tenderness
- Enlargement
- No loss of attachment, etc

Eruption Gingivitis

Gingival changes that occur during the eruption of tooth manifest as eruption gingivitis, which is a transient state of discomfort for the patient.

The gingival changes during eruption of teeth are categorized systematically as:

Pre-eruptive changes: Prior to eruption of teeth, gingiva appears as a firm blanched bulge in the area pertaining to the teeth and appears to conform to the contour of the underlying crown of the tooth.

Eruptive changes: Formation of gingival margin is the hallmark of this stage where gingival sulcus develops during crown eruption penetrating oral mucosa. Gingiva appears erythematous, edematous, rounded and sensitive. In this stage, accompanying sensitivity and tooth position leads to poor oral hygiene; as plaque accumulates, the inflammation continues to worsen; sometime leading to bad breath, etc. The symptoms usually subside with the attainment of normal occlusal position of the tooth.

Pre-pubertal Gingivitis

Since 19th century, evidence has accumulated to support the concept that tissues of the periodontium are

modulated by endogenous sex steroid hormones, viz. androgens, estrogens and progestin.

The principal explanations for hormone induced gingival changes are due to:

- Changes in microbiota in dental plaque
- Immune function
- Vascularity
- Cellular function in the gingiva

Puberty is not a single episode but a complex process of endocrinological events that produce changes in the physical appearance and behavior of adolescents.

Clinical features include of pre-pubertal gingivitis include:

- Enlarged interdental areas
- Spontaneous and easily stimulated gingival bleeding
- Edematous gingiva
- At times associated with sensitive gums

Treatment:

- By age of 18, the hormonal effect usually subsides
- Management includes scaling and home care instructions
- If required appropriate nutritional supplements to aid general health status.

Menstrual Cycle Associated Gingivitis

Following menarche, there is a periodicity of sex steroid hormone secretion over a 25-30 day period–the menstrual cycle–during which clinically significant inflammatory changes in the gingiva have been observed.

Most common gingival changes involve

- Minor signs of inflammation during ovulation
- Increase in GCF flow, etc.

Most young adolescents during the menstrual cycle will present with a very mild form of the disease.

Leukemia Associated Gingivitis

This neoplastic disease results in abnormal and uncontrolled proliferation of immature leukocytes. This condition is characterized by:

- Leukemic gingival enlargement (due to gingival infiltration by leukemic cells)
- Increased gingiva pockets leading to increased plaque accumulation
- Bluish red discoloration of gingiva
- Gingival enlargement that occupies the interdental papilla and partially covers the crown of the teeth
- Edematous gingiva
- Spontaneous bleeding due to relative thrombocytopenia
- Increased incidence of gingival bacterial infection and prone to acute gingival necrosis and pseudomembrane formation
- Presence of constant gnawing pain.

These changes are attributed either due to underlying pathology or drug influenced changes too.

Management:

- Establish early diagnosis
- Use appropriate drugs and modify when required
- Observe meticulous oral hygiene measures both at home care and professionally.

Gingival Diseases Modified by Medication

Drug induced Gingival Enlargement

The disfiguring overgrowth of gingiva is a significant outcome principally associated with anticonvulsant drugs such as Phenytoin, Immuno-suppressors such as Cyclosporine and Calcium channel blockers such as Nifedipine, Verapamil, Diltiazem and Sodium valproate.

Common clinical characteristics include:

- Lesions appear within first 3 months of drug therapy
- Initially appears as painless enlargement of Interdental papilla; marginal gingiva which later becomes lobulated and progresses to cover the clinical crowns
- Formation of pseudo pockets
- A tendency to occur more often in anterior gingiva
- Higher prevalence in younger age groups
- Condition regresses or disappears after cessation of drug therapy.

Etiology: Drugs such as phenytoin stimulate the growth of genetically distinct populations of gingival fibroblasts resulting in the accumulation of connective tissues, this accumulation of connective tissue is further aided by reduced catabolism of collagen molecules (Hassell and Heftic 1991).

Management:

- Replacement with alternate drug if possible

- Meticulous oral hygiene care
- Gingivectomy in some cases as per need suggests

Gingival Disease Associated with Malnutrition

Although some nutritional deficiencies can significantly exacerbates the response of gingiva to plaque bacteria, the precise role of nutrition in the initiation or progression of gingival diseases remains to be elucidated.

Scorbutic Gingivitis

It results due to vitamin C/ascorbic acid deficiency. Vitamin C deficiency results in:

- Defective formation and maintenance of collagen
- Retardation or cessation of osteoid formation
- Increased capillary permeability
- Hyporeactivity of contractile elements of peripheral blood vessels
- Sluggishness of blood flow.

Clinical features:

- Gingival enlargements
- Hemorrhagic, bluish red gingiva
- Bleeding on slightest provocation
- Gingival inflammation, etc.

Management:

- Vitamin C supplementation
- Appropriate oral hygiene reinforcements and maintenance

Gingival Abscess

A gingival abscess is an acute, painful rapidly expanding lesion localized to the gingiva. Most gingival abscesses are detected on the marginal gingiva or the papilla. Gingival abscesses usually arise from an insult such as trauma caused by food which forces bacteria into the tissue.

It is usually characterized by a bright red gingival swelling which converts into a lesion that appears as a pointed and fluctuant mass from which purulent exudates may be expressed. The lesion is generally self-limiting, ultimately rupturing if permitted to progress. It may be accompanied by pyrexia. It is best treated by debridement.

Pericoronitis

It is the inflammation of gingiva covering the partially erupted tooth, most commonly occurring in erupting third molars, but also seen during eruption of first and second molars in children. Pericoronitis is caused due to food entrapment and its bacterial putrification below the coronal flap which becomes

- Inflamed
- Swollen
- Tender

If interferes with occlusion it gets traumatized and very painful.

Management:

- Debridement and antibiotic therapy
- Some cases require partial or complete Operculectomy.

Acute Herpetic Gingivostomatitis

Causative pathogen: Herpes simplex serotype - Infection imparts lifetime immunity.

Clinical manifestation:

Prodromal symptoms

- Malaise
- Fever
- Headache
- Irritability
- Dysphagia
- Lymphadenopathy
- Arthralgia
- Anorexia

Oral manifestations:

- Initially vesicles seen on mucosa of lips, tongue and gingiva.
- Gingival inflammation characterized by diffuse erythematous, shiny appearance.
- Vesicles rupture to form small, ragged and painful ulcers that are covered by grey membrane and erythematous halo present.
- An ulcer persists for 7-10 days and heals spontaneously, leaving no scars.

Management:

- Isolation and bed rest

- Antipyretics and analgesics to control fever and relieve pain
- Topical application of anesthetic agents like lidnocaine
- Copious oral fluid intake
- Steroids are contraindicated
- Use of antibiotics in case of superimposed secondary infection

Acute Necrotizing Ulcerative Gingivitis

Causative Pathogens: Spirochetes and fusiform bacteria (Borrelia vincentii, Bacillus fusiformis, Prevotella intermedia).

Synonyms

- Ulcerative gingivitis
- Vincent's infection
- Trench mouth
- Fusospirochetal disease.

Incidence: Peak incidence in late teens and 20's occasionally occurs in 6-12 years olds.
Etiology: Stress related.

Clinical characteristics

- Sudden onset and episodic progress
- Initially papillary gingiva appears swollen and papillary necrosis leads to a "**punched out**" appearance of papilla tip
- Gingival soreness
- Spontaneous bleeding
- Presence of metallic taste
- Excessive salivation
- Halitosis
- Ulcers spread laterally to marginal gingiva
- Ulcers are painful to touch and are covered by yellowish gray pseudomembrane formation; which when removed reveals underlying raw bleeding surface.

Management:

- Removal of the pseudomembrane in the first visit followed by other treatments in next subsequent visits.
- Local debridement
- Subgingival curettage
- Topical application of anesthetic agents to relieve patient discomfort
- Use of equal mixture of warm water and 3% Hydrogen peroxide as a mouthwash to rinse once in every 2 hours
- Twice daily rinses of 0.12% chlorhexidine
- Appropriate antibiotic therapy using penicillin/erythromycin/metronidazole
- Meticulous oral hygiene measures
- Bed rest and isolation
- Nutritional supplementation and copious intake of fluids
- Frequent periods of clinical evaluation and maintenance required.

Hereditary Gingival Fibromatosis

Synonyms

- Gingivomatosis elephantiasis
- Juvenile hyaline fibromatosis
- Hereditary gingival hyperplasia.

Clinical Features

- It is an autosomal dominant condition.
- It is a slowly progressive gingival enlargement which develops upon eruption of permanent dentition; however it can also be seen in primary dentition.
- It can be localized or generalized and may cover the occlusal surfaces of teeth.
- Enlarged gingiva is non-hemorrhagic and firm, but there can be an overlay of gingival inflammation which can augment enlargement.
- It is due to accumulation of specific populations of gingival fibroblasts resulting in an abnormal accumulation of connective tissue.
- Affects all three parts of the gingiva which differentiates it from drug induced gingival enlargement.

Management

- Gingivoplasty and Gingivectomy as case suggests.
- Meticulous oral hygiene measures to reduce plaque accumulation.
- Frequent dental visits to assess the condition and maintenance.

PERIODONTAL DISEASE AS IN CHILDREN AND ADOLESCENTS

I. Periodontal abscess
II. Adult onset periodontitis

III. Early onset periodontitis
- Localized pre-pubertal periodontitis (LPP)
- Generalized pre-pubertal periodontitis (GPP)
- Localized juvenile periodontitis (LJP)
- Generalized juvenile periodontitis (GJP)

IV. Systemic diseases with associated periodontal disease
- Hypophosphatasia
- Leukocyte adhesion deficiency
- Papillon-Lefevre syndrome
- Down syndrome
- Chediak-Higashi syndrome
- Neutropenia
- Langerhans cell histiocytosis
- Acute leukemia
- IDDM, etc

Note: On the latest classification

Periodontal Abscess

A periodontal abscess is an acute suppurative inflammation of the deeper periodontal tissues caused by an infection of Pyogenic bacteria. This results in a rapid destruction of the attachment apparatus and alveolar bone. Glickman (1979) suggested five mechanisms of abscess formation:

1. Extension of a pocket into the supporting periodontal tissues along the lateral aspect of the tooth root.
2. Lateral extension of the inner pocket surface into the connective tissue of the pocket wall.
3. In the sinus pocket, an abscess may develop into deeper portions due to the formation of a cul-de-sac.
4. Incomplete removal of calculus results in tissue shrinkage with subsequent occlusion of the pocket opening.
5. Following traumatic injury of a tooth or a perforation of the lateral wall of the root during endodontic treatment.

Periodontal abscesses are
- Painful to palpation
- Sensitivity of the associated tooth to occlusion, mastication or gentle percussion
- Overlying gingiva may be distended and edematous and affected tissue will appear shiny with red to reddish blue hue.
- At times affected tooth may extrude from its socket
- Mobility may be present
- Presence of regional lymphadenopathy and slight elevation in body temperature
- Destruction of periodontal attachment
- Deep pockets present
- Purulent exudate is expressed from gingival margin with gentle pressure.

Management
- Drainage and debridement of lesion
- Root planing and curettage can be advocated
- Patient to be on antibiotic therapy.

Early Onset Periodontitis

Prepubertal Localized Periodontitis

Onset: Adolescent or pre-pubertal age

Teeth involved: Restricted/Localized to some primary and permanent teeth

Predisposing factors:
- Interproximal caries
- Faulty restoration
- Crowding
- Plaque retentive areas, etc.

Microbial association: Non-specific bacterial flora.

Clinical features:
- Gingival inflammation
- Incidental attachment loss
- Deeper pockets
- Increased plaque accumulation
- Presence of bone loss

Management:
- Gingival curettage
- Antibiotic therapy if needed
- Oral hygiene care
- Correction and removal of plaque retentive areas.

Generalized Prepubertal Periodontitis

It is characterized by generalized acute gingival inflammation with rapid attachment loss and bone destruction

leading to mobility and migration of teeth. It is best managed by meticulous oral hygiene and elimination of predisposing factors.

Aggressive Periodontitis

Synonyms:

- Juvenile periodontitis
- Rapidly progressive periodontitis

Historical evidence and background:

In 1923 Gottlieb –	"Diffuse atrophy of alveolar bone"
1928 Gottlieb –	"Deep cementopathia"
1938 Wannenmacher-	"Parodontitis Marginalis progressiva"
	"Peridontosis"
1967 Chaput and-colleagues	"Juvenile periodontitis"
1969 Butler	"Early onset periodontitis"
	"Aggressive periodontitis".

Definition: "A disease of the periodontium occurring in an otherwise healthy adolescent which is characterized by rapid loss of alveolar bone about more than one tooth of the permanent dentition".

Causative pathogen: Actinobacillus actinomycetem-comitans.

Clinical characteristics:

1. *Localized aggressive periodontitis*
- Usually has an age of onset around puberty
- Localized first/molar/incisor presentation with interproximal attachment loss of at least two permanent teeth, one of which is a first molar, and involving no more than two teeth other than first molar and incisor
- Presence of little or no plaque or local irritants
- Rapid destruction of periodontal ligament
- Rapid development of midline diastema and mobility of first molar
- Characteristic "arc shaped loss of alveolar bone extending from the distal surface of the second premolar to the mesial surface of the second molar.

2. *Generalized aggressive periodontitis:*
- Usually affects individuals under the age of 30
- Generalized interproximal attachment loss affecting atleast three permanent teeth other than first molars and incisors.
- Episodic periodontal destruction with periods of advanced destruction followed by a period of quiescence of variable length.
- Similar to LAP; inconsistency of local irritants and risk factors to the amount of destruction.
- Increasing depths of pockets
- Increased mobility of affected teeth
- Radiographic findings reveal severe bone loss and periodontal destruction.

Management:

- Combination of scaling and root planning
- Effective antibiotic therapy
- Drugs combinations like
  — Metronidazole + Amoxicillin
  — Tetracycline
  — Doxycycline
- Use of mouth rinses
- Meticulous oral hygiene
- Advocate Bone regenerating procedures as case demands.

Systemic Diseases with Associated Periodontal Problems

Hypophosphatasia (Rathbun Syndrome)

Characteristic features

- Premature loss of deciduous teeth to severe abnormalities leading to neonatal death
- Pulp chambers may be abnormally large
- Presence of abnormal cementum
- Permanent teeth are often not affected.

Management:

- Extraction of mobile primary teeth
- Advocate conventional periodontal therapy for permanent dentition.

Leukocyte Adhesion Defect

- Autosomal recessive defect

- Poor leukocyte adherence
- Severe gingivitis and periodontitis leads to tooth loss due to rapid periodontal destruction
- Early exfoliation of primary dentition
- Bone marrow transplant is the effective mode of management.

Papillon-Lefevre Syndrome

Papillon-Lefevre syndrome is a rare autosomal recessive trait with an incidence of between one and four persons per million. Parental consanguinity is demonstrated in about one-third of cases.

Papillon-Lefevre syndrome is characterized by:

- Palmar-plantar hyperkeratosis
- Rapid destruction of alveolar bone and periodontium of both primary and permanent dentitions, commencing at the time of tooth eruption
- Calcification of falx cerebri and choroid plexus
- Retardation of somatic development
- Otitis media is commonly seen

Periodontal effects appear almost immediately after tooth eruption when gingiva become erythematous and edematous. Plaque accumulates in the deep crevices and halitosis can ensue.

The primary incisors are usually affected first and can display marked mobility by the age of 3 years. By the age of 4 or 5 years, all the primary teeth may have exfoliated. Following such tooth loss, the gingival appearance resolves and may well return to health only for the process to be repeated as the permanent dentition starts to erupt. Almost majority of the teeth are lost by the age of 14-15 years. There is dramatic alveolar bone destruction, often leaving atrophied jaws.

Management:

- Psychological re-enforcement of affected individual
- Meticulous oral hygiene
- Prosthetic rehabilitation for edentulism

Down's Syndrome (Trisomy 21)

The genetic disorder first described in 1866 by John Langdon Down is caused by an autosomal inherited trisomy of chromosome 21.

It is characterized by:

- Atypical orofacial appearance
- Flattened facial profile with epicanthic folds
- Expanded bridge of the nose
- Opened mouth
- Protruding fissured tongue
- Retarded growth
- Mental deficiency
- Muscle hypotonia
- Joint hyperflexibility
- Congenital heart disease.

It has been recognized that affected individuals frequently manifest an aggressive form of periodontal disease affecting both the primary and permanent dentition which may lead to early exfoliation of teeth. Periodontal destruction is often generalized although lesions are more severe around lower anterior teeth; it is characterized by formation of deep periodontal pockets associated with heavy plaque accumulation and intense gingival inflammation.

Although bacterial plaque, calculus and other potential local irritants are present and oral hygiene is frequently poor owing to the physical and mental limitations of these patients, the severity of periodontal destruction far exceeds that explainable by these local factors alone.

Management:

- Reinforce the care-giver with oral hygiene measures
- Regular periodic professional care.

Chediak-Higashi Syndrome

It is a rare-autosomal disease caused by a lysosomal defect leading to anomalies of blood cells and neutrophil dysfunction.

This disease presents with variable oculocutaneous albinism, strabismus, photophobia, nystagmus and recurrent cutaneous and respiratory infections.

The onset of the disease for the majority of patients occurs before age of 10 years, with a mean of 5.9 years (Blume and Wolff, 1972). Neutropenia, gingivitis and severe periodontal disease have also been described as characteristic of this disease. Early onset with severe periodontal destruction leading to rapid loss of

periodontal attachment, severe mobility and premature exfoliation of teeth are some of the frequent findings.

Langerhans Cell Histiocytosis

Langerhans cell histiocytosis is of importance to the dental profession because of the fact that oral soft tissue and bony lesions are common and may be the earliest manifestation of the disease. These include:

- Loosened teeth
- **"Periodontosis like symptoms"**
- Premature eruption of primary teeth with gingival bleeding
- Gingival inflammation, ulcers or hypertrophy
- Halitosis
- Severe bone loss causing early exfoliation of primary teeth
- Radiographically affected molars are classically described as **"floating teeth".**

Management:

- Optimizing oral hygiene
- Removal of teeth with excessive bony involvement
- Appropriate chemotherapy.

Insulin-Dependent Diabetes Mellitus (Type 1)

There is increased incidence of gingivitis, increased risk and earlier onset of periodontitis and alveolar bone resorption.

Incidence increases after puberty with age. Neutrophil decreased function is seen.

- Xerostomia or reduced salivary flow
- Recurrent gingival abscesses
- Increased frequency of caries
- Altered microbial flora
- Increased susceptibility to infections
- Reduced wound healing.

Management:

- Appropriate diet planning
- Plan for morning visits/appointments
- Antibiotic prophylaxis before any dental procedure, etc.

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Dental Management of Children with Special Needs

INTRODUCTION

Impairment becomes a disability for a child only if he or she is unable to carry out the normal activities of his or her peer group. For some children impairment is a permanent feature in their lives although it may become a disability only if they are unable to take part in every-day activities.

The term "**Dentally disabled**" refers to patients who have some gross condition or deficit in their oral cavities, which necessitates special dental treatment for example, a cleft lip /palate. By contrast, children who are "**disabled for dentistry**" are those who have a physical and/ or intellectual or emotional condition that may prevent them from being treated in a routine manner. They should not be called as disabled children who need special attention but as "**children with special needs**".

The need for special dental care arises because of:
Difference in dental disease prevalence
Dental disease/treatment may be life threatening
The modifications required to treatment plans
The need for special facilities
Treatment may be time consuming.

CLASSIFICATION OF CHILDREN WITH SPECIAL NEEDS

A. Intellectually impaired:
Cerebral palsy, birth anoxia, severe infections, autism, microcephaly, metabolic disorders, major trauma, some craniofacial syndromes like Down syndrome, Fragile X syndrome, dyslexia, etc.
B. Physically impaired:
Developmental and degenerative
C. Sensory impairment
D. Medically compromised
Common medical disabilities include:
1. Congenital heart defects
Heart murmurs, ventricular and atrial septal defects, pulmonary stenosis, patent ductus arteriosus, Tetralogy of Fallot.
2. Acquired cardiovascular disorders
Rheumatic fever, disease of pericardium and myocardium and other cardiovascular problems
3. Disorders of blood
Bleeding disorders: Hemophilia, Von-Willebrand's disease, Thrombocytopenia
Blood dyscrasias: Anemias, enzymatic deficiencies, sickle cell anemia, Thalassemia and leukemia.
Respiratory disorders: Asthma and cystic fibrosis.
Convulsive disorders: Febrile convulsions and epilepsy.
Metabolic and endocrine disorders: Diabetes, Adrenal insufficiency, Thyroid and renal disorders.
4. Neoplasm.
5. *Organ transplantation:* Pretransplant, immediate post-transplant and stable
Post-transplant stages
E. *Immunogenic disability:* Human immuno-deficiency syndrome, etc.
F. Combination of impairments.

INTELLECTUALLY IMPAIRED

Cerebral Palsy

Cerebral palsy is the most common handicapping disorder. It is a disorder of movement and posture caused by brain damage which occurs in the early stages of development. Cerebral palsy cannot be cured, but it does not get worse with time. Approximately half of all children with cerebral palsy at their first birthday "outgrow" signs of the disorder by their seventh birthday.

Overview

- Cerebral palsy is not a single disease, but a group of disorders that occur when a baby's brain is damaged.
- The prevalence of severe C.P. is around 2 for every 1,000 live births.

Types of Cerebral Palsy

Cerebral palsy may be classified according to the resulting problems with posture and control of movement. The classifications are

- **Spastic**: above-normal tone or stiffness of the muscles of the body.

- **Hypotonic**: weak, "floppy," lacking normal muscle tone.
- **Athethotic**: slow, writhing, involuntary movements of hands and feet. Athetosis is caused by problems in the extrapyramidal system of the brain.

Cerebral palsy may also be classified according to the motor disability associated with it, such as: Hemiplegia, Diplegia, Dyskinesia, and Quadriplegia.

- **Spastic hemiplegia**: one-sided neurologic defect. The arm is more affected than the leg.
- **Spastic diplegia**: scissoring walking pattern, with toe-walking.
- **Dyskinesia**: difficulty walking, with some spasticity.
- **Spastic quadriplegia**: all limbs are affected, with multiple medical complications.

Etiology: The cause of cerebral palsy is poorly understood, but it is most likely caused by a variety of factors.

- Cerebral palsy can be associated with prenatal, perinatal, or postnatal events.
- Prenatal factors cause 70-80% of cases of cerebral palsy. The developing brain is subject to injury at any time, due of its complexity and vulnerability.
- The most common finding in children with cerebral palsy is prenatal injury to the portion of the brain lying next to the middle cerebral artery.
- The clinical finding of prenatal brain injury - leukomalacia - predicts cerebral palsy better than the ultrasonic finding of intracranial hemorrhage. Periventricular leukomalacia is the medical term used to describe death of the white matter of the brain in the area of the lateral ventricles.
- Recent studies have shown that difficulties during birth and delivery are not a common cause of cerebral palsy.

Prenatal Risk Factors

- Prematurely born infants have a higher incidence of cerebral palsy than babies born at term.
- The rate of cerebral palsy is at least 25 times higher among infants who weigh less than 1,500 grams at birth, compared to full-sized newborns.
- Any infection of the pregnant mother, such as rubella (German measles) or dental infection causes a risk to the unborn child.
- Maternal drug or alcohol abuse.
- Maternal thyroid disorder.

Special Oral and Dental Health Concerns for Children with Cerebral Palsy

- Orofacial dysfunction is a severe health problem, as well as a problem for acceptance by peers and society.
- Cerebral palsied children have drooling, eating, drinking, and speaking disorders.
- More than 90% of children with cerebral palsy have oral motor dysfunction. The severity of oral dysfunction makes it difficult for some cerebral palsied children to be adequately nourished.
- Drooling is not due to excessive production of saliva, but to a poor and disorganized swallowing pattern.
- There is abnormal neuromuscular coordination of the tongue, lips, and cheeks - which leads to poor dental alignment and periodontal problems.
- Trauma of the face and mouth occur much more frequently in children who have cerebral palsy.
- Children with cerebral palsy may demonstrate self injurious behavior, including: tongue, cheek, and lip biting; finger, arm, and hand chewing.
- Protective oral appliances may be useful in combating self-injurious behavior.
- Children who are affected by cognitive disability or mental retardation often practice damaging oral habits, including Bruxism, Rumination, Pouching, and Pica.
- **Bruxism:** This is clenching, grinding, and gnashing of teeth. It is a frequent finding in children with cerebral palsy. The treatment for bruxism may include the use of a soft or hard mouth guard if the child can tolerate it.
- **Rumination:** This is the re-chewing, regurgitation, and re-swallowing of previously ingested food. This habit causes the acidic contents of the stomach to travel up into the mouth, and bathe the teeth in acid. Rumination can lead to demineralization, and loss of tooth structure.
- **Pouching:** This is the placement of food or medicine between the cheek and teeth for a long period of time. This habit can cause dental decay.

- **Pica:** This is the compulsive eating of non-edible substances, including: sand, dirt, and paint chips. Pica can lead to destruction of tooth structure and damage of oral soft tissue.

Oral Findings in Children with Cerebral Palsy

- Children with cerebral palsy frequently have gastroesophageal reflux, as well as episodes of vomiting. Either problem can lead to dental erosion, or loss of tooth structure.
- Gingival overgrowth, due to seizure medications, is a frequent problem in children with cerebral palsy.
- **Orofacial findings in spastic cerebral palsy:**
  The head is tensely reclined.
  The mouth is open, and facial movements are tense.
  The tongue is hypertonic and cigar-shaped.
  There is tongue thrust during swallowing and speaking.
  Since the upper lip is underdeveloped, it does not produce enough pressure on the front teeth to align them correctly.
- **Orofacial findings in athethotic cerebral palsy:**
  The tongue shows spontaneous wave-like movements.
  There may be an abrupt and wide opening of the mouth, which can lead to jaw dislocation.
  There is an uncoordinated movement of tongue, jaw, and face muscles.
- **Orofacial findings in hypotonic cerebral palsy:**
  The tongue is large, flat, and protruded.
  Facial movements are weak, and the upper lip is inactive.

Role of the Dentist

- The dentist should try to schedule appointments for children with cerebral palsy early in the day.
- Obtain the child's medical history before the first appointment so that any necessary medical consultations can be arranged.
- Try to develop a good rapport with the child.
- Gain the cooperation of the cerebral palsied child by using behavior management techniques such as: tell-show-do, positive reinforcement, and voice control.
- A child with severe cognitive disability may require repetition of commands and requests, which will enhance comprehension.
- A child with severe visual impairment needs a verbal description of the planned dental procedures. This will help prevent fear and anxiety.
- Communication can also be accomplished using nonverbal techniques, especially for children with hearing impairment
- The dentist may need to use sedation techniques to calm a child – if the child's medical situation permits. Some children can only be treated under general anesthesia, however.
- Children with cerebral palsy may have a severe gag reflex – making it difficult to take dental radiographs.
- Two modified radiographic techniques for use in children with cerebral palsy are:
  The 45 degree oblique head plate and the reverse bite wing (buccal technique)
- **Oblique plate radiographic technique:** a film cassette is held against the patient's cheek. The patient's had is rotated and tilted. The X-ray cylinder is placed just inferior and posterior to the angle of the mandible on the opposite side of the face.
- **Buccal bite wing technique**: the film packet is placed between the teeth and the cheek. The X-ray cylinder is then placed below the lower border of the mandible on the opposite side of the face.
- When dental treatment is performed, stainless steel crowns are often used when the posterior teeth have caries.
- Fixed bridgework is usually not done for patients with cerebral palsy because of the increased risk of falling and dental injury. Patients with frequent seizures should normally not have fixed bridgework done because of the possibility of damage to the supporting teeth or bone during a seizure-related fall.
- The dentist should discuss the option of Myofunctional therapy for young children who have Orofacial and tongue hypotonia. This treatment may increase the muscle tone of the lips, as well as keep the tongue inside of the mouth.
- The dentist should instruct parents on proper home dental hygiene procedures.

- Counsel parents about growth and development of the teeth and Orofacial structures.
- Provide relevant dietary counseling.
- Periodic dental recall appointments are highly recommended in order to supervise and evaluate a patient's oral hygiene. Recall appointments also allow the dentist to monitor any gingival overgrowth which may be caused by anti-seizure medications.

Home Dental Care for Children with Cerebral Palsy

- Choose a well-lit location so that you can look into your child's mouth.
- No matter what position you are using for brushing your child's teeth, remember to always support the head.
- Give lots of praise while brushing your child's teeth.
- Parents should help brush their children's teeth every day, after every meal. Brush the tongue, since this will help prevent halitosis.
- Parents can help make children's teeth more decay-resistant by using an FDI-approved children's toothpaste. Place only a pea-sized drop of toothpaste on the toothbrush.
- Up to the age of three, parents should only use baby tooth cleanser – to avoid fluorosis discoloration of the adult teeth.
- Children taking oral medications should have their teeth cleansed after each dose of medication. Nearly 100% of children's medications contain sucrose, which can increase the risk of developing dental caries.
- Children should have their first oral/dental health evaluation by the age of 12 months, or within 6 months of the eruption of the first tooth.
- Parents should not let their children drink fruit juice or sweetened drinks from a bottle or "tippy" cup, since this prolongs the exposure of teeth to harmful sugar.
- Parents should provide healthy, balanced meals for children. Plenty of healthy snacks should be available for children. They should limit the amount of sugar-laden foods and snacks in the diet. Cheese products actually fight dental caries.

OROFACIAL REGULATION THERAPY

- The orofacial regulation therapy concept includes: Functional diagnostics of oral sensorimotor dysfunctions; a special manual stimulation and facilitation program, which helps to control and improve head and body posture; and the use of removable activating palatal plates, and other orthodontic appliances.
- Treatment using these activating orthodontic appliances should only be done in conjunction with a special physiotherapy program.
- **Description of the myofunctional appliance for spasticity:** It includes a stimulating palatal plate, which helps to reduce tongue thrust. This removable appliance is worn every day, about one hour at a time, for a total of four hours each day. This "palatal button" appliance is not worn during sleep or feeding, however this appliance may be modified, later on, to include upper lip stimulators.
- **Description of the myofunctional appliance for hypotonia**: It acts by stimulating the facial "motor points." The upper lip may be stimulated with "bumpers" which are attached to a "vestibular wire."

AUTISM

Autism was first described in 1943 by the American child psychologist, Leo Kanner. Autism is a type of neuro-developmental disorder, and usually appears within the first three years of a child's life. The hallmark of autism is the lack of communication skills. Affected children also have problems with language, behavior, and social skills.

Autism is a lifelong condition, and its cause is unknown. Environmental and genetic factors do contribute to the development of autism, but most children with autism have normal physical health.

It is a pervasive developmental disorder defined behaviorally as a syndrome consisting of abnormal development of social skills, limitations in the use of interactive language, and sensorimotor defects. Autism specifically affects brain function in the areas responsible for the development of communication and social interaction skills.

Children with autism may appear normal, but the disorder may prevent them from functioning and communicating in socially appropriate ways. The incidence of autistic disorder is seven per 10,000 persons. It is more common in males than females (4:1 ratio).

Etiology

The exact cause of autism is unknown, although it may be linked to brain injury and genetics. There are many biologic causes, but none of them are unique to autism. However, the causes of autism include:

- There is a familial genetic tendency for autism. There is a 3-8 percent risk of recurrence if a family already has one autistic child.
- Fragile X syndrome.
- Tuberous sclerosis.
- Prenatal factors such as intrauterine rubella, and cytomegalic inclusion disease.
- Postnatal factors such as untreated phenylketonuria, infantile spasms, and herpes simplex encephalitis.

Signs and Symptoms

No two children affected by autism display the same behaviors or symptoms.

Early Symptoms of Autism in Patients

- A baby who doesn't babble or gesture by the age of 12 months.
- A baby who lacks eye contact with its mother by the age of 12 months.
- A baby who resists being held or cuddled by its mother.
- A baby who doesn't respond when its mother says its name.
- A baby who appears to be deaf.
- An infant who doesn't say single words by the age of 16 months.

Tips for Recognizing Children with Autism

- Autistic children will often run away from caretakers or health care workers. This activity is called elopement.
- About 50% of autistic children are non-verbal.
- Autistic children may appear stubborn.
- They may exhibit echolalia, or may exhibit rambling speech.
- They may exhibit unusual self-stimulating behavior including hand flapping or rocking back and forth.
- They may appear deaf or not responsive to you.
- They may not be able to answer simple questions.
- They may be sensitive to sound, bright lights, odors, and touch.
- Seizures occur in 25% of autistic children.

Diagnosis

Experienced clinicians can reliably diagnose autism in children younger than three years of age. The typical presenting symptoms of autistic disorder are delayed speech, or challenging behavior before the age of three.

Indications for Formal Developmental Evaluation

- No babbling, pointing, or other gestures by age 12 months.
- No single words by 16 months of age.
- No two-word spontaneous phrases by 24 months of age.
- Loss of previously learned language or social skills at any age.

Diagnosing Autism

- Two levels of evaluation are needed: First, a general screening for developmental problems or risk factors. Second, another evaluation is needed to actually establish the diagnosis of autism.
- Metabolic and/or genetic testing to rule out other conditions with manifestations similar to autism.
- Serologic studies, to see if a child has been infected with herpes simplex, intrauterine rubella, or cytomegalic inclusion disease.
- Hearing tests, to determine if language delay is due to a hearing problem. Two hearing tests are used: the behavioral audiometry test, and the brainstem auditory evoked responses test.
- Neuroimaging, such as MRI, is performed if a structural brain lesion is suspected.

Treatment

Although there is currently no cure for autism, early diagnosis and intervention can significantly enhance the child's social functioning later in life. Early detection and early intensive remedial education and behavioral therapy are the most important measure which needs to be taken. Patience and time are vital to working with these children.

Behavioral Problems in Autism

- Impairment of social skills.
- Echolalia.
- Sensorimotor deficiencies.
- Limited interactive language skills.
- Seizure disorders.
- Mental retardation. Seventy-five percent of autistic persons have some level of mental retardation.
- Stereotypic behavior.
- Self-injurious behavior.
- Problems with symbolic thinking.

Dental care Protocol for Autistic Child

- Offer parents and children the opportunity to tour the dental clinic, so that they may ask questions, touch equipment, and get used to the place. Allow autistic children to bring comfort items, such as a favorite toy.
- Children with autism need sameness and continuity in their environment. A gradual and slow exposure to the dental office and staff is therefore recommended.
- Solicit suggestions from the parent or caregiver on how best to deal with the child.
- Autistic children are easily overwhelmed by sensory overload. This can cause "stimming" (flapping of arms, rocking, screaming, etc). Autistic children are hypersensitive to loud noises, sudden movement, and things that are felt.
- Make the first appointment short and positive.
- Approach the autistic child in a quiet, non-threatening manner. Don't crowd the child.
- Use a "tell-show-do" approach to providing care. Explain the procedure before it occurs. Show the instruments that you will use. Provide frequent praise for acceptable behavior.
- Invite the child to sit alone in the dental chair to become familiar with the treatment setting.
- Autistics will want to know what is going to happen next. Explain what you are doing so it makes sense to them. Explain every treatment before it happens.
- Always tell the autistic child where and why you need to touch them, especially when using dental or medical equipment.
- Talk in direct, short phrases. Talk calmly. Autistics take everything literally – so watch what you say. Avoid words or phrases with double meanings.
- Once the dental patient is seated, begin a cursory examination using only your fingers. Keep the light out of the eyes.
- Next, use a toothbrush, or possibly a dental mirror to gain access to the mouth.
- Praise and reinforce good behavior. Ignore poor behavior.
- Invite the parent of caregiver to hold the child's hand during the dental examination.
- Some autistic children can be calmed by moderate pressure, such as by using a papoose board to wrap the child. One the other hand, "light" touch (such as by air from the dental air syringe) can agitate them. For instance, you are more likely to have problems wrapping a blood pressure cuff around the arm than by inflating it!
- Some children will need sedation or general anesthesia so that dental treatment can be accomplished. Sedation of autistic children who are 8 years and older simply does not work.
- Inappropriate behavior should be ignored.

Down syndrome

Down syndrome is one of the most common genetic syndromes. It occurs in about one of 800 live births. The facial expressions and oral problems of children with Down syndrome are unique and characteristic.

Approximately 95 percent of Down syndrome children have an extra copy of chromosome 21, which makes their chromosome count 47 - instead of the normal 46. Children with Down syndrome (DS) have varying

degrees of intellectual impairment. There is often a severe delay in language development. More than 50 percent of children with DS will live past the age of 50.

Etiology

- In 95 percent of cases, the syndrome is caused by an extra copy of chromosome 21, which failed to separate (nondisjunction) at the time of egg or sperm formation.
- In 4 percent of cases, Down syndrome (DS) is caused by the attachment of an extra copy of chromosome 21 to another chromosome (translocation).
- The remaining 1 percent of DS case is a mosaic of normal and abnormal cells which are produced after conception (mosaicism). The result is that some cells have 46 chromosomes (normal) and some have 47 (abnormal).

Prenatal Diagnosis of Down Syndrome

- Serum multiple-marker testing is recommended for all low-risk pregnant women at 15-18 weeks of pregnancy. This test can detect 80 percent or more of Down syndrome pregnancies.
- The chorionic villus sampling and amniocentesis procedures are tests which provide fetal cells whose chromosomes can be analyzed. These two procedures are reserved for women with a high-risk of having children with genetic abnormalities. Since these two procedures are invasive, they increase the risk of damage to the fetus.

Natal Diagnosis of Down Syndrome

- At birth, the diagnosis of Down syndrome (DS) is based on examination of a child's physical features, as well as the identification (karyotyping) of chromosomal abnormalities in the cells.
- A newborn with DS will often have a flaccid (hypotonic) musculature.
- Upslanting of the eyes (oblique palpaberal fissures) is present in 70 to 98 percent of DS children.
- Congenital heart defects occur in 40 to 50 percent of newborns with DS. The most common cardiac problem is a complete atrioventricular canal defect.

Risk Factors for Down Syndrome

- Women over the age of 35 have a substantially increased risk of conceiving a child with a genetic abnormality.
- If one parent has a chromosome 21 translocation abnormality, or if the first child has Down syndrome caused by a translocation of chromosome 21, then the risk of having a second child with this syndrome is dramatically increased.

Craniofacial Features in Down Syndrome

- Upslanting of the eyes (upward-slanting of the palpebral fissures).
- Closely-set eyes (hypotelorism).
- An extra fold of skin towards the inner aspect of the eyes (epicanthal folds).
- Downward slanting of the corners of the mouth.
- The muscles of the lower lip are hypotonic, causing the lower lip to be everted.
- A relatively enlarged tongue (relative macroglossia). This may be due to the fact that DS children have a small oral cavity and a narrow palate.
- A narrow palate, which is due to the sides of the palate being abnormally thick.
- Bruxism is common.
- More than 90 percent of DS children develop fissures in their tongue by the age of 5. This may be due to developmental causes as well as chronic mouth breathing.
- An open bite. This open mouth posture is due to relative macroglossia and mouth breathing. Mouth breathing is caused by having a small nasal airway.
- Smaller-than-usual oropharyngeal and nasopharyngeal airway size.
- About 13 percent of children with DS have increased mobility of the cervical spine (atlantoaxial instability). This problem precludes participation in contact sports for the affected individuals.

Dental Features in Children with Down Syndrome

- Dental malocclusion and malalignment, including: cross bites, mandibular prognathism, and anterior open bite.

- Lower-than-average incidence of dental caries.
- High incidence of periodontal disease, beginning in the teen years. This is due in great part to the compromised immune system of the DS child.
- Primary and permanent teeth are missing (congenitally) in 50 percent of DS children.
- Hypoplasia and hypocalcification of teeth occurs often.
- Very small (Microdontia) teeth are present in 35 to 55 percent of the primary and permanent teeth of DS children.
- Delayed eruption of teeth in 75 percent of cases. The delay in eruption may last as long as two to three years. The delay is partially due to thyroid disease.

Role of a Dentist

- Try to schedule appointments for children with Down syndrome (DS) early in the day.
- Obtain the child's medical history before the first appointment so that any necessary medical consultations can be arranged.
- Try to develop a good rapport with the child.
- Gain the cooperation of the DS child by using behavior management techniques such as: tell-show-do, positive reinforcement, voice control, mouth prop, and general anesthesia.
- Instruct the parent on proper home dental hygiene procedures.
- Counsel parents about growth and development of the teeth and orofacial structures.
- Provide relevant dietary counseling.
- Offer myofunctional therapy for young children who have orofacial and tongue hypotonia. This treatment may increase the muscle tone of the lips, as well as keep the tongue inside of the mouth. The name of this treatment is: Castillo-Morales' Orofacial Therapy.
- Be aware that most children with DS have a decreased number of T cells in their immune system. This is what causes a high rate of oral infections and periodontal disease.
- Diagnose and treat periodontal disease, especially in DS teens. Consider scaling and root planing the teeth as often as every three months to prevent the progression of periodontal disease. Chlorhexidine gluconate mouth rinse may be beneficial.
- Obstructive sleep apnea (OSA) occurs in 54 to 100 percent of DS children. OSA precludes routine in-office sedation for many children with DS.
- Avoid manipulation of the neck of DS children who have atlanto-axial (cervical spine) instability.
- Provide antibiotic prophylaxis against sub-acute bacterial endocarditis (SBE) for children with heart defects. Note that a complete atrioventricular canal defect is common in infants, and mitral valve prolapse is common in young adults with DS.

Home Dental Care for Children with Down Syndrome

- Choose a well-lit location so that you can look into your child's mouth.
- No matter what position you are using for brushing your child's teeth, remember to always support the head.
- Give lots of praise while brushing your child's teeth.
- Parents should help brush their children's teeth every day, after every meal.
- Brush the tongue. This will help prevent halitosis.
- Parents can help make children's teeth more decay-resistant by using an FDI-approved children's toothpaste. Place only a pea-sized drop of toothpaste on the toothbrush. Until a child is 3 years old, parents should only use baby tooth cleanser - to avoid causing fluorosis discoloration of the adult teeth.
- Children taking oral medications should have their teeth cleansed after each dose of medication. Nearly 100% of children's medications contain sucrose, which can increase the risk of developing dental caries.
- Children should have their first oral/dental health evaluation by the age of 12 months, or within 6 months of the eruption of the first tooth.
- Parents should not let their children drink fruit juice or sweetened drinks from a bottle or "tippy" cup, since this prolongs the exposure of teeth to harmful sugar.
- Parents should provide healthy, balanced meals for children. They should limit the amount of sugar-laden foods and snacks in their diet. Plenty of healthy snacks should be available for children. Cheese products actually fight dental caries.

Moebius Syndrome

In 1892, a German physician named Moebius described a syndrome whose findings included muscle weakness on both sides of the face, an inability to move the eyes from side to side, and hand deformity. This collection of findings was latter called Moebius syndrome.

Etiology

- Insufficient blood supply to the oral and facial structures, which are nourished by the developing primitive subclavian artery, in the fetus.
- Unfavorable intrauterine posture of the fetus, in which pressure from the shoulder of the fetus results in significant pressure on the facial nerve - resulting in facial muscle weakness.
- Intrapartum (during delivery) pressure in which the head of the fetus is pushed against a bony prominence of the mother, such as the pubic rami. This can lead to damage of the child's facial nerve.
- Any interference with normal fetal circulation or circulation in the uterus (any transient ischemic insult).
- Poor development (hypoplasia), lack of development, or degeneration of the central brain nuclei.

General Features of Moebius Syndrome

- Muscular weakness on both sides of the face, due to facial nerve paralysis. Children with Moebius syndrome have difficulty showing facial expression.
- Inability to move the eyes from side to side. This paralysis of the lateral rectus eye muscle is caused by palsy of the abducens nerve.
- Congenital deformities of the limbs, such as clubfoot, webbed fingers (syndactyly), or other deformities of the hand or foot.
- Mild mental retardation in 10 percent of affected children.
- Occasional anomalies of the ears.

Oral Features of Moebius Syndrome

- Muscular weakness on both sides of the face, due to facial nerve paralysis. This leads to difficulty in eating, swallowing, and clearing food and liquids from the oral cavity.
- Poor muscle tone of the tongue, or quivering of the tongue, due to paralysis of the tongue's (hypoglossal) nerve.
- A small lower jaw (micrognathia).
- A high arched palate, due to poor tongue function.
- Difficulty with feeding. A tendency to choke on food or liquids.
- Speech impediments.
- Poorly formed enamel on the teeth (enamel hypoplasia).

Medical Treatment for Children with Moebius Syndrome

Certain surgical procedures aim to relieve the facial paralysis in affected children. The surgeon transfers functioning muscle tissue from an unaffected part of the body to the affected area of the face. This surgical procedure helps to animate the face.

Oral Care for Children with Moebius Syndrome

- Modify and train parents on oral hygiene techniques, depending on the child's age. For small infants, the gums need to be cleaned once or twice a day with a piece of clean gauze. This will help to establish a healthy oral environment for the baby teeth. Infants should be introduced to the toothbrush around the age of one.
- Advice to parents:
  - Parents should not put children to sleep with a bottle containing any liquid other than water. Parents should encourage their infants to begin drinking from a cup around their first birthday.
  - Parents should help brush their children's teeth every day, after every meal.
  - Parents should not let their children drink fruit juice or sweetened drinks from a bottle or "tippy" cup, since this prolongs the exposure of teeth to harmful sugar.
  - Parents should provide healthy, balanced meals for children. They should limit the amount of sugar-laden foods and snacks in their diet. Plenty of healthy snacks should be available for children. Cheese products actually fight dental caries.

  - Parents can help make children's teeth more decay-resistant by using an FDI-approved children's toothpaste. Place only a pea-sized drop of toothpaste on the toothbrush. Until a child is 3 years old, parents should only use baby tooth cleanser - to avoid causing fluorosis discoloration of the adult teeth.
- Children taking oral medications should have their teeth cleansed after each dose of medication. Nearly 100% of children's medications contain sucrose, which can increase the risk of developing dental caries.
- Children should have their first oral/dental health evaluation by the age of 12 months, or within 6 months of the eruption of the first tooth.

CONGENITAL HEART DISEASE

Types

It can be cyanotic and acyanotic

Cyanotic

Features include cyanosis, hypoxemia, clubbing of fingers, and dyspnea on exertion, paroxysmal dyspneic attack and heart murmur.

Acyanotic

In this case there is minimum or no cyanosis seen.

Rheumatic heart disease: Antibiotic prophylaxis for SABE to be done.

Cardiac pace makers: Electromagnetic radiation in the dental office will interfere with normal pace maker function. The sources include electrosurgery/cautery units, ultrasonic devises, casting equipment, electronically controlled timers, microwave ovens, electric spot welders, wireless sets, and electric pulp testers; curing lights, etc. These should be switched off during any procedures for patients with cardiac pace makers.

In case of accidental interference

1. Turn off all sources
2. Cardiopulmonary resuscitation
3. Oxygen administration.

Management

It includes history taking, consultation with the physician, sedation premedication, antibiotic prophylaxis, anesthesia and surgical considerations. Contraindications are acute MI (3-6 mos.), CHF, controlled arrhythmias and uncontrolled hypertension.

RENAL DISEASE

Chronic Renal Failure

Oral Findings

Palor of the oral mucosa due to anemia. Anemia is a result of decreased erythropoietin output, decreased erythrocyte production, reduction erythrocyte life due to hypertension. Acidosis, waste product retention and electrolyte imbalance are seen. Hematomas formations, tendency to bruise and increased bleeding time, chronic marginal gingivitis, uremic gingivostomatitis resembling candidiasis are seen.

Hard tissue findings include loss of lamina dura, loss of trabeculation, ground glass appearance, and large bony lesions resembling giant cell tumors. Teeth mobility also reported. Tetracycline stains, uremic blood pigment stains, and incremental defects are the other features.

Dental Management

Factors that may influence are lack of compliance with home care request, transfer of regular unpleasant medical experiences to dental care, overprotection of the child by parents and psychological problems.

Bleeding is a concern in surgeries. Aspirin is contraindicated. Medications are used with caution for hypertension. Herpetic gingivostomatitis, severe caries, cellulitis which compromise fluid intake may lead to electrolyte imbalance and managed carefully with the help of a physician.

For dialysis patient:

- problem of serum hepatitis
- bleeding due to heparin
- optimum time for dental treatment is one day after dialysis

- increased calculus deposits (altered Ca, PO_4 solubility), increased oral prophylaxis.

For kidney transplant patient:
- Prophylactic antibiotic
- Elimination of dental sepsis.

DIABETES MELLITUS

Features include polyurea, polydipsia, polyphagia, muscular weakness, obesity or alternative weight loss, and decreased resistance to infections, poor wound healing, xerostomia and some periodontal diseases.

Management

Infections produce alteration of Carbohydrate metabolism leading to acidosis. Mild infections too may be lethal. Antibiotic usage should be prompt to treat all infections including those of oral cavity.

Antibiotic prophylaxis should be given to prevent spread of infection before the procedure by consulting the physician. Morning appointments are preferable because the blood glucose levels are better balanced. Strict oral hygiene measures are advocated.

BRONCHIAL ASTHMA

Asthma is the term doctor's use when your child's breathing becomes obstructed or blocked. Obstruction is caused when:
- Linings of airways become swollen
- Airways produce thick mucus
- Muscles around airways tighten and make the airways narrower

The obstruction leads to a smaller and smaller space through which air can move. Old stale air is trapped in the alveoli and fresh air cannot enter. Breathing in and out becomes much more difficult. This can be very scary for children to experience. It can be frightening for parents to see.

Etiology

Children with asthma have extra sensitive lungs that overreact to certain triggers. Most asthma symptoms start when bronchial tubes or airways are bothered by certain triggers. Triggers can vary widely. It is important to understand which triggers cause your child to develop symptoms. You can then work to reduce or prevent exposure to the triggers that may cause problems.

Many allergens or irritants in the environment can start an asthma episode. Most acute asthma is triggered by allergy or infection. Most long-term asthma is allergy related. Some common triggers are:
- **Irritants**
  - Cigarette smoke
  - Fire smoke
  - Perfume
  - Aerosol sprays such as from paint and hairspray
  - Weather changes and breathing cold air or very humid air
- **Allergens**
  - Mold, spores
  - Dust mites
  - Cockroaches
  - Animal dander
  - Trees, grass, ragweed, pollen
- **Infections**
  - Respiratory infections such as the common cold
- **Exercise**
  - Exertion
  - Strong emotions such as crying and laughing

Symptoms may include most of the following:
- Coughing
- Wheezing
- Shortness of breath
- Tightness in the chest
- Increased mucus production
- Increased rate of breathing
- Associated symptoms may include:
- Feeling weak, tired, sad, restless or nervous
- Pale or clammy skin
- Changes in behavior such as needing to be alone or close to a parent

Classification of asthma based upon etiology (Table 24.1)

Table 24.1: Classification of asthma based upon etiology

Classification of asthma by etiology
Aspirin-induced Asthma
Coexistent Asthma and Chronic Obstructive Pulmonary Disease
Cough-Equivalent Asthma
Exercise-Induced Asthma
Extrinsic Asthma
Factitious Asthma
Intrinsic Asthma
Mixed Asthma
Occupational Asthma
Potentially Fatal Asthma

Severity Based National Institutes of Health (NIH) Classification of Asthma (Table 24.2)

In 1997, the National Heart, Lung and Blood Institute (NHLBI) published categories of asthma. Categories are based on the frequency of symptoms over a 3-4 month period. The categories help the doctor determine the best way to treat your child's asthma. The classification also is the basis for the stepwise approach to Asthma Treatment.

- **Mild Intermittent Asthma**
  - Symptoms less than twice a week
  - Symptoms at night less than twice a month
  - No symptoms between episodes
- **Mild Persistent Asthma**
  - Weekly, but not daily symptoms
  - Episodes that may affect activity and sleep
  - Symptoms at night more than twice a month
- **Moderate Persistent Asthma**
  - Daily symptoms requiring bronchodilator inhaler use
  - Episodes that affect activity and sleep
  - Symptoms at night more than once a week
- **Severe Persistent Asthma**
  - Continuous symptoms
  - Episodes those are frequent
  - Symptoms at night all the time
  - Activities are limited because of symptoms
  - Symptoms occur while on maximal therapy

Table 24.2: Classification of asthma by severity

CLASSIFICATION	MEDICATIONS(S) USED (FREQUENCY)	CLINICAL FEATURES BEFORE TREATMENT	LUNG FUNCTION AT TIME OF TREATMENT
Step 4 Severe Persistent	Inhaled corticosteroids or inhaled β_2 agonist or ipratropium bromide or β_2 agonist tablets or syrup and/or oral corticosteroids (all used daily)	Continuous symptoms Frequent exacerbations Frequent nighttime symptoms Limited physical activity	FEV/PEF > 60 percent of predicted values PEF variability > 30 percent
Step 3: Moderate Persistent	Inhaled corticosteroids or inhaled β_2 agonist or ipratropium bromide or β_2 agonist tablets or syrup (all used daily)	Daily symptoms Exacerbations that affect activity Nighttime asthma symptoms > one time per week Daily use of inhaled short-acting β_2 agonist	FEV/PEF > 60 percent to < 80 percent of predicted values PEF variability > 30 percent
Step 2: Mild Persistent	Inhaled corticosteroids or cromolyn or inhaled β_2 agonist or ipratropium bromide or β_2 agonist tablets or syrup (all used daily)	Symptoms > two times per week but < one time per day Exacerbations that may affect activity Nighttime asthma symptoms > two times per month	FEV/PEF > 80 percent predicted value PEF variability 20-30 percent
Step 1: Mild Intermittent	Inhaled β_2 agonist or ipratropium bromide, but not more than three times per week (both used as needed)	Symptoms > two times per week Brief exacerbations (from a few hours to a few days) Nighttime asthma symptoms < two times per month Asymptomatic and normal lung function between exacerbations	FEV/PEF > 80 percent of predicted value PEF variability < 20 percent

ORAL HEALTH CHANGES IN PATIENTS WITH BRONCHIAL ASTHMA

- **Increased rate of caries development and reduced salivary flow:**
  - This phenomenon has been attributed to prolonged use of β_2 agonists, which is associated with diminished salivary production and secretion.
  - As reduced salivary flow is accompanied by concomitant increases in lacto-bacilli and *Streptococcus mutans* in the oral cavity, it is not unlikely that this particular change among asthmatic people may be one of the major contributing factors in the noted increased caries rate.
  - In addition to reduced salivary concentrations and increases in cariogenic micro-biota, higher rates of caries have been observed in people with asthma, possibly due to anti-asthmatic medications containing fermentable carbohydrate and sugar.
  - It is evident that oral prophylactic strategies should be used to address the heightened caries risk in asthmatic people.
  - This includes increased frequency of dental maintenance visits, fluoride interventions and adherence to caries-prevention measures.
- **Oral mucosal changes:**
  - The use of nebulized corticosteroids can result in throat irritation, dysphonia and dryness of mouth, oropharyngeal candidiasis and, rarely, tongue enlargement. These side effects may be attributed to the topical effects of these medications on the oral mucosa, as only 10 percent to 20 percent of the dose from an inhaler actually reaches the lungs; the rest remains in the oropharynx.
  - Using a spacer and rinsing the mouth with water after steroid inhalation can minimize the potential for candida growth.
- **Gingivitis:**
  - Use of inhaled steroids has been linked to increased levels of gingivitis; however, the common practice of mouth-breathing in asthmatic people, as well as various immunological factors, also may contribute to the observed increase in gingival inflammation.
  - It also has been suggested that asthmatic children exhibit more calculus than do healthy children. This possibly is caused by increased levels of calcium and phosphorus found in submaxillary and parotid saliva in children with asthma.

Management in Dental Care

- The administration of safe dental treatment for an asthmatic child depends on his or her pulmonary function, propensity for developing an acute asthmatic episode at the time of treatment, immune function and adrenal status.
- Preventing a sudden episode of airway obstruction is essential when treating an asthmatic patient.
- The frequency of asthmatic attacks, precipitating agents, the types of pharmacotherapy used and the length of time since an emergency visit owing to acute asthma all should be taken into account when identifying the risk of an acute exacerbation.
- Patients are instructed to carry sprays or aerosols to be used whenever there is attack
- As a general rule, elective dentistry should be performed only on asthmatic children who are asymptomatic or whose symptoms are well-controlled.
- The symptomatic person should not be treated, and the presence of asthmatic symptoms such as coughing and wheezing necessitate reappointment.
- However, the clinician also should realize that a child could have significant airway obstruction and yet be asymptomatic during dental treatment.
- Dental treatment should be postponed until the patient has fully recovered. If stress or psychological factors played a role in previous attacks, plans to reduce stress should be implemented to prevent future attacks during dental treatment.

Dental care for asthmatic patients (Table 24.3)
Emergency protocol for managing asthmatic exacerbation in a dental setting (Table 24.4)
General Recommended Drug Therapy (Based on the National Institutes of Health (NIH) Guidelines for the Stepwise Approach for Managing Asthma)

- **Mild Intermittent Asthma:**
  - Bronchodilators as needed

- **Mild Persistent Asthma:**
  - Inhaled corticosteroids or
  - Intal, theophylline and
  - Bronchodilators as needed and
  - Serevent or leukotriene modifiers if needed
- **Moderate Persistent Asthma:**
  - Inhaled corticosteroids with or without
  - Serevent, leukotriene modifiers, Tilade or theophylline and
  - Bronchodilators as needed
- **Severe Persistent Asthma**:
  - Several medications including daily oral corticosteroids

Quick Relief Medications—Asthma Relievers

These medications are used to relieve asthma symptoms such as cough, chest tightness and wheezing.

- **Short-acting beta2-agonists:** These are the medications of choice for relief of sudden symptoms and prevention of exercise- induced asthma. They work faster, use less medication and have fewer side effects than oral medications. Trade (generic) names include Maxair (pirbuterol acetate), Proventil and Ventolin (albuterol).
- **Anticholinergics:** Inhaled anticholinergics are bronchodilators that relax airways and prevent bronchial constriction. Trade (generic) name includes Atrovent (ipratropium bromide).
- **Corticosteroids (oral):** When a severe asthma episode cannot be controlled by inhaled medications, it may be necessary to give an oral corticosteroid medication to prevent further inflammation. These medications work in about 6-12 hours. Their peak effectiveness is 24 hours later. While not quick acting, they can prevent the progress of an episode. They can also speed recovery and prevent early relapses. Trade (generic) names include Deltasone and Orasone (prednisone), Methylprednisolone, Prednisolone.

SEIZURE DISORDERS IN CHILDREN

Seizures occur when nerve cells in the body misfire. Seizures are a common childhood neurologic disorder, because the young nervous system is more susceptible to seizures than the adult nervous system. About 4 to 10 percent of children have experienced an unprovoked seizure, with no recurrence. The vast majority of seizures cease spontaneously.

Table 24.3: Dental care for asthmatic patients

General Oral Health Care Instructions
Prescribe fluoride supplements for all asthmatic patients, but especially for those taking β_2 agonists
Instruct patients to rinse their mouths after using an inhaler
Reinforce oral hygiene instructions to help minimize gingivitis
Be aware of possible need to prescribe antifungal agents for patients who chronically use nebulized corticosteroids
Before Treatment
Schedule appointments for late morning or afternoon
Assess severity of asthmatic condition
Consider antibiotic prophylaxis for immunosuppressed patients
Consider corticosteroid replacement for adrenally suppressed patients
Avoid using dental materials that may elicit an asthmatic attack
Use techniques to reduce the patient's stress:
— Avoid using barbiturates
— Avoid using nitrous oxide in people with severe asthma
Have supplemental oxygen and bronchodilators available in case of acute asthmatic exacerbation
During Treatment
Use vasoconstrictors judiciously
Avoid using local anesthetics containing sodium metabisulfite
Use rubber dams judiciously
Avoid eliciting a coughing reflex
After treatment
Be aware that some patients may have an adverse reaction to nonsteroidal anti-inflammaroty drugs
Use tetracycline judiciously
Avoid use of erythromycin in patients taking theophylline
Avoid use of phenobarbitals in patients taking theophylline

Table 24.4: Emergency protocol for managing asthmatic exacerbation in a dental setting

Assessment of Severity

Accute exacerbations are manifested by episodes of bronchospasm and resulting hypoxia and hypercarbia. Management strategy is directed at determining the level of hypoxia and correcting it. The following indicate that the exacerbation is severe:

— peak expiratory flow rate, or PEFR, is at or below 50 percent of reference value;
— oxygen saturation is below 90 percent;
— bronchodilator does not improve PEFR by at least 10 percent after two treatments;
— patient has difficulty speaking;
— patient is struggling for air.

Managing an Acute Asthmatic Attack

1. Discontinue the dental procedure and allow the patient to assume a comfortable position.
2. Establish and maintain a patent airway and administer β_2 agonists via inhaler or nebulizer
3. Administer oxygen via face mask, nasal hood or cannula. If no improvement is observed and symptoms are worsening, administer epinephrine subcutaneqoulsy (1:1,00 solution, 0.01 milligram/kilogram of body weight to a maximum dose of 0.3 mg).
4. Alert emergency medical services.
5. Maintain a good oxygen level until the patient stops wheezing and/or medical assistance arrives.

Recurrent seizures which are not correctable are considered epilepsy. Children with epilepsy often have an underlying central nervous system disorder which causes behavioral problems, as well as cognitive impairment.

Signs and Symptoms of Childhood Seizures

- Seizures may present in differing ways—ranging from a brief staring spell to a life-threatening major motor seizure.
- An aura before a generalized seizure. A generalized seizure is one that simultaneously arises in several parts of the brain.
- Loss of consciousness.
- Bodily muscle spasms and violent convulsive movements.
- Temporary lack of breathing.
- Bowel or bladder incontinence.
- Increased salivation and perspiration.
- Confusion after the seizure, followed by deep sleep.

Etiology

- Genetic factors.
- Bacterial meningitis or encephalitis.
- Trauma to the head.
- Childhood fever.
- Brain tumor or stroke.
- Disturbances of the body's metabolism or electrolytes.

Epileptic Syndromes of Infancy and Childhood

- **Neonatal Seizures**: They are epilepsies of undetermined origin, and have 35% to 50% mortality.
- **Benign Focal Epilepsy**: Genetically inherited. Onset of seizures between 3 and 13 years of age.
- **West's Syndrome/infantile spasms**: The majority of children have an underlying CNS disorder. The problem may be misdiagnosed as colic. Over 90% of affected children are developmentally delayed.
- **Complex Partial Epilepsy**: Occurs at any age.
- **Lennox-Gastaut Syndrome**: Presents as generalized seizures.
- **Juvenile Myoclonic Epilepsy**: Genetically inherited. Onset between 12 and 18 years of age. The hallmark is an early morning, Myoclonic (brief jerk) seizure.
- **Febrile Seizures**: Strong genetic predisposition. Occurs in 3 percent of children between the ages of six months and five years. One third of febrile seizures are "complex," since they are either multiple, focal, or prolonged.
- **Status Epilepticus**: The grand mal is the most harmful type of status epilepticus seizure. It is defined as more than 30 minutes of continuous seizure activity, or two or more sequential seizures.

Classification of Childhood Seizures

- **Partial Seizures**: Arise in a specific area of the brain (cerebral cortex).
  Simple Partial Seizures (consciousness is maintained).
  Complex Partial Seizures (consciousness is impaired).
- **Generalized Seizures**: Convulsive (consciousness impaired): includes generalized tonic-clonic and Grand mal seizures.

Non-convulsive (altered mental status): includes absence, Myoclonic, tonic and Atonic seizures.

- **Unclassified Epileptic Seizures**

Diagnostic tools for Childhood Seizures

- EEG (electroencephalogram) is recommended for all children who experience a first seizure (except for febrile seizure).
- Blood Tests for all children who experience vomiting, diarrhea, or dehydration.
- Lumbar puncture for children younger than 18 months who are suspected of having meningitis or encephalitis.
- MRI (magnetic resonance imaging) for children with:
  Refractory seizures.
  Significant cognitive or motor impairment.
  Possible temporal lobe abnormalities.

Chemotherapy for Childhood Seizures

- Lorazepam is currently the drug of choice for treating status epilepticus seizures.
- All seizure medications have side effects.

Management of Status Epilepticus in Dental Setting

- Treatment of this medical emergency begins with evaluating and managing the "ABC's" - Airway, Breathing, and Circulation.
- Position the child to best manage ventilation, control the airway, and prevent injury. Remove all sharp restorative/surgical instruments immediately from the vicinity on/of the dental chair. Provide passive restraint to protect the child from injury.
- Deliver oxygen via a face mask. The contraction of the diaphragm during the tonic phase of the seizure may cause apnea and hypoxemia.
- An intraoral suction device will be needed. The tongue of the child, along with the increased airway secretions during a seizure may obstruct the airway.
- Dental treatment should be postponed until the patient has fully recovered. If stress or psychological factors played a role in previous attacks, plans to reduce stress should be implemented to prevent future attacks during dental treatment.

BLOOD DYSCRASIAS

Sickle Cell Anemias

It is a disease with malformed RBCs. The features include protrusion of the maxillary arch, jaundice of the extra/intraoral soft tissues.

Dental Considerations

- Minimize the stress because stress reduces the oxygenation of the tissues
- Control infection
- For sedation, only those drugs which dies not depress respiration to be used
- Maintenance of a patent airway
- Local anesthesia is not contraindicated.

HEMOPHILIAS

Factor VIII deficiency (Hemophilia A): it is of X-linked recessive inheritance with defect in factor VIII, anti-hemophilic factor. Only males are affected.

Features include spontaneous hemorrhage, easy bruising, hemarthrosis and prolonged bleeding after surgery. There will be normal bleeding time, prothrombin time, normal thrombin time, prolonged partial thromboplastin time and normal platelet count. Factor VIII assay shows 0% to 40%.

Management includes replacement of factor VIII, use of EACA (antifibrinolytic), and local and topical measures.

Factor IX Deficiency (Christmas Disease)

It is caused by the deficiency of factor IX (plasma thromboplastin component). It is also inherited as X-linked recessive trait. This is one fourth as prevalent as factor VIII deficiency.

Von Willebrand Disease

It is of autosomal dominant inheritance. There is prolonged bleeding time, decreased platelet adhesiveness, decreased platelet aggregation and possible prolonged partial thromboplastin time if factor VIII is less than 30%.

There are hemorrhages from mucous membrane, easy bruising, and menorrhagia. It becomes milder with age. Both males and females are affected. Management

includes replacement with cryoprecipitate in severe cases and local and topical measures.

Dental Management of Hemophiliacs

Prevention is of primary importance prevention of dental disease, and if disease exists, prevention of hemorrhage while the disease is controlled or eliminated. Brushing and flossing should be done with care at home. Oral prophylaxis can be done without replacement if there is minimal trauma to soft tissues. Rotary cleansing should be confined to coronal areas only. Deep scaling may require transfusion in severe hemophiliacs. Mild to moderate bleeding may be controlled with local measures like pressure or topical agents.

Local Anesthesia

Infiltration anesthesia can be given without factor coverage. A 30% to 40% level of factor coverage is done for block anesthesia. Aspiration is done to prevent Hematoma formation. Hematomas may cause potential airway obstruction due to loose connective tissue surrounding during posterior superior alveolar block or mandibular block. Periodontal injections are of choice in some cases. No replacement for intrapulpal injections.

Conservative Dentistry and Prosthodontic

Rubber dam is used to minimize trauma to the tissues. Judicious use of suctions and saliva ejector are to be avoided to avoid suction Hematomas. Wedges or matrices are used to avoid laceration to the papilla. In routine crown preparation retraction is used to expose marginal areas.

Impression trays are trimmed and edges are coated with soft wax to minimize trauma.

Pulpal Therapy

As in a normal patient but local hemostasis may be needed. Root filling should be 1 to 2 mm short of the radiographic apex.

Oral Surgery

305 to 505 replacements prior to surgery. 7 to 10 years day regimen of EACA (epsilon amino caproic acid), 100-200 mg /kg (upto 5 gm) every 6 hrs is given. EACA is contraindicated in kidney infections.

Post-Surgical Care

In case of bleeding second replacement is given. To prevent clot injury, clear liquids are advised for 24 hours after which full liquid diet begins. Utensils, straws and hard foods are contraindicated. Milk and dairy products will aggravate clot dissolution.

Full-length primary tooth requires same considerations as permanent tooth. For tissue borne primary tooth, topically applied thrombin or other hemostatic agent and pressure are used.

Cellulose bandage is used to protect the wound. Absorbable oxidized cellulose products dipped in powdered or liquid thrombin is placed inside the socket (apical 1/3rd). Topical application of concentrated anti-hemophilic factor is also preferred.

Orthodontic Therapy

No problems for orthodontic therapy. Careful appliance designing is done to prevent trauma to soft tissues. Serial extraction procedures are done as any other extraction procedure.

General Care

Chances of acquiring hepatitis are common from patients. Patients have chances of hepatitis due to transfusions.

LEUKEMIAS

General features include skin pallor, Lymphadenopathy, petechia and ecchymosis of skin.

Intraoral findings include gingival bleeding, gingival hypertrophy, mucosal pallor and pain or numbness in the mandible due to pressure of leukocyte infiltration on nerves.

Radiographic findings are loss of lamina dura, resorption of alveolar bone (apical third), alteration in periodontal space, destruction of cancellous bone and alteration in the crypts of developing teeth (position and morphology).

Candidiasis, ANUG, herpes simplex infections, Hepatitis due to frequent IV medication and Blood sampling are common.

General management: consultation with the physician. Therapeutic antibiotic should be used because of reduced resistance to infection. It is also a psychosocial problem because of fear of health care, finance, low priority for dentistry and lack of compliance for oral hygiene measures.

Oral and Craniofacial Complications of Childhood Leukemia Treatment

Neither chemotherapy nor radiotherapy differentiates between normal and malignant cells. The result is that the treatment for leukemia causes both oral and craniofacial complications. Oral problems may make it difficult for a child to receive all of his or her cancer treatment. For many leukemia patients, the oral complications are very painful and have potentially lethal consequences. Sometimes, the leukemia treatment must be stopped completely.

Causes of Oral and Craniofacial Complications of Leukemia Treatment

- Cancer treatment has a toxic effect on normal cells, as well as on cancer cells. The mouth is especially affected because its cells are replaced at a high rate. Chemotherapy and radiation therapy affect the ability of cells to divide – which creates big problems for the normal tissue repair process in the mouth.
- Chemotherapeutic agents work by affecting the rapidly dividing cells of the target tumor. These drugs also affect the protective mucosal lining of the oral cavity, the oral microbial balance, and the healing response of the oral cavity.
- Toxic changes to the oral mucosa develop when destruction of the basal cells of the epithelium exceeds the growth (proliferation) of new cells. These toxic changes result in stomatitis – which is an inflammation of the oral mucosa. Some of the highly stomatotoxic, chemotherapeutic agents include: Methotrexate, Mercaptopurine, 5-fluorouracil, and Bleomycin.
- Oral and craniofacial complications are a result of the following: the effect of chemotherapeutic agents on the bone marrow; the direct cytotoxic effects on the mucosal lining of the oral cavity; the tissue changes associated with total body radiation and radiation to the head and neck; the body's response to allogenic bone marrow transplantation and the associated immunosuppression during therapy; and the side-effects of drugs other than the anti-neoplastic drugs.
- Chemotherapeutic agents also affect the hemopoietic cells of the bone marrow, and decrease the number of neutrophils – all of which raises the risk of oral infection and immunosuppression.
- The peak age of chemotherapy treatment for childhood leukemia occurs between 2 and 3 years of age, and coincides with a critical period for dental and craniofacial growth and development in children.

Complications Caused by Chemotherapy

- The most common oral problems are: inflammation of the mucous membranes of the mouth; infection and suppression of leukocyte formation; problems with sense of taste; pain; dry mouth; and weakness of the immune system.
- Mucositis is the inflammation of the oral lining of the mouth (the mucosa) - along with redness, loss of the epithelial barrier, and ulceration.
- For many patients, mucositis is the worst part of cancer treatment. Oral mucositis may appear from 4 to 7 days after the initiation of chemotherapy. Mucositis most commonly affects the soft (nonkeratinized) oral mucosa - including the soft palate, oropharynx, buccal and labial mucosa, floor of the mouth, and the underside (ventral) and lateral surfaces of the tongue. Complete resolution of mucositis occurs seven to fourteen days after its onset.
- A dramatic decrease in the amount of salivary immunoglobulins IgA and IgG occurs.
- A dramatic decrease in the number of infection-fighting neutrophils. As a result, oral infections are common.
- Fungal (candida) infections of the oral mucosa are common, and can cause a burning sensation, distortion of taste, and problems with swallowing.
- Viral infections, especially the reactivation of herpes simplex virus type I (HSV-1), are serious because they

can cause pain and problems with hydration and nutrition.

- Spontaneous oral bleeding is due to a cytotoxic, drug-induced, decrease in the number of platelets (thrombocytopenia). This dramatic decrease in platelets leads to spontaneous oral bleeding when the platelet count goes below 20,000 per cubic mm.
- The cells which form dentin (the odontoblasts), and the cells which form enamel (the ameloblasts), can be damaged by chemotherapy agents if these cells are in a susceptible phase of their cell cycle (the M or S stage). The end result may be teeth which have short, thin, tapered roots, or hypomineralized or hypomature enamel.

Complications Caused by Radiation Therapy

- Radiation damages the blood vessels, cartilage, salivary glands, bone, and muscles of the neck and jaw. This results in exposed bone, tissue breakdown, pain, and tissue death (necrosis).
- Mucositis occurs when doses of radiation greater than 180 cGy/day are given 5 days per week.
- When radiotherapy in adolescents is directed at the salivary glands, the most immediate effect is oral dryness (xerostomia), caused by damage to the salivary glands. The parotid gland then produces only thick, ropy saliva-after two or three doses of irradiation of 200 cGy/dose. Irreversible salivary gland injury occurs when the radiation dose exceeds 5,000 cGy.
- Direct radiation of the jaw, face, and head produces the most serious side effects in children, because they are growing and developing so rapidly. Head and neck radiation in children may cause incomplete development of the jaws, the bone around the eyes (the orbits), and the facial skeleton. These complications are due to damage to the cartilaginous growth centers.
- Head and central nervous system irradiation may interfere with normal craniofacial development. This is because radiation directed at the hypothalamus and pituitary glands will affect their function – resulting in decreased production of Growth Hormone and Thyroid-Stimulating Hormone.
- Head and neck radiation in children may cause arrested tooth development, which causes: small teeth (microdontia), wasting-away (atrophy) of the overlying soft tissue, enamel malformation (hypoplasia), incomplete calcification of teeth, and arrested root development. Teeth with arrested root development have roots with severe tapering and narrowing, and blunting of the apex.

Complications Caused by Bone Marrow Transplantation

- The "pre-transplant" protocol includes chemotherapy and/or radiotherapy. Both of these can cause undesirable oral and craniofacial effects. The effects include graft-versus-host-disease (GVHD), xerostomia, infections, and late dental and craniofacial abnormalities.
- Children undergoing an allogenic bone marrow transplant (BMT) often develop GVHD, in which the transplanted T-lymphocytes recognize the child's body as foreign, and attack it. The oral cavity may be the first or only site showing GVHD.
- An increasing level of xerostomia and/or generalized stomatitis which appears 100 or more days after the BMT are indications of chronic GVHD. Xerostomia may be present as long as 6 to 8 months after the BMT.
- Chronic GVHD causes oral infections after 100 days post-BMT. These opportunistic infections are facilitated by: xerostomia, long-term steroid or antibiotic use, and systemic immunosuppression. The oral infections include: periodontal infections, dental abscesses, herpes simplex infections, cytomegalovirus infections, and candida infections.
- The younger the child, the greater the risk for craniofacial and dental developmental abnormalities. Children who receive total body irradiation have more severe problems with dental development than those who receive only chemotherapy.

Management of the Oral Complications of Leukemia Treatment in Children

The greatest challenge for young patients may be dealing with the oral complications of leukemia treatment. The

oral complications are very painful and have potentially lethal consequences. Up to 90 percent of children undergoing leukemia treatment may suffer oral complications. The complications occur because neither chemotherapy nor radiotherapy differentiates between normal and malignant cells. Oral problems may make it difficult for a child to receive all of his or her cancer treatment. Sometimes, the leukemia treatment must be stopped completely.

The severity of oral complications can be greatly reduced when an aggressive approach to oral care is taken prior to leukemia treatment. Elimination of pre-existing dental problems, as well as good oral hygiene during leukemia therapy can reduce the occurrence and severity of oral complications. Managing the oral complications is therefore important for the young patient's well-being.

Management of Oral Complications

- Oral management before leukemia treatment.
- Oral management during leukemia treatment.
- Oral hygiene during leukemia treatment.
- Emergency dental care during leukemia treatment.
- Oral management after bone marrow transplantation.
- Oral management after completion of leukemia treatment.

Oral Management before Leukemia Treatment

- At least one month prior to initiation of leukemia treatment, all children should be evaluated by either a general dentist or pediatric dentist. During this evaluation, the dentist will note the health status of the oral cavity, identify any potential oral problems, and plan for treatment which may reduce oral complications during and after cancer therapy.
- A panoramic radiograph should be attempted in all children who can cooperate before cancer treatment is started.
- Oral hygiene and dental rehabilitation should be as aggressive as possible before cancer treatment is started, especially when bone marrow transplantation is planned. Dental treatment should only be provided after consultation with the child's oncologist
- The dentist should treat existing dental problems in a manner which will decrease the risk of oral complications later. The dentist will extract teeth which: have acute or chronic infection, are mobile, or grossly carious. Pulpotomies and pulpectomies should not be performed on primary teeth, due to possible treatment failure and subsequent infection. Ideally, all extractions should be completed 10 days prior to the initiation of cancer treatment.
- Orthodontic appliances, braces, removable retainers, and space maintainers should be removed prior to cancer treatment.

Oral Management during Leukemia Treatment

- The child should rinse with cold sterile water, or cold normal saline as often as possible, but at least 6 times a day. This will: keep the oral tissues clean and wet, help with the removal of thick saliva, remove debris, and decrease the risk of opportunistic infections.
- An effective way to increase the child's comfort during leukemia treatment is to encourage the intake of popsicles, ice chips, and icy drinks.
- Lip moisturizer should be used, and it should be water or lanolin based. It should be applied with a cotton tip applicator several times each day.
- Young patients should also rinse and spit with water after episodes of vomiting or emesis. This will prevent stomach acids from decalcifying the teeth and irritating the oral tissues. To control persistent nausea, the dentist or physician may need to prescribe Ondansetron.
- A good dietary management program should encourage the intake of: bland, non-acidic foods with high moisture content (ice-cream, popsicles, milk shakes, custard, cream soups, mashed potatoes, Carnation Instant Breakfast, and puree foods).
- Chlorhexidine rinses are not recommended for children because: they contain alcohol, desiccate the oral tissue, and have an unpleasant taste.
- The daily use of topical fluoride gel is contraindicated in children less than six years of age because of the increased risk of fluorosis.
- Fungal infections, such as candida, need to be treated aggressively. Fluconazole is often prescribed for fungal infections related to cancer treatment.

- All mucositis lesions should be biopsied for the presence of herpes simplex virus, unless the child is already taking Acyclovir.

Oral Hygiene during Leukemia Treatment

- The child should rinse with cold sterile water, or cold normal saline as often as possible, at least 6 times a day. This will keep the oral tissues clean and wet, help with removal of thick saliva, remove debris, and decrease the risk of opportunistic infections.
- The child should brush with a soft toothbrush after every meal. Tooth brushing should be done under the supervision of hospital staff to ensure that it is being done safely. Sponges or foam toothettes are not recommended because they are not effective in removing plaque and debris; the use of toothettes is a last resort.
- Brushing should be discontinued when the platelet count is less than 20,000 per cubic mm, or when the absolute neutrophil count is less than 500 per cubic mm. Below these levels, dental hygiene should be performed using only a moist gauze or spongy toothette.
- Children under 3 years of age should use a Baby Tooth Cleanser instead of toothpaste. Children older than 3 should use a non-tartar control, non-peroxide, flavor-free children's fluoridated toothpaste.
- Hydrogen peroxide rinses are not recommended because they may delay wound healing and cause drying of the mucosa.

Emergency Dental care during Leukemia Treatment

- All emergency dental procedures should be performed after consultation with the pediatric oncologist.
- Appropriate timing of dental procedures is critical, since the peripheral white blood cell counts change dramatically during chemotherapy. The risk of infection is greatest when the child's neutrophil count reaches a low point, generally 7 to 10 days after induction therapy.
- Dental treatment should only be performed if the absolute neutrophil count (ANC) has improved and exceeds 1,000 per cubic mm, and if the platelet count (at least 50,000 per cubic mm) and function are adequate.
- Dental surgical procedures, as well as administration of local anesthetic blocks should be avoided during periods of thrombocytopenia. Clotting factors should be normal. A platelet transfusion may be needed if the platelet count is less than 50,000 per cubic mm.
- Prophylactic antibiotic coverage should be provided according to the American Heart Association's recommendations, and in consultation with the child's oncologist.
- Some of the circumstances requiring prophylaxis: when chemotherapy is delivered through a vascular access device, such as a central venous catheter or Hickman line; when the child has an ANC of less than 1,000 per cubic mm and/or the WBC count is less than 2,000 per cubic mm; when the child is on long-term immunosuppressive drugs, such as cyclosporine or prednisone.
- All patients who have a central line must have antibiotic coverage according to the American Heart Association's guidelines before undergoing dental procedures that are likely to cause bleeding or bacteremia. The dentist may need to provide extra antibiotic coverage even if the child is already taking prophylactic antibiotics.

Oral Management after Bone Marrow Transplantation

- Children who have received a BMT may not be able to undergo any dental procedures for up to one year after the procedure, due to the profound impairment of the immune function and Graft Versus Host Disease.
- Before providing any dental treatment for BMT patients, the dentist should at least be aware of the child's platelet count and the absolute neutrophil count. The child's oncologist must also be consulted.
- Elective dental work should be deferred when the ANC is less than 1,000 per cubic mm, due to the increased risk of developing an infection. The severity of infection is inversely related to the number of

neutrophils, since they are the body's first line of defense.

Oral Management after Completion of Leukemia Treatment

- The child should have a dental recall appointment every 3 months for the first year after treatment, and every 6 months thereafter.
- During the dental recall visits, the dentist should review the latest blood laboratory studies to determine if the child's hematological status has returned to normal.
- The dentist should inform the parents about the possible long-term side-effects of cancer treatment on the teeth, oral mucosa, and craniofacial complex.
- If radiation therapy was directed at the head or neck region, the dentist should consult with the radiation oncologist before performing any dental extractions or osseous surgery. A panoramic radiograph should be attempted in all children who can cooperate, especially if the BMT was performed prior to 7 years of age.

FETAL ALCOHOL SYNDROME AND FETAL ALCOHOL EFFECTS

Fetal alcohol syndrome (FAS) is an alcohol-related birth disability. The condition occurs from alcohol use during pregnancy. When a pregnant woman drinks alcohol, it passes through the placenta and is absorbed by the unborn baby. The alcohol can harm the embryo and fetus even if the mother feels no effects.

FAS is a condition which includes physical, mental and behavioral abnormalities. Most children with FAS will have different facial features. Most will have problems with growth. Permanent brain injury frequently occurs.

Not all children with FAS are alike. The effects of alcohol use during pregnancy will vary. Some children are more severely affected than others. Some show more of the reasoning and behavioral problems than the physical features. Some have a normal IQ while others do not. Many will have learning disabilities. Each child will have his or her own special needs, problems and potential.

Some children are affected by alcohol but do not have all the features of FAS. These children may be diagnosed with Fetal alcohol effects (FAE). They may have normal growth and a more normal appearance. They are more likely to have a normal IQ than a child with FAS. Like children with FAS, those with FAE can have mild to severe problems. These may involve problems with reasoning, behavior and learning.

The Danger of Alcohol Use in Pregnancy

There is no known safe amount of alcohol use during pregnancy. There is also no known time when drinking alcohol is safe. Alcohol can do more damage to the developing embryo and fetus than illegal or legal drugs. Pregnant women should not drink alcohol at all. No alcoholic beverage is safe during pregnancy.

According to **Streissguth and Little (1994)**

- **Alcohol use in the first trimester may:**
  - Cause the greatest brain damage
  - Impair cell development
  - Affect major organs such as the heart, liver and kidneys
  - Cause facial malformations
  - Cause miscarriage
- **Alcohol use in the second trimester may:**
  - Impair brain development
  - Cause miscarriage which may be life threatening for the mother
  - Damage muscles, skin, teeth, glands and bones
- **Alcohol use in the third trimester may:**
  - Impair brain and lung development
  - Prevent adequate weight gain for the fetus
  - Cause early labor and delivery

Characteristics of FAS/FAE

Not all children with FAS/FAE are alike. The effects range from mild to severe. They depend on the amount of alcohol used and the time in which it is used. They also depend on the mother's diet, age and drinking history. Typically, children with FAS/FAE have more physical, developmental and behavioral problems than other children. Early identification is the first step to an improved prognosis.

The following are the most common characteristics found in children affected by alcohol use during pregnancy. Remember, not all children with FAS/FAE have all the characteristics.

Physical Characteristics Related to FAS/FAE

- **Growth deficiencies may include:**
  - Low birth weight
  - Small size for age in weight and length
  - Small head for age
  - Failure to thrive
- **Facial malformations may include:**
  - Short eye slits
  - Droopy eyelids
  - Widely spaced eyes
  - Nearsightedness
  - Crossed eyes
  - Short upturned nose
  - Low and/or wide bridge of the nose
  - Flat or smooth area between the nose and lip
  - Thin upper lip
  - Flat midface
  - Small underdeveloped jaw
- **Other effects may include:**
  - Large or malformed ears
  - Underdeveloped fingernails or toenails
  - Short neck
  - Poor eye-hand coordination
  - Hearing problems
  - Joint and bone abnormalities
- **Dental effects:**
  - Poor oral hygiene
  - Oral soft tissue lesions like dentoalveolar abscess, angular cheilitis, herpetic and aphthous ulcerations are common
  - Enamel opacities / hypoplasia
  - Higher incidence of caries
  - Delayed eruption of teeth
  - Malocclusion
  - Cleft anomalies

Behavioral Characteristics Related to FAS/FAE

Children with FAS/FAE often have behavior problems due to brain injury. Some have more behavioral problems than others. Some are so severely affected that they cannot function independently in the community. Behavior problems will vary. They include:

- Hyperactivity
- Stubbornness
- Impulsiveness
- Passiveness
- Fearlessness
- Irritability
- Sleep difficulties
- Teasing or bullying of others

Other Effects may Include

- Hypersensitivity to sound and touch
- Difficulty with change
- Organizational difficulties
- Poor self-image
- Over stimulation difficulties
- Depression or withdrawal
- Problems with truancy
- Problems with sexuality

Learning Difficulties Related to FAS/FAE

Children with FAS/FAE frequently have learning difficulties. These difficulties result from poor thinking and processing skills. Information may be known, but cannot be applied to different situations. Learning may occur in spurts. Easy learning periods may be followed by harder ones. During difficult periods, children may have trouble remembering and using their learned information. Because of inconsistent learning, teachers may think they are just not trying. They may label them as lazy or stubborn.

Difficulties may Include

- Developmental delays
- Attention deficit
- Poor organization skills
- Problems with memory
- Poor mathematical skills
- Difficulty with abstract concepts
- Difficulty learning from past experiences
- Difficulty understanding cause and effect
- Speech delays, stuttering and stammering

Malbin (1994) on Inappropriate Social Skills Related to FAS/FAE

Children with FAS/FAE often show socially inappropriate behavior due to impaired practical reasoning skills. They also may be unable to consider results of their actions. They may miss cues used as subtle messages like gestures and facial expressions.

They may be socially and emotionally immature and have difficulty getting along with peers

Children with FAS/FAE can be easily influenced by others. Due to their trusting nature and eagerness to please, random attraction to strangers may occur. They may be vulnerable to manipulation and victimization. This can cause concern for caregivers. Constant supervision may be required.

Positive Characteristics of Children with FAS/FAE

Children with FAS/FAE have many valuable qualities and talents. With early identification, intervention and family support many can develop their best qualities. These children may be:

- Cuddly, cheerful and tactile
- Friendly and happy
- Caring, kind, loyal, nurturing and compassionate
- Trusting and loving
- Determined, committed and persistent
- Curious and involved
- Energetic, hard working and athletic
- Artistic, musical and creatively intelligent
- Fair and cooperative
- Highly verbal
- Kind with younger children and animals
- Able to have long-term visual memory
- Able to participate in problem solving

MENTAL RETARDATION

It is a significantly sub-average general intellectual functioning, existing concurrently with deficits in adaptive behavior and manifested during the development period. It is a common cause of developmental delay and the most handicapping of the childhood diseases. Children with an IQ below 70 are known as mentally retarded and within 70 to 85 are known as borderline.

Causes: Downs's syndrome, premature birth, trauma and maternal infections.

IQ	*Level of impairment*	*Ability*
85-70	Borderline	Slow learner
69-55	Mild	Educatable
54-40	Moderate	Trainable
39-25	Severe	Dependent
<24	profound	Totally dependent

Slow learners can adapt to dental situation easily. They usually succeed in meeting the challenge.

- **Mild retardation** can cope with simple preventive and short predictable operative procedures. They often try hard to please every one and tend to thrive on affection.
- **Moderate retardation** cases respond well in dental situation. They may require sedation/restraints for simple procedures.
- **Severe/profound** cases cannot cope with even simple tasks and general anesthesia is a must.

Oral Manifestations

Anomalies of teeth and face, malocclusion, open bite, eruption disturbances, hypoplasia/hypocalcification, dental caries in early age groups and periodontal diseases in older age groups.

Proper oral hygiene measures, fluoride applications are needed. Chlorhexidine mouth rinses are advocated. Behavior management techniques based on the need to be advocated.

Hearing Impairment

About one out of every thousand newborns have profound or severe hearing loss. About three times as many infants are born with mild to moderate hearing loss. Another 3/1000 children develop hearing loss during childhood.

The ability to develop speech is directly related to the ability to hear sounds. The first year of life is very important for language development. The goal of treatment is therefore to enhance the process of language development as early as possible.

Types of Hearing Impairment

- Conductive hearing loss is caused by a mechanical problem in the ear. It is the most common type of hearing problem in children. This type of hearing loss is often caused by earaches (Otitis media).
- Sensorineural hearing loss occurs when there is damage to the inner ear, or neural portions of the hearing mechanism. The most common disease affecting the inner ear is loss of the hair cells of the cochlea.

Etiology

- Disease in any part of the hearing system can cause hearing loss in children.
- Repeated ear infections may lead to permanent hearing loss.
- Malformations of the external auditory canal and middle ear can cause conductive hearing loss.
- A benign tumor, called a cholesteatoma, can result in significant conductive hearing loss.
- Acquired sensorineural hearing loss can be caused by many factors such as: bacterial meningitis, viral infections, loud noises, head trauma, or autoimmune disorders. Mumps is a frequent cause of unilateral hearing loss.

Diagnosis

- All infants and children should be screened for hearing loss as early as possible so that treatment may be started as soon as possible.
- The birth admission hearing screen is performed on newborns before their discharge from the hospital. Severe bilateral hearing loss is usually diagnosed during the first week of life when the newborn does not respond to voices or sounds.
- Tympanometry is used to help diagnose the cause of conductive hearing loss. This test does not require the active participation of the child. The testing device consists of a sound source and a microphone which are placed in the ear canal for a few seconds.
- Acoustic reflex testing uses a similar device, and measures the contraction of the tiny stapedius muscle of the middle ear.

Treatment

- Conductive hearing loss can be improved by surgical drainage of ear fluids (Myringotomy), or by a hearing aid.
- Sensorineural hearing loss can be treated by hearing aids.
- Children with profound bilateral hearing loss are candidates for a cochlear implant. The cochlear implant is fairly complex, and uses electrodes which stimulate the auditory nerve of the cochlea.

Role of Dentist

- Avoid delays in starting dental treatment.
- Keep dental procedures fairly short.
- Invite a parent into the dental operatory to help with communication.
- Don't wear the protective mask when speaking to the hearing impaired child.
- Reduce background noise and music during the appointment.
- Explain to the child what will be done, and demonstrate some of the instruments that will be used.
- The dentist should consider learning some basic hand signs to improve communication.
- The dental staff should be sensitive to the child's non-verbal communication.

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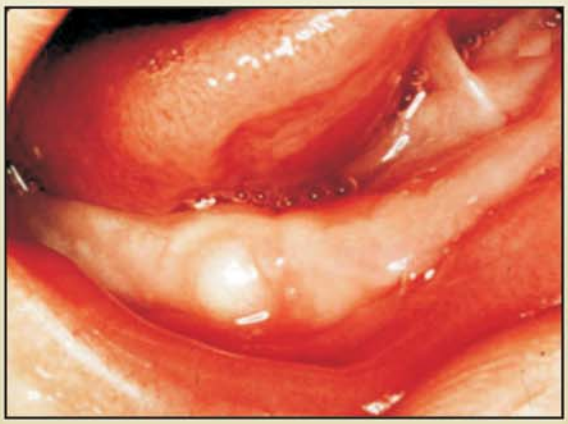

Oral Pathologic Conditions

INTRODUCTION

The incidence of pathological conditions of the mouth and perioral structures differs between children and adults. For example, mucoceles are more common in the young, whereas squamous cell carcinomas occur more frequently in older individuals. The management of pathology in the child differs from that in the adult. Growth and development may be affected by the disease, or its treatment. On a more practical basis, anesthetic considerations for surgical treatment of simple pathological conditions can make management more complex. This chapter deals with those conditions that occur exclusively, or more commonly in children.

LESIONS OF THE ORAL SOFT TISSUES

Conditions affecting the oral mucosa and associated soft tissues can be classified as: infections, ulcers, vesiculobullous lesions, white lesions, cysts, and tumors.

INFECTIONS

Viruses, bacteria, fungi, or protozoa may cause infections of the oral mucosa.

Viral Infections

Herpetic

Primary herpes simplex infection—acute herpetic gingivostomatitis: This condition usually occurs in children between the ages of 6 months and 5 years. Circulating maternal antibodies usually protects young babies.

Symptoms:

- Incubation period of 5-7 days
- Prodromal symptoms precede 1-2 days
- Fever, headache, malaise, nausea, vomiting
- Vesicles with inflammatory base appear on the mucosa and rapidly collapse to form numerous small red lesions. These initial lesions enlarge slightly and develop central areas of ulceration, which are covered by yellow fibrin
- Gingiva becomes extremely erythematous with distinctive punched out erosions along the midfacial free gingival margins

Secondary herpes simplex infection: Secondary infection with herpes simplex usually occurs at the labial Mucocutaneous junction and presents as a vesicular lesion which ruptures and produces crusting

Herpes Varicella-zoster

Shingles, which is caused by the Varicella zoster virus, is much commoner in adults than children. The vesicular lesion develops within the peripheral distribution of a branch of the trigeminal nerve. Chickenpox, a more common presentation of Varicella–zoster in children, produces a vesicular rash on the skin. The intraoral lesions of chickenpox resemble those of primary herpetic infection. The condition is highly contagious.

Mumps

Mumps produces a painful enlargement of the parotid glands. It is usually bilateral. The causative agent is a myxovirus. Associated complaints include headache, vomiting and fever. Symptoms last for about a week and the condition is contagious.

Measles

The intraoral manifestation of measles occurs on the buccal mucosa. The lesions appear as white speckling surrounded by a red margin and are known as Koplick's spots. The oral signs usually precede the skin lesions and disappear early in the course of the disease. The skin rash of measles normally appears as a red maculopapular lesion. Fever is present and the disease is contagious.

Rubella

German measles does not usually produce signs in the oral mucosa, however, the tonsils may be affected. Protection against the diseases of mumps, measles, and Rubella can be achieved by vaccinating children in their years with MMR vaccine.

Herpangina

This is a coxsackievirus A infection. It can be differentiated from primary herpetic infection by the different location of the vesicles, which are found in the tonsillar or

pharyngeal region. Herpangina lesions do not coalesce to form large areas of ulceration. The condition is short-lived.

Hand, Foot, and Mouth Disease

This coxsackievirus A infection produces a maculopapular rash on the hands and feet. The intraoral vesicles rupture to produce painful ulceration. The condition lasts for 10-14 days.

Infectious Mononucleosis

The Epstein-Barr virus causes this condition. It is not uncommon among teenagers. The usual form of transmission is by kissing. Oral ulceration and petechial hemorrhage at the hard/soft palate junction may occur. There is lymph node enlargement and associated fever. There is no specific treatment. It should be noted that the prescription of Ampicillin and amoxicillin can cause a rash in those suffering from infectious mononucleosis. These antibiotics should be avoided during the course of the disease. Treatment of the viral illnesses is symptomatic and relies on analgesia and maintenance of fluid intake. It must be remembered that aspirin should be avoided in children less than 12 years of age.

Human Papilloma Virus

This is associated with a number of tumor-like lesions of the oral mucosa, which are discussed below.

Bacterial Infection

Staphylococcal Infections

Staphylococci and streptococci may be cause impetigo. This can affect the angles of the mouth and the lips. It presents as crusting vesiculobullous lesions. The vesicles coalesce to produce ulceration over a wide area. Pigmentation may occur during healing. The condition is self-limiting, although antibiotics may be prescribed in some cases. Staphylococcal organisms can cause osteomyelitis of the jaws in children. Although the introduction of antibodies has reduced the incidence of severe forms of the condition, it can still be devastating. In addition to aggressive antibiotic therapy, surgical intervention is required to remove bony sequestrate.

Streptococcal Infections

Streptococcal infections in childhood vary from a mucopurulent nasal discharge to tonsillitis, pharyngitis, and gingivitis. Scarlet fever is a ß hemolytic streptococcal infection consisting of a skin rash with maculopapular lesions of the oral mucosa. It is associated with tonsillitis and pharyngitis. The tongue shows characteristics changes from a strawberry appearance in the early stages to a raspberry like form in the later stages.

Congenital Syphilis

Congenital syphilis is transmitted from an infected mother to the fetus. Oral mucosal changes such as rhagades, which is a pattern of scarring at the angle of the mouth, may occur. In addition, this disease may cause characteristic dental changes in the permanent dentition. These include Hutchinson's incisors (the teeth taper towards the incisal edge rather than the cervical margin) and mulberry molars (globular masses of enamel over the occlusal surface).

Tuberculosis

Tuberculous lesions of the oral cavity are rare: however tuberculous lymphadenitis affecting submandibular and cervical lymph nodes is occasionally seen. These present as tender enlarged nodes, which may progress to abscess formation with discharge through the skin. Surgical removal of infected glands produces a much neater scar than that caused by spontaneous rupture through the skin if the disease is allowed to progress.

Cat-scratch Disease

This is a self-limiting disease which presents as an enlargement of regional lymph nodes. The nodes are painful and enlargement occurs up to 3 weeks following a cat-scratch. The nodes become suppurative and may perforate the skin. Treatment often involves incision and drainage.

Fungal Infections

Candida

Neonatal acute candidiasis (thrush) contracted during birth is not uncommon. Likewise young children may develop the condition when resistance is lowered or after antibiotic therapy. Easily removed white patches on an erythematous or bleeding base are found. Treatment with Nystatin or Miconazole is effective (those under 2 years of age should receive 2.5 ml of a Miconazole gel (25 mg/ml) twice daily; 5 ml twice daily is prescribed for those under 6 years of age, and 5 ml four times a day for those over 6 years of age).

Actinomycosis

Actinomycosis can occur in children. It may follow intraoral trauma including dental extractions. The organisms spread through the tissues and can cause dysphagia if the submandibular region is involved. Abscesses may rupture on to the skin and long-term antibiotic therapy is required. Penicillin should be prescribed and maintained for at least 2 weeks following clinical care.

Protozoal Infections

Infection by *Toxoplasma gondii* may occasionally occur in children. The principal reservoir of infection being cats. Glandular toxoplasmosis is similar in presentation to infectious mononucleosis and is found mainly in children and young adults. There may be a granulomatous reaction in the oral mucosa and there can be parotid gland enlargement. The disease is self-limiting, although an anti-Protozoal such as pyrimethamine may be used in cases of severe infections.

ULCERS

Traumatic ulceration of the tongue, lips, and cheek may occur in children, especially after local anesthesia has been administrated recurrent aphthous oral ulceration not associated with systemic disease is often found in children. One or more small ulcers in the non-attached gingiva may occur at frequent intervals. In the young child the symptoms may be mistaken for toothache by a parent. The majority of aphthous ulcers in children are of the minor variety (less than 5 mm in diameter). These usually heal within 10-14 days. Treatment other than reassurance is often unnecessary.

However, topical steroids (Adcortyl in Orabase or Corlan pellets) may be prescribed in severe cases. Older children may benefit from the use of antiseptic rinses to prevent secondary infection. In the absence of a history of major aphthous ulceration any ulcer lasting for longer than 2 weeks should be regarded with suspicion and biopsied.

VESICULOBULLOUS LESIONS

Vesiculobullous lesions cause ulcers in the later stages of such conditions. Viral causes have been mentioned above. Similarly, conditions such as epidermolysis bullosa and erythema multiformae can produce oral ulceration in children. The major vesiculobullous conditions such as pemphigus and pemphigoid are rare in young patients.

Epidermolysis bullosa is a term that covers a number of syndromes, some of which are incompatible with life. The skin is extremely fragile and mucosal involvement may occur. The act of suckling may induce bullae formation in babies. In older children, effective oral hygiene may be difficult as even mild trauma can produce painful lesions.

The oral lesions of erythema multiformae usually affect the lips and anterior oral mucosa. There is initial erythema followed by bullae formation and ulceration. The pathogenesis of the condition is still unclear; however, precipitating factors include drug therapy and infection. Treatment includes the use of steroids and oral antiseptic and analgesics rinses to ease the pain.

WHITE LESIONS

Trauma of either a chemical or physical nature, for example, burns and occlusal trauma can cause white patches intraorally.

White Spongy Naevus

The white spongy naevus (also known as the oral epithelial naevus) is a rough folded lesion that can affect any part of the oral mucosa. It often appears in infancy. It is benign.

Leucoedema

This is a folded, white translucent appearance found in children of races who exhibit pigmentation of the oral mucosa. It is considered as variation of normal.

Candidiasis

The white patches of acute fungal candidiasis mentioned above are readily removed in contrast to the white lesions discussed here.

Geographic Tongue

This condition may be seen in children. It is normally symptomless, although some patients complain of discomfort with spicy foods. Areas of the tongue appear shiny and red due to loss of filiform papillae. These red patches are surrounded by white margins. These areas disappear before reappearing in other regions of the tongue. The condition is benign and requires no treatment apart from reassurance to the child and parent.

CYSTS

Mucoceles

The peak incidence of mucoceles in the second decade of life; however they are not uncommon in younger children including neonates. Mucoceles are caused by trauma to minor salivary glands or ducts and are often located on the lower lip. They are the commonest non-infective causes of salivary gland swelling in children. Salivary tumors are rare in this age group.

Ranula

This appears as a bluish swelling of the floor of the mouth. It is essentially a large mucocele. It may arise from part of the sublingual salivary gland.

Bohn's Nodules

These gingival cysts arise from remnants of the dental lamina. They are found in neonates. They usually disappear spontaneously in the early months of life.

Gingival Cyst of the Newborn/Epstein's Pearls (Fig. 25.1)

Lesion (or lesions) appearing as white dots on the tooth bearing gingiva. These keratin-containing soft tissue cysts are harmless; they disappear with tooth eruption; no treatment is necessary.

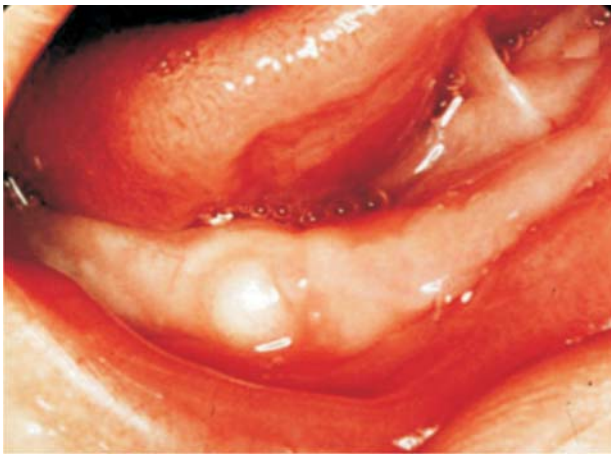

Fig. 25.1: Gingival cyst of the newborn/Epstein's pearls

Gingival cysts of the newborn are relatively common. They appear as single or multiple discrete white spots on the gingiva overlying tooth bearing areas. They are also known as "dental lamina cysts of the newborn" or, more commonly, as "Epstein's pearls." These lesions are, on microscopic examination, small keratin-filled cysts; however, they are not related nor behave like odontogenic keratocysts. No treatment is necessary as these lesions will disappear as teeth erupt.

Dermoid Cysts

These are rare lesions of the floor of the mouth. They appear as intraoral and Submental swellings. They are derived from epithelial remnants remaining from fusion of the mandibular processes.

Lymphoepithelial Cyst

In the past this was termed branchial arch cyst as it was thought to arise from epithelial remnants of a branchial arch. They are normally found in the sternomastoid region, although they can present in the floor of the mouth. Histologically the cyst wall contains lymph tissue. The tissue of origin is thought to be salivary epithelium.

Thyroglossal Cyst

This cyst which arises from the thyroglossal duct epithelium may present intraorally. The mouth, however,

is a rare site. Most arise in the region of the hyoid bone.

TUMORS

Congenital Epulis

This is a rare lesion that occurs in neonates. It normally presents in the anterior maxilla. It consists of granular cells covered by epithelium and is thought to be reactive in nature. This is a benign lesion and simple excision is curative.

Melanotic Neuroectodermal Tumor

This rare tumor occurs in the early months of life, usually in the maxilla. The lesion consists of epithelial cells containing melanin with a fibrous stroma. Some localized bone expansion may occur. The condition is benign and simple excision is curative.

Squamous Cell Papilloma

This is a benign condition that occurs in children. The small cauliflower-like growths, which vary in color from pink to white, are usually solitary lesions. They may be due to the human papillomavirus.

Verruca Vulgaris

This condition also known as the common wart may present as solitary or multiple intraoral lesions. These may be associated with skin warts. They are probably caused by the human papillomavirus.

Focal Epithelial Hyperplasia

This is a fairly common lesion that presents as a firm pink lump. It normally affects the buccal mucosa at the occlusal level. They are caused by trauma. They are usually symptomless unless further traumatized and are easily removed.

Fibrous Epulis

This is present as a mass on the gingiva. Color varies from pink to red depending upon the degree of vascularity of the lesion. It consists of an inflammatory cell infiltrate and nature fibrous tissue, occasionally a calcified variant is found. Surgical excision is curative.

Pyogenic Granuloma

These commonly occur on the gingiva usually in the anterior maxilla. They are probably a reaction to chronic trauma, especially from a sub-gingival calculus. Due to their etiology they have a tendency to recur after removal.

Peripheral Giant Cell Granuloma

This dark red swelling of the gingiva can occur in children. It often arises interdentally. Radiographs may reveal some loss of the interdental crest. The central giant cell granuloma shows much greater bone destruction. This condition is thought to be a reactive hyperplasia. Unless excision is completed it will recur.

Hemangiomas

Hemangiomas are relatively common in children. They are malformations of blood vessels. They are divided into cavernous and capillary variants, although some lesions contain elements of both. Capillary hemangiomas may present as facial birthmarks. The cavernous hemangioma is a hazard during surgery if involved within the surgical site. It is a large blood-filled sinus that will bleed profusely if damaged. The extent of a cavernous hemangioma can be established prior to surgery using either angiography or magnetic resonance imaging (MRI) scanning. Small hemangiomas are readily treated by excision or cryotherapy. Larger lesions are amenable to laser therapy.

Sturge-Weber Syndrome

Sturge-Weber angiomatosis is a syndrome consisting of a hemangioma of the leptomeninges with an epithelial facial hemangioma closely related to the distribution of branches of the trigeminal nerve. Mental deficiency, hemiplegia and ocular defects can occur. Intraoral involvement may interfere with the timing of eruption of the teeth (both early and delayed eruption have been reported).

Lymphangiomas

Lymphangiomas are benign tumors of the lymphatic. The vast majority are found in children. The head and neck

region is a common site. The cystic hygroma is a variant that appears as a large neck swelling, which may extent intraorally to involve the floor of the mouth and tongue.

Neurofibromas

These may present as solitary or multiple lesions. They are considered hamartomas (a haphazard arrangement of tissue). They present intraorally as mucosal swellings on the tongue or gingivae. Multiple oral neuromas are a feature of the multiple endocrine neoplasia syndromes. As the oral signs may precede the development of more serious aspects of this condition (such as carcinoma of the thyroid). Children presenting with multiple lesions should be referred to an endocrinologist.

Orofacial Granulomatosis

Orofacial granulomatosis (OFG) is not a tumor in the true sense nor a distinct disease entity, but describes a clinical appearance. Typically there is diffuse swelling of one or both lips and cheeks, folding of the buccal reflected mucosa and occasionally gingival swelling and oral ulceration. This may represent a localized disturbance due to an allergic reaction to foodstuffs, toothpaste, or even dental materials. Alternatively, the appearance may be due to any underlying systemic condition such as sarcoidosis or Crohn's disease.

Melkersson-Rosenthal Syndrome

This is a condition that generally begin during childhood. It consists of chronic facial swelling (usually the lips), facial nerve paralysis and fissured (scrotal) tongue.

Malignant Tumors of the Oral Soft Tissues

Epithelial Tumors

Malignant tumors of the oral epithelium, such as squamous-cell carcinoma are rare in children. Malignant salivary neoplasms are also uncommon, although mucoepidermoid carcinomas haven reported in young patients.

Lymphomas

Hodgkin's and non-Hodgkin's lymphomas have been reported in children; however, they are relatively rare in the pediatric age group. An exception is Burkitt's lymphoma, which is endemic in parts of Africa and occurs in those under 14 years of age. Indeed, in these areas the condition accounts for almost half of all malignancy in children. Burkitt's lymphoma is multifocal, but a jaw tumor (more often in the maxilla) is often the presenting symptom. Burkitt's lymphoma is strongly linked to the Epstein-Barr virus as a casual agent.

Rhabdomyosarcoma

These malignant tumors of skeletal muscle are present in patients around 9 to 12 years of age. The usual site is the tongue. Metastases are common and the prognosis is poor.

LESIONS OF THE JAWS

These can be divided into: cysts, developmental conditions, osteodystrophies and tumors.

Cysts (Table 25.1)

Primordial Cyst

Epithelial lined jaw cysts appearing as radiolucency in the alveolar process unassociated with a tooth; depending on their lining some are difficult to remove; complete surgical excision will cure these cysts.

Table 25.1: Types of cysts based on their epithelial source

Epithelial source	*Types of cyst*
Odontogenic	• Apical cyst • Dentigerous cyst • Primordial cyst • Odontogenic keratocyst • Residual cysts
Non-odontogenic (Fissural)	• Nasopalatine duct cyst • Median palatal cyst • Globulomaxillary cyst • Nasolabial cyst

If tooth development ceases early, a primordial cyst may form instead. Some believe that primordial cysts arise from the dental lamina, an early developmental structure, forming a cyst instead of a tooth. Others believe that they arise from degeneration of the stellate reticulum during the cap or bell stages stopping tooth development and forming a cyst instead. However they form, these uncommon cysts are not associated with a nearby tooth

because they developed instead of one. Because primordial cysts develop instead of a tooth, often the associated tooth is missing. In case where all teeth are accounted for, it is assumed that an extra tooth (supernumerary) would have developed it the cyst hadn't developed instead. The third molar region is the most common location for primordial cysts.

Primordial cysts appear as radiolucencies in tooth-bearing jaw areas. Unless large enough to produce cortical plate expansion or displacement of adjacent teeth, a primordial cyst is invisible to intraoral clinical examination. On radiographs, this cyst appears as a well-demarcated unilocular radiolucency located within the alveolar process near the crest of ridge. Sometimes they may appear below the roots of a tooth or between the roots of adjacent teeth.

If lined with keratinizing epithelium, primordial cysts may be difficult to remove; if completely removed, primordial cysts can be cured. Complete removal of primordial cysts can be expected with osteotomy and curettage. However, if lining keratinization is present, complete removal may be more difficult. As will be discussed next, there are clues that can be observed during surgery suggesting the presence of keratinization and the likelihood of recurrence.

Eruption Cyst (Fig. 25.2)

Eruption cysts are really dentigerous cysts that present as swellings of the alveolar mucosa. They may precede the eruption of both primary and permanent teeth. When filled with blood they are often called eruption hematomas.

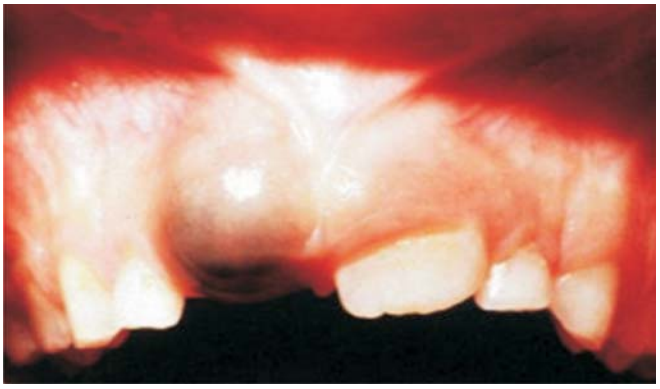

Fig. 25.2: Eruption cyst associated with the erupting permanent right maxillary central incisor

Occasionally, a tooth with a dentigerous cyst surrounding its crown erupts into the mouth. As the surrounding cyst collides with the oral mucosa a bluish dome-shaped lesions result. This lesion is known as an "eruption cyst". When discovered, a radiograph will reveal an unerupted tooth with a radiolucent lesion surrounding the crown. While removal of the lesion is standard procedure, it is necessary to submit the removed tissue for microscopic examination. Most of the lesions will be dentigerous cysts; however, more serious lesions may be encountered occasionally.

Dentigerous Cyst

This is the commonest jaw cyst in children. Dentigerous cysts are common lesions developing around the crown of an unerupted tooth. It is estimated that about 10 percent of impacted teeth form dentigerous cysts. These lesions are presumed to arise from either the reduced enamel epithelium (formed from consolidation of the outer and inner enamel epithelia after enamel deposition is complete) or from rests of adontogenic epithelium not incorporated into the reduced enamel epithelium.

Unless dentigerous cysts are very large, their presence cannot be detected on intraoral clinical examination. They are unaccompanied by any signs or symptoms. Dentigerous cysts are detected by observation of a well-demarcated radiolucency around the crown of an unerupted tooth. The lesions are usually relatively small (less than 2.0 cm in diameter) and are usually unilocular. If not detected early, dentigerous cysts can become large and multilocular, features that suggest their transformation into more serious lesions.

The most serious complications associated with dentigerous cysts are their associated with one of two more serious lesions, the odontogenic keratocyst or the ameloblastoma. The potential for these transformations justify preventive removal of impacted third molar teeth. As the incidence of these unfavorable transformations rise, with age and size of dentigerous cysts, ealy recognition and removal of this lesion is essential.

Small unilocular lesions are managed successfully through osteotomy, extraction of the impacted tooth, and removal of the lesion. Impacted cuspids may be salvaged

by removal of the lesion only. Large lesions may require extensive surgery including jaw resection. With complete removal and confirmed absence of transformation, dentigerous cysts should not recur.

Radicular Cyst

These cysts related to the apex of a non-vital tooth do occur in children, although they are rare in the primary dentition. They are often symptomless and are discovered radiographically. Lateral periodontal cysts are very rare in children. Apical/radicular cysts are caused by inflammatory activation of epithelial rests. They are very common lesions associated with chronic periapical inflammation, presumably, activation of epithelial rests of Malassez.

Unless the lesion is very large or there is suppuration (suppurative apical periodontitis), apical cysts are invisible on intraoral clinical examination. The lesion is usually not associated with signs or symptoms (asymptomatic); however, patients may report past drainage and/or pain in the area. The offending tooth will have a deep carious lesion, deep restoration, or inadequate root canal filling. It will not be responsive to standard pulp testing procedures (i.e. is non-vital). Radiographic examination will reveal a well-demarcated unilocular radiolucency at the tooth apex.

They are cured with surgical excision and removal of cause, they are not dangerous lesions. They do not become malignant or develop into ameloblastoma. If left to grow, radicular cysts can become fairly large; some may reach 4-5 cm in diameter. Apical cysts are treated by osteotomy and curettage. Complete removal should be expected; as a consequence, they should not recur. However, it should be kept in mind that the cause of this lesion, the non-vital tooth, must be dealt with by adequate endodontic therapy or extraction.

Odontogenic Keratocysts

A jaw cyst of dentigerous or primordial origin lined with keratinizing epithelium appearing as radiolucency around the crown of an unerupted tooth or in a tooth-bearing area unassociated with a tooth; this cyst may be difficult to remove surgically and may recur.

The Odontogenic Keratocyst is the most aggressive of the jaw cysts. It has a high rate of recurrence due to the fact that remnants left after subtotal removal will regenerate. These cysts may be found in children and may be associated with the Gorlin-Goltz syndrome. Keratocysts associated with this syndrome appear in the first decade of life, whereas the syndromic basal-cell carcinomas are rare before puberty. Other signs and symptoms include: multiple basal-cell carcinomas, bifid ribs, calcification of the falx cerebri, hypertelorism and frontal and temporal bossing.

If large, keratocysts may produce jaw swelling. Like other cysts, small odontogenic keratocysts cannot be detected on intraoral clinical examination. However, these cysts can become larger in a shorter period of time than nonkeratinizing cysts causing jaw expansion. Most adontogenic karatocysts arise in dentigerous cysts. Because most dentigerous cysts are located in the mandibular third molar region, it stands to reason that most odontogenic keratocysts are located there too.

Their radiolucencies may be small and unilocular or large and multilocular. Odontogenic keratocysts produce a radiolucency located in one of two general areas: around the crown of an unerupted tooth or in the alveolar process unassociated with a tooth. The radiolucency in either site may be small, well-demarcated, and unilocular; however, by the time all too many are discovered, many have become large, multilocular radiolucencies. Some replace much of the jaw. The large, multilocular lesions produce a "soap-bubble" appearance mimicking the radiographic of a serious jaw neoplasm, the ameloblastoma.

Keratocysts are difficult to remove: this is why they recur. It should be obvious that the most common and significant complication of odontogenic keratocysts is incomplete removal. It is the unique features of the cyst lining that hinders surgeons in removal of these lesions. So, imcomplete removal causes the high recurrence rates that are associated with odontogenic keratocysts. It is estimated that almost 30 percent of these cysts recur.

If they can be removed completely, keratocysts can be cured. Osteotomy and curettage will completely remove many odontogenic keratocysts. Given that recurrence rates vary from 10-30 percent, standard therapy will cure 70-90 percent of them. The larger the

lesion becomes, the more difficult it is to completely remove. As a consequence, early diagnosis will insure the highest cure rates. The largest odontogenic keartocysts are occasionally treated by marsupialization.

Non-Odontogenic Fissural Cysts (Table 25.2)

Fissural cysts are not related to teeth but to fusions of upper jaw bones due to the epithelium trapped during development of the oral cavity and face. The occurrence of most is rare. Like the odontogenic cysts, the fissural cysts are lined with epithelium supported by a fibrous connective tissue wall. The maxilla is mentioned preferentially because fissural cysts do not arise in the mandible as the mandible is not formed by the fusion of separate structures. The maxilla is the jaw in which fissural cysts arise. The maxilla arises from several distinct processes, each of which is covered with epithelium. Fissural cysts occur where the developmental processes fuse: the midline, between the premaxilla and maxilla, and the primitive connection between the oral and nasal cavities. In addition to these, there are two rare soft tissue cysts of fissural origin.

Table 25.2: Locations and names of non-odontogenic cysts

Location	*Name of the Non-odontogenic cyst*
Nasopalatine duct cyst	Papilla Palatini Cyst Nasopalatine Duct Cyst
Midline of hard palate	Median palatal cyst
Between premaxilla and maxilla	Globulomaxillary cyst
Along side nose	Nasolabial cyst

Nasopalatine Duct Cysts

The cyst of the papilla palatini and incisive canal are Nasopalatine duct cysts. In early embryonic life just after the oral and nasal cavities are separated, an epithelial tube connects them for a time. Later on, this "nasopalatine duct" disappears; however, remnants of it may persist in the anterior maxilla. A well-known foramen marks the site: the incisive canal. Epithelial rests are located within the incisive canal. There are two manifestations of nasopalatine duct cysts: cysts of the papilla palatini and incisive canal cysts.

Cyst of the Papilla Palatini

A rare true soft tissue cyst appearing as a swelling in the maxilla midline just lingual to the central incisor teeth (the incisive papilla); surgical removal will cure it.

Activation of epithelial rests in the incisive papilla may produce a cyst there. Once in a great while, epithelial rests derived from the nasopalatine duct proliferate forming a cyst within the papilla confined to the palatal mucosa. This is a soft tissue cyst known as the "papilla palatini cyst" (the "papilla palatini" is another name for the "incisive papilla"). This cyst causes a soft-tissue swelling; it is not visible on radiographs.

This cyst causes a swelling of the incisive papilla that is asymptomatic unless it becomes infected secondarily. Being confined to soft tissue, the papilla palatini cyst does not create a radiographic abnormality and, therefore, cannot be detected on radiographs. Excision of the lesion will cure it. These cysts do not become large or transform into anything more serious.

Nasopalatine Duct Cyst (Incisive Canal Cyst) (Fig. 25.3)

A common true jaw cyst appearing as a radiolucency in the maxilla midline just lingual to the central incisor teeth (in the incisive canal); surgical removal will cure this cyst.

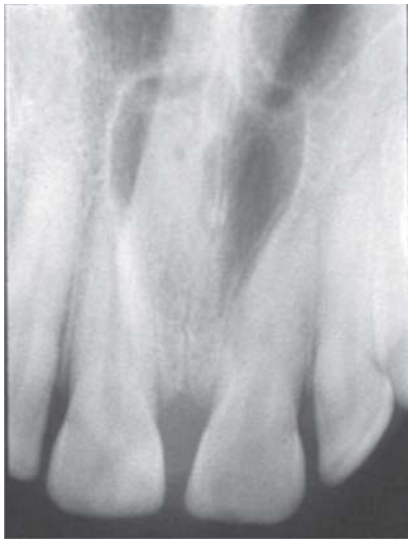

Fig. 25.3: Radiograph showing the presence of a nasopalatine duct cyst

Activation of incisive canal rests may produce the most common fissural cyst there. The incisive canal arises from epithelial rests located within the tissues of the incisive canal. It is the most common fissural cyst and may affect 1 percent of the population.

Incisive canal cysts cannot usually be detected on clinical examination. Unless large, an incisive canal cyst is undetected by intraoral clinical examination. On rare occasions the cyst may become secondarily infected producing a fistula and suppurative drainage through the incisive papilla.

Incisive canal cysts appear as radiolucencies behind the maxillary incisor teeth. Many incisive canal cysts appear as heart-shaped radiolucency is located just posterior to the maxillary incisor teeth. The radiolucency is usually small (<1.0 cm); however, it may attain large size (> 2.0 cm). The nearby incisors cause a diagnostic challenge: as the lesion may appear, in anterior films, to lie above the incisor root simulating a periapical lesion. The absence of restorations or caries and the responsiveness to pulp vitality tests will rule out the diagnosis of a periapical lesion. These simple observations and tests will eliminate unnecessary endodontic therapy. The true size and shape of incisive canal radiolucencies can be determined by using occlusal films and orienting the X-ray beam at right angles to the hard palate. Incisive canal cysts are nonkeratinizing; their connective tissue walls are unique.

Surgical excision will cure incisive canal cysts. Simple excision of incisive canal cysts will be sufficient to prevent their recurrence. These lesions have no neoplastic potential nor do their linings keratinize.

Median Palatal Cyst

An uncommon true jaw cyst appearing as a radiolucency in the maxilla midline posterior to the incisive canal; surgical removal will cure this cyst.

Activation of trapped rests in the palatal midline may cause a medial palatal cyst. If epithelial rests located in the mid-palatal maxillary suture should proliferate, a median palatal cyst may form. This is uncommon but should be kept in mind if a mid-palatal swelling is encountered. Small lesions are usually invisible on clinical intraoral examination. However, these cysts do not have to become very large to raise the palatal mucosa causing an intraoral swelling. If this should occur, the absence of a diseased tooth as well as redness, pain, fever, and other signs of acute inflammation will convince the clinician that the swelling is not a palatal abscess.

Median palatal cysts appear as radiolucencies in the midline of the hard palate. The median palatal cysts produce radiolucencies in the midline of the palate well posterior to the incisive canal. While they are small in their early stages, being asymptomatic, they are difficult to see on standard radiographs, and may become quite large before detected. Like the incisive canal cysts, the radiolucencies of median palatal cysts are best seen on occlusal films. Median palatal cysts are nonkeratinizing; surgical excision will cure them.

Globulomaxillary Cyst (Fig. 25.4)

An uncommon true jaw cyst appearing as a radiolucency between the roots of vital maxillary lateral and cuspid (canine) teeth; surgical removal will cure this cyst.

It is postulated that epithelial rests may exist in the junction of the embryonic median nasal and maxillary processes. It is further postulated that this junction forms the suture between the premaxilla and maxilla and that epithelial rests may occur in these sutures. Because these cysts are supposed to arise from the globular portion of the median nasal process and maxillary process, they are called "Globulomaxillary cysts". If stimulated, these

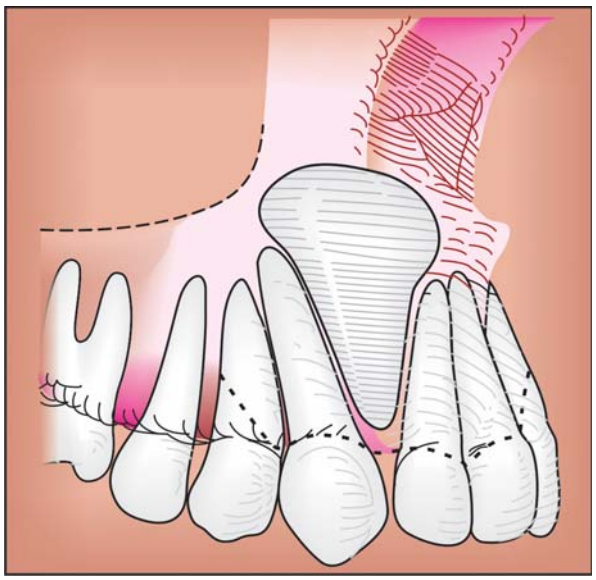

Fig. 25.4: Diagrammatic representation of the location of globulomaxillary cyst

rests may give rise to a cyst that produces radiolucency between the lateral incisor and cuspids.

Globulomaxillary cysts are usually asymptomatic and therefore are undetected on routine clinical intraoral examinations. Their presence is usually observed by discovery of radiolucency between the lateral incisor and cuspid teeth on periapical radiographs. The radiolucent lesion is usually shaped like an upside-down pear spreading adjacent roots.

Microscopic examination reveals a cyst that is lined with stratified squamous nonkeratinizing epithelium. There is no potential for this lesion to keratinize or to show neoplastic transformation. Simple excision will cure the lesion and prevent its recurrence. As already mentioned, the most significant problem globulomaxillary cysts pose is differentiating them from periapical lesions associated with a non-vital lateral incisor or cuspid tooth.

Nasolabial Cyst (Nasoalveolar Cyst)

A rare true soft tissue cyst appearing as a facial swelling of the upper lip and the side of the nose; surgical removal will cure this cyst.

On rare occasions, a cyst will develop from proliferation of epithelium enclaved during development of the middle face and/or the tear (lacrimal) duct. The resulting cyst produces a characteristic swelling of the upper lip that extends upward obliterating the fold between the side of the nose and the cheek. This typical pattern of swelling has earned the lesion the designation "Nasolabial cyst".

The lesion is completely asymptomatic; there is no pain, redness, fever, or other signs of acute inflammation, ruling out, or course, swelling produced by an abscess. Because the lesion is confined solely to the soft tissues, it is not detected by routine radiographic examination. If suspected, however, contrast material may be injected into the lesion producing a radiopaque image that will provide information about its size and extent.

Nasolabial cysts are lined with stratified squamous nonkeratinizing epithelium; and have no patential to undergo neoplastic transformation. They are excised through an incision made in the labial vestibule (preventing a facial scar). Complete excision will cure these lesions and prevent their recurrence.

Hemorrhagic bone cyst

The hemorrhagic bone cyst is a condition that may be found in children and adolescents. It occurs most commonly in the mandible in the premolar/molar region. It is often a chance radiographic finding and normally asymptomatic. Radiographically it appears as a scalloped radiolucency between the roots of the teeth. It regresses spontaneously or after surgical investigation.

Developmental Conditions

Numerous developmental conditions may affect the oral and perioral structures. These range from minor problems (e.g. Tongue-tie) that are readily treated under local anesthesia, to severe craniofacial disorders (e.g. Crouzon's syndrome) requiring a combined interdisciplinary approach between maxillofacial and neurosurgery. Readers should refer to specialized texts for a full description of congenital law abnormalities. It is important to remember that patients with developmental Orofacial abnormalities may have other congenital disorders, such as cardiac defects, which may influence routine dental treatment.

Osteodystrophies

Fibrous dysplasia: This can occur as one of three variants namely: monostotic, polyostotic or as part of Albright's syndrome (where associated conditions include skin pigmentation and precocious puberty in females). The monostotic type is the most common to affect the jaws, especially the maxilla. The disease presents as a slow-growing bony expansion that produces facial asymmetry and misalignment of teeth. Radiographically there is a fine granular radiopacity.

Cherubism: In this rare condition there is a characteristic fullness of the cheeks and jaws. Initial presentation is commonly between 2 and 4 years of age. Size increases during growth. It is self-limiting and regression occurs in adulthood. Cosmetic surgery may be employed after active growth has finished. Multilocular radiographic radiolucencies occur at the angles of the mandible and the maxillary tuberosity. Histologically the lesion is similar to the giant-cell granuloma.

Tumors of the Jaws

Odontome

Odontomes are hamartomas that contain dental calcified tissue. They are classified as compound (a collection of discrete tooth-like structures) and complex (a haphazard arrangement of dental tissue). Compound odontomes are most commonly found in the anterior maxilla. The complex type is usually located in the premolar/molar regions of both jaws. Odontome are usually symptomless and are diagnosed radiographically. The mean age of patients at diagnosis is 15 years. Occasionally an odontome will become inflected when partially erupted and surgical excision is required. Similarly, removal is indicated if an odontome is interfering with the eruption of a neighboring tooth or is needed as part of an orthodontic treatment plan.

Juvenile Ossifying Fibroma

This benign lesion differs from the adult ossifying (or cemento-ossifying) fibroma in that growth is rapid. It consists of fibrous tissue with a varying amount of mineralized material. It usually affects the mandible. Radiographs show a well-circumscribed radiolucency with 'speckling'. Surgical excision is required.

Central Giant-Cell Granuloma

This swelling of bone usually affects the mandible. Radiographically there is a well-defined radiolucency with occasional resorption of associated teeth. Histologically there are large numbers of osteoclast-like cells in a vascular stroma. Surgical curettage is curative.

Histiocytosis

Langerhans' cell Histiocytosis, formerly known as Histiocytosis X is a condition that predominantly affects children. Bone is replaced by Langerhans' cells so producing sharply defined radiographic radiolucencies.

Ameloblastoma

Although more commonly found in adults, this locally invasive neoplasm can occur in children. It is usually found in the mandible. It is slow growing, and is often symptomless in the early stages. As it progresses it causes a bony swelling, which appears as a Multilocular radiolucency in the jaw. Surgical resection to sound bone is necessary for a cure.

Ameloblastic Fibroma

This rare lesion usually affects a younger age group than the ameloblastoma. The average age of patients at diagnosis is 14 years. It is a benign tumor. A related lesion is the Amelobastic fibro-odontoma. This lesion contains dentine and enamel and occurs in children under 10 years of age.

Primary Intraosseous Carcinoma

This is a very rare tumor but when it occurs it is usually in children. It is thought to arise from Odontogenic epithelium and shows rapid growth.

Sarcomas

Sarcomas of the jaws are rare; however, the highly malignant Ewing's sarcoma occurs in children between the ages of 5 and 15 years of age. The mandible is usually the bone affected and the prognosis is poor.

ORAL MANIFESTATIONS OF SYSTEMIC DISEASE

In addition to specific pathological oral conditions, diseases that affect other systems of the body can produce oral manifestations, for example, Crohn's disease. In addition, disorders such as chronic renal failure and diabetes can predispose to periodontal disease and there may be poor resistance to the spread of odontogenic infection.

It is not only the oral soft tissues that are affected by systemic conditions. The temporomandibular joint can be involved in juvenile rheumatoid arthritis and the jaws can be affected in hyperparathyroidism (giant-cell tumors). In some cases an oral condition may be the presenting feature of a systemic disease and dental practitioners should not hesitate to refer children with abnormal oral signs for further investigation.

26

Child Abuse and Neglect

INTRODUCTION

Child abuse is a phenomenon, which as described by S.J.Breiner, an American scientist has been present in our world since times when people first began to record their history.

Child abuse is defined as the non-accidental physical injury, minimal or fatal, inflicted upon children by persons caring for them (Selwyn et al 1985).

Child abuse consists of any act, or failure to act, that endangers a child's physical or emotional health and development.

Neglect

According to child welfare information Gateway more children suffer from neglect than from physical and sexual abuse combined.

Neglect is a very common type of child abuse, yet victims are not identified, primarily because neglect is a type of child abuse that is an act of omission.

Neglect is a pattern of failure to provide a child's basic needs. A single act of neglect might not be considered child abuse, but repeated neglect is definitely child abuse.

Historical Overview

Child abuse is considered as a problem as old as mankind with reports of

- Infanticide in Greek and Roman culture and in other societies.
- 1628 Massachusetts-**"The Stubborn Child Act"** with death penalty for a disobedient child.
- 1874 "**Mary Ellen"** case victim of child abuse and neglect who was subjected to physical abuse and she was removed from an abusive home and brought to court under laws governing cruelty to animals.
- 1875-First society for prevention of cruelty to children in New York City.
- 1946-Caffey presented the X-ray findings of child abuse.
- 1960-Henry Kempe reported **"Battered child syndrome".**

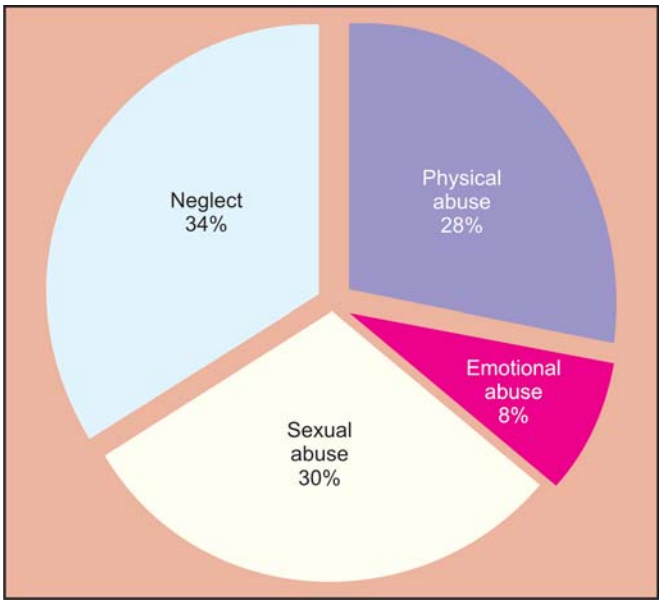

Fig. 26.1: Graphical representation of types of child abuse

TYPES OF CHILD ABUSE (FIG. 26.1)

There are four major types of child abuse:

i. Physical abuse
ii. Sexual abuse
iii. Emotional abuse
iv. Neglect

Physical Abuse

- Any non-accidental injury or trauma to the body of a child by a parent, guardian, on sibling or a stranger.
- It is otherwise termed as physical violence against a child resulting from physical aggression.
- The injury from physical abuse may be the result of:
  - Beating, slapping or hitting
  - Pushing, shaking, kicking or throwing
  - Pinching, biting, chocking or hair-pulling
  - Burning with cigarettes, scalding water, or other hot objects.
  - Severe physical punishment.

Is Physical Punishment the Same as Physical Abuse?

Physical abuse is an injury resulting from physical aggression. Physical punishment is the use of physical

force with the intent of inflicting body pain, but not injury, for the purpose of correction or control. But at times physical punishment can easily get out of control and become physical abuse.

Types of Physical Child Abuse

Shaken Baby Syndrome (SBS)

- Shaking a baby to try to stop inconsolable crying.

Signs and symptoms: There are various signs and symptoms of shaken syndrome. The consequences of less severe cases may not be bought to the attention of medical professionals and may never be diagnosed.

Common symptoms are:

- Lethargy/decreased muscle tone
- Extreme irritability
- Decreased appetite, poor feeding or vomiting for no apparent reason
- Grab type bruises on arms or chest are rare
- No smiling or vocalization
- Poor sucking or swallowing
- Difficulty in breathing
- Seizures.
- Head or forehead appears larger
- Inability to life head
- Inability to eyes to focus.

Physical consequences:

- The brain rotates within the skull cavity, injuring or destroying brain tissue.
- When shaking occurs, blood vessels feeding the brain can be torn, leading to bleeding around the brain.
- Retinal bleeding is very common.
- Leading to long term consequences like:
  - Learning, physical, visual, speech, hearing disabilities.
  - Cerebral palsy
  - Seizures
  - Behavior disorders
  - Cognitive impairments
  - Even death immediately/delayed.

Battered Child Syndrome

A type of physical abuse reported by Henry Kempe in 1960 characterized by;

- Multiple asymmetrical fractures at different stages of healing.
- Subdural hematoma
- Bruise marks shaped like hands, fingers or objects, or unexplained bruises in areas where normal childhood activities would not usually result in bruising
- Separated sutures
- Bulging fontanelles
- Evidence of unexplained abdominal injury
- At times even unexplained unconsciousness in infant.
- Evidence of fractures at the tip of long bones on spiral type fractures that result from twisting
- Fractured ribs, especially in the back.

Investigations:

- Radiographs, MRI or CT-Scan.

Medical conditions that mimic abuse:

- Osteogenesis imperfecta
- Hemophilia
- Von Willebrand's disease.

Munchausen Syndrome by Proxy

- Also known as factitious disorder by proxy/pediatric condition falsification.
- It is a condition in which a care giver, usually the mother, feigns or induces an illness in another person, usually her or his child, to gain attention and sympathy as the "worried" parent.
- It was coined by Sir Roy Meadow in 1977.
- Munchausen syndrome was first described by Dr. Richard Asher in 1951 for adults who fabricated symptoms about themselves and produced signs of illnesses.
- The most clinically useful definition is provided by Bools et al (1992).

"Illness in a child which is fabricated by a parent, or someone in loco parentis. The child is presented for medical assessment and care, usually persistently, often resulting in multiple medical procedures. The perpetrator denies knowledge of the etiology of the child's illness. Acute symptoms and signs of the illness decrease when the child is separated from the perpetrator".

Etiology:

- Development disturbances, personal history and current life stressors have been implicated.
- Mothers themselves may have suffered from Munchausen syndrome.
- A child may be resented for having good health.
- Illness may be fabricated in order to prevent a child from becoming independent.
- Abusing the child to escape marital discord or other life stressors.
- Pleasure is possibly derived from manipulating as many aspects of medicine.
- A childhood history of emotional insecurity, parents that are excluding, rejecting or abusive.
- An underlying personality disorder.

Signs that may indicate Munchausen syndrome by proxy:

- A child who has one or more medical problems that do not respond to treatment or that follow an unusual course that is persistent, puzzling and unexplained.
- Insignificant physical/laboratory findings that do not correlate with clinical feature.
- A highly attentive parent who is reluctant to leave their child's side and who themselves seems to require constant attention.
- A parent, who appears to be medically knowledgeable and/or fascinated with medical details and hospital gossip, appears to enjoy hospital environment.
- The signs and symptoms of a child's illness do not occur in the parent's absence.
- A family history of similar or unexplained illness or death in a sibling, etc.
- This kind of abuse is very difficult to diagnose and needs extensive medical evaluation, testing and detailed interrogation with child and parent separately.

Sudden Infant Death Syndrome (SIDS)

It is any sudden and unexplained death of an apparently healthy infant aged one-month to one year. The term cot death is often used in United Kingdom, and crib death in North America.

SIDS can be due to any reason but child abuse can be a reason.

Sexual Abuse

- Any sexual behavior or activity with a minor or the exploitation of a child/minor, by an adult, for the sexual pleasure of someone else. This includes:
  - Fondling
  - Violations of bodily privacy
  - Child pornography
  - Exposing children to pornography
  - Luring a child for sexual liaisons
  - Sexual exploitation
  - Exhibitionism
  - Sexual intercourse with a child
  - Trauma to mouth because of oral sex.

Emotional Abuse

Emotional child abuse is another person's attitude, behavior to failure to act that interfere with a child's mental health or social development.

Emotional abuse has more long lasting negative psychiatric effects than either physical or sexual abuse.

Emotional abuse can range from a simple verbal insult to an extreme form of punishment

- Ignoring, withdrawal of attention, rejection
- Lack of physical affection
- Lack of positive re-inforcement
- Threats
- Habitual blaming
- Degradation, etc.

Neglect

Types of Neglect

Physical neglect: Is not providing for a child's physical needs, which are:

- Food
- Clothing appropriate for weather
- Supervision
- A home that is hygienic and safe.

Educational neglect:

- Failure to provide proper education rightful to the age of the child.

Emotional/psychological neglect:

- Not providing emotional support and love which is affection, attending to the child's emotional needs, psychological care as needed.

Medical neglect:

- Failing to provide needed basic medical attention to child's health care.
- It also includes "Dental neglect":
  - Dental caries, periodontal diseases and other oral conditions, if left untreated, can lead to pain, infection and loss of function. These undesirable outcomes can adversely affect learning, communication, nutrition and other activities necessary for normal growth and development.
  - Dental neglect is willful failure of parent or guardian to seek and follow through with treatment necessary to ensure a level of oral health essential for adequate function and freedom from pain and infection.

Child Abuse and the Handicapped Child

Handicapped children are at risk for abuse because they may be:

- Less able to defend themselves physically
- Less able to articulate the fact of abuse
- Unable to differentiate between appropriate and inappropriate physical contact, whether it be violent or sexual
- More dependants on others for assistances or care and therefore, more trusting, since dependency and trust often translate into compliance and passivity
- Reluctant to report instances of abuse for fear of losing vital linkage to major care providers.

Factors for Child Abuse (Fig. 26.2)

Zirpoli (1986) asserts that there are four primary factors which contribute to child abuse:

- Parent factors
- Sociocultural factors
- Environmental factors
- Child factors.

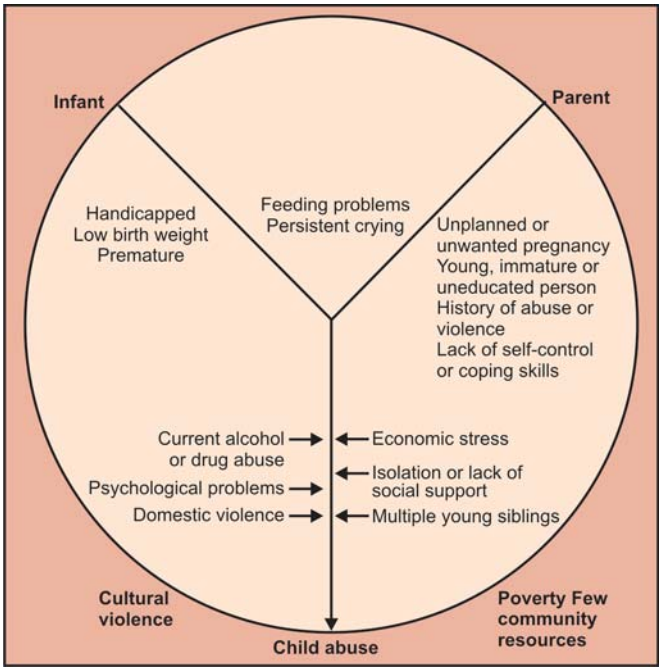

Fig. 26.2: Path to child abuse

INDICATORS OF CHILD ABUSE

Indicators are the signs, symptoms or clues which may mean that a child has been abused or may be at risk for abuse.

- Indicators do not prove that a child has been abuse.
- Indicators may be seen in the child's physical health or behavior, as well as those of the child's caregivers.
- Adults who abuse children may show certain behaviors and attitudes that make other people worry if they should be caring for children.

THE DENTIST'S ROLE IN CHILD ABUSE AND NEGLECT INTERVATION

(As stated by the American Dental Association)

In order to properly comply with the legal mandate for all health professionals to report suspected cases of child maltreatment, dentists must be cognizant of their responsibilities as outlined by the American Dental Association. These responsibilities include:

- To observe and examine any suspicious evidence that can be ascertained in the dental clinic.

Possible Indicators of Neglect		
Physical Indicators in Children	**Behavioral Indicators in Children**	**Behaviors Observed in Adults who Neglect Children**
• An infant or young child may: – Not be growing as expected. – Be losing weight – Have a "wrinkly old face". – Look pale – Not be eating well • Not dressed properly for the weather • Dirty or unwashed • Bad diaper rash or other skin problems • Always hungry • Lack of medical and/or dental care • Signs of deprivation which improve with a more nurturing environment (e.g. hunger, diaper rash)	• Does not show skills as expected • Appears to have little energy • Cries very little • Does not play with toys or notice people • Does not seem to care for anyone in particular • May be very demanding of affection or attention from others • Older children may steal food, drink alcohol or take drugs, break the law • Takes care of a lot of their needs on their own • Has a lot of adult responsibility at home • Discloses neglect (e.g. says there is no one at home)	• Does not provide for the child's basic needs • Has a disorganized home life, with few regular routines (e.g. always brings the child very early, picks up the child very late). • Does not supervise the child properly (e.g. leaves the child alone, in a dangerous place, or with someone who cannot look after the child safely) • May indicate that the child is hard to care for, hard to feed, describes the child as demanding • May say that the child was or is unwanted • May ignore the child who is trying to be loving • Has difficulty dealing with personal problems and needs • Is more concerned with own self than the child • Is not very interested in the child's life (e.g. fails to use services offered or to keep child's appointments, does not do anything about concerns that are discussed)

Possible Indicators of Physical Abuse (Fig. 26.3)		
Physical Indicators in Children	**Behavioral Indicators in Children**	**Behaviors Observed in Adults who Neglect Children**
• See Figure 26.5 for questionable injuries • Bruises in the same area of the body • Bruises in the space of an object (e.g. spoon, hand/ fingerprints, belt) • Burns – From a cigarette – In a pattern that looks like an object (for example, iron) • Wears clothes to cover up in warm weather • Patches of hair missing • Signs of possible head injury: – Swelling and pain – Nausea or vomiting – Feeling dizzy – Bleeding from the scalp or nose. • Signs of possible injury to arms and legs: – Pain – Sensitive to touch – Cannot move properly – Limping • Breathing causes pain • Difficulty in raising arms • Human bite marks • Cuts and scrapes inconsistent with normal play • Signs of female genital mutilation (e.g. trouble going to the bathroom)	• Cannot remember how injuries happened • The story of what happened does not match the injury • Refuses or is afraid to talk about injuries • Is afraid of adults or of a particular person • Does not want to be touched • May be very: – Aggressive – Unhappy – Withdrawn – Obedient and wanting to please – Uncooperative • Is afraid to go home • Runs away • Is away a lot and when comes back there are signs of a healing injury • Does not show skills as expected • Does not get along well with other children • Tries to hurt him/herself (e.g. cutting oneself, suicide) • Discloses abuse	• Does not tell the same story as the child about how the injury happened • May say that the child seems to have a lot of accidents • Severely punishes the child • Cannot control anger and frustration • Expects too much from the child • Talks about the child as being bad, different or "the cause of my problems" • Does not show love toward the child • Does not go to doctor right away to have injury checked • Has little or no help caring for the child

Possible Indicators of Sexual Abuse		
Physical Indicators in Children	**Behavioral Indicators in Children**	**Behaviors Observed in Adults Who Neglect Children**
• A lot of itching or pain in the throat, genital or anal area • A smell or discharge from the genital area • Underwear that is bloody – Trying to go the bathroom – Sitting down – Walking – Swallowing • Blood in urine or stool • Injury to the breasts or genital area: – Redness – Bruising – Cuts – Swelling • Pregnancy	• Copying the sexual behavior of adults • Knowing more about sex than expected • Details of sex in the child's drawings / writing • Sexual actions with other children or adults that are inappropriate • Fears or refuses to go to a parent, relative, or friend for no clear reason • Does not trust others • Changes in personality that do not make sense (e.g. happy child becomes withdrawn) • Problems or change in sleep pattern (e.g. nightmares) • Very demanding of affection or attention, or clinging • Goes back to behaving like a young child (e.g. bed wetting, thumb-sucking) • Refuses to be undressed, or when undressing shows fear • Tries to hurt oneself (e.g. uses drugs or alcohol, eating disorder, suicide) • Discloses abuse	• May be very protective of the child • Clings to the child for comfort • Is often alone with the child • May be jealous of the child's relationships with others • Does not like the child to be with friends unless the parent is present • Talk about the child being "sexy" • Touches the child in a sexual way • May use drugs or alcohol to feel free to sexually abuse • Allows or tries to get the child to participate in sexual behavior

Possible Indicators of Emotional Abuse		
Physical Indicators in Children	**Behavioral Indicators in Children**	**Behaviors Observed in Adults who Neglect Children**
• The child does not develop as expected • Often complains of nausea, headaches, stomach aches without any obvious reason • Wets or dirties pants • Is not given food, clothing and care as good as what the other children get • May have unusual appearance (e.g. strange haircuts, dress decorations)	• Is unhappy, stressed out, withdrawn, aggressive or angry for long periods of time • Goes back to behaving like a young child (e.g. toileting, constant rocking) • Tries too hard to be good and to get adults to approve • Tries really hard to get attention • Tries to hurt oneself (e.g. uses drugs or alcohol, suicide) • Criticizes oneself a lot • Does not participate because of fear of failing • May expect too much of him/herself so gets frustrated and fails • Is afraid of what the adult will do if she/he does something the adult does not like • Runs away • Has a lot of adult responsibility • Does not get along well with other children • Discloses abuse	• Often rejects, insults or criticizes the child, even in front of others • Does not touch or speak to the child with love • Talks about the child as being the cause for problems and things not going as wished • Talks about or treats the child as being different from other children and family members • Compares the child to someone who is not liked • Does not pay attention to the child and refuses to help the child • Isolates the child, does not allow the child to see others both inside and outside the family (e.g. locks the child in a closet or room) • Does not provide a good example for children on how to behave with others (e.g. swears all the time, hits others) • Lets the child be involved in activities that break the law • Uses the child to make money (e.g. child pornography) • Lets the child be involved in activities that break the law • Lets the child see sex and violence on TV, videos and in magazines • Terrorizes the child (e.g. threatens to hurt or kill the child or threatens someone or something that is special to the child) • Forces the child to watch someone special being hurt • Asks the child to do more than she/he can do

Possible Indicators of Exposure to Family Violence		
Physical Indicators in Children	**Behavioral Indicators in Children**	**Behaviors Observed in Adults who Neglect Children**
• The child does not develop as expected • Often complains of nausea, headaches, stomach aches without any obvious reason • Physical harm, whether deliberate or accidental, during or after a violent episode, including: – While trying to protect others – The result of objects thrown	• May be aggressive and have temper tantrums • May show withdrawn, depressed, and nervous behaviors (e.g. clinging, whining, a lot of crying) • Acts out what has been seen or heard between the partners • Tries too hard to be good and to get adults to approve • Afraid of: – Someone's anger – One's own anger (e.g., killing the abuser) – Self or other loved ones being hurt or killed – Being left alone and not cared for • Problems sleeping (e.g., cannot fall asleep, afraid of the dark, does not want to go to bed, nightmares) • Bed-wetting • Tries to hurt oneself (e.g,eating disorders, uses drugs or alcohol, suicide) • Stays around the house to keep watch, or tries not spend much time at home • Problems with school (e.g., trouble paying attention, poor marks, misses school a lot) • Expects a lot of oneself and is afraid to fail and so works very hard and gets good marks in school • Takes the job of helping/protecting other family members • Does not get along well with other children • Runs away from home • Cruelty to animals • Older children may steal, hurt others, join a gang or break the law • Child may act out sexually • Child expresses the belief that she/he is responsible for the violence • Discloses family violence	• Abuser has trouble controlling self • Abuser has trouble talking and getting along with others • Abuser uses threats and violence (e.g., threatens to hurt, kill or destroy someone or something that is special; cruel to animals) • Forces the child to watch a parent/ partner being hurt • Abuser is always watching what the partner is doing • Abuser insults, blames and criticizes partner in front of others • Jealous of partner talking or being with others • Abuser does not allow the child or family to talk with or see others • The abused person is not able to care properly for the children because of isolation, depression, trying to survive, or because the abuser does not give enough money • Holds the belief that men have the power and women have to obey • Uses drugs or alcohol • The abused person seems to be frightened • Discloses family violence • Discloses that the abuser assaulted or threw objects at someone holding a child

- To record, per legal and court rules, any evidence that may be helpful in the case, including physical evidence and any comments from questioning or interviews.
- To treat any dental or Orofacial injuries within the treatment expertise of the dentist, referring more extensive treatment needs to a hospital or dental/ medical specialist.
- To establish/maintain a professional therapeutic relationship with the family.
- To become familiar with the perioral signs of child abuse and neglect and to report suspected cases to the proper authorities consistent with state low.

Identification (Fig. 26.4)

For the dental professional to be able to identify the signs of maltreatment that a child present with, he or she must be knowledgeable of not only the types of abuse or neglect, mentioned previously, but the various physical and behavioral manifestations that may be exhibited. The ability to properly identify suspicious injuries to the head, face, mouth, and neck of a child is imperative for dentists. The following information outlines the signs and symptoms or the four types of child maltreatment with emphasis placed on the locations on the child where they may occur.

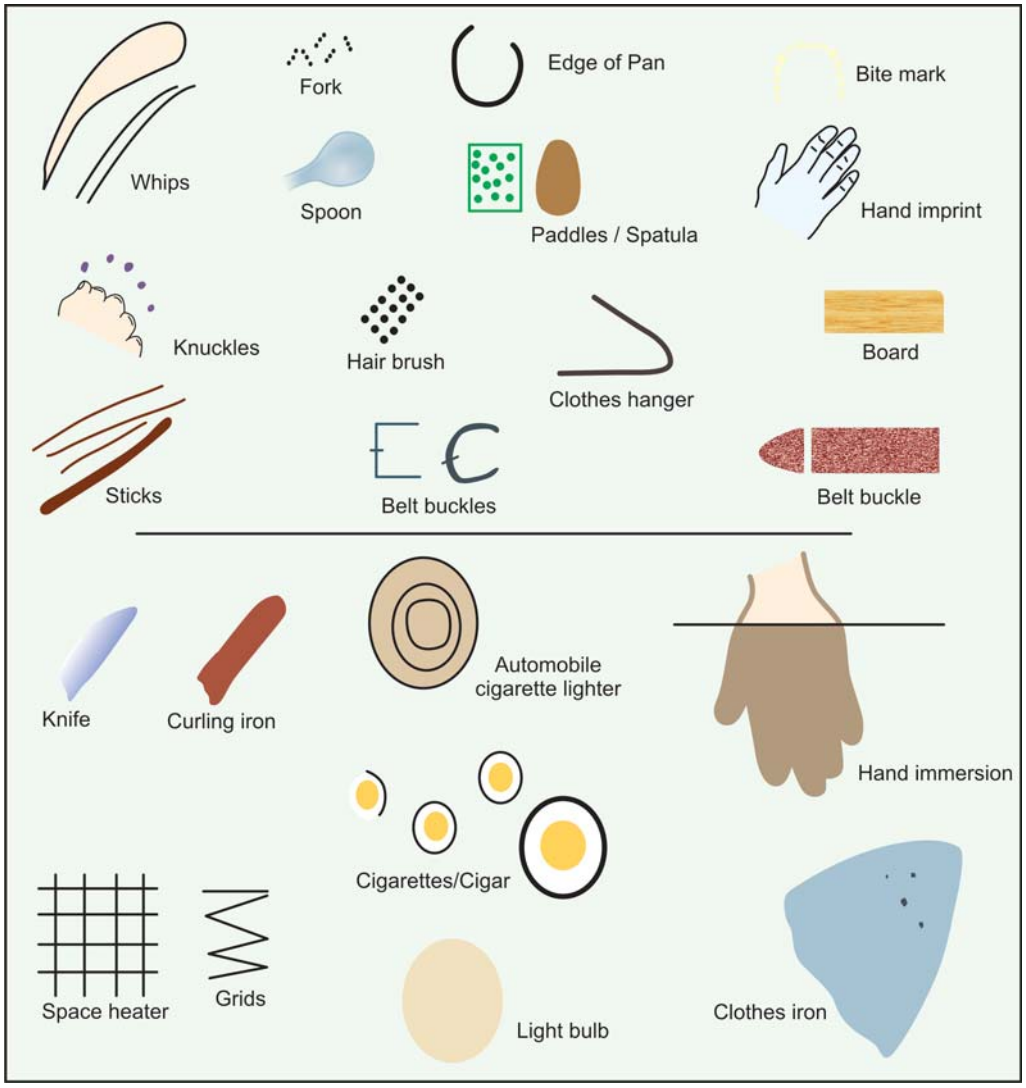

Fig. 26.3: Patterns of abusive marks on an abused child

Physical Abuse (Fig. 26.5)

It may result in numerous types of injuries including contusions, ecchymosis, abrasions, lacerations, fractures, burns, bites, hematomas, retinal hemorrhaging, traumatic, and dental trauma. A list of head and orofacial injuries that dentists should be alert for include common sites for bruises indicators for child abuse):

- Scalp and hair: Subdural hematomas (cause more serious injuries and deaths than any other form of abuse), traumatic alopecia, subgaleal hematomas, and bruises behind the ears.
- Eyes: Retinal hemorrhage, ptosis, and periorbital bruising.
- Ears: Bruising of the auricle and tympanic membrane damage.
- Nose: Nasal fractures or an injury resulting in clotted nostrils.
- Lips: Lacerations, burns, abrasions, or bruising.
- Mouth: Labial or lingual frenum tears (characteristic of more severely abused children), burns of lacerations of the gingiva, tongue, palate, or floor of the mouth.
- Maxilla or mandible Past or present fractures to facial bones, condyles, or symphysis of mandible. Malocclusion may be a result of this type of injury.

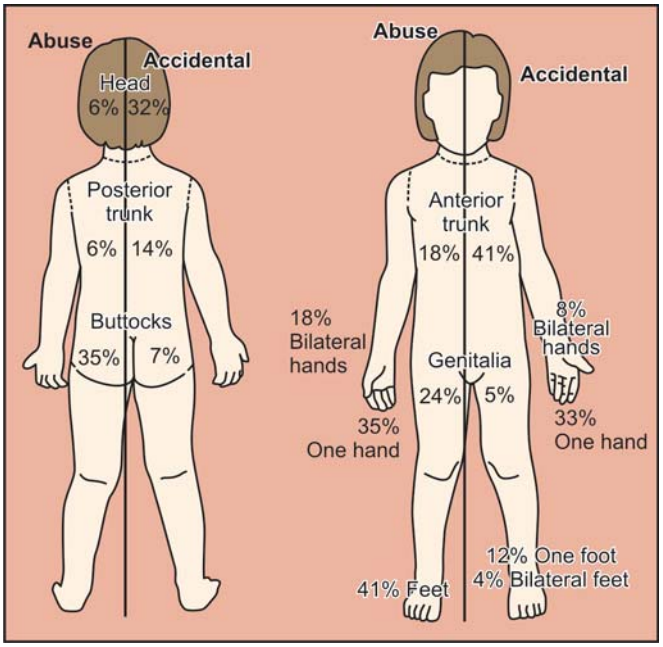

Fig. 26.4: Identifying the patterns of abuse

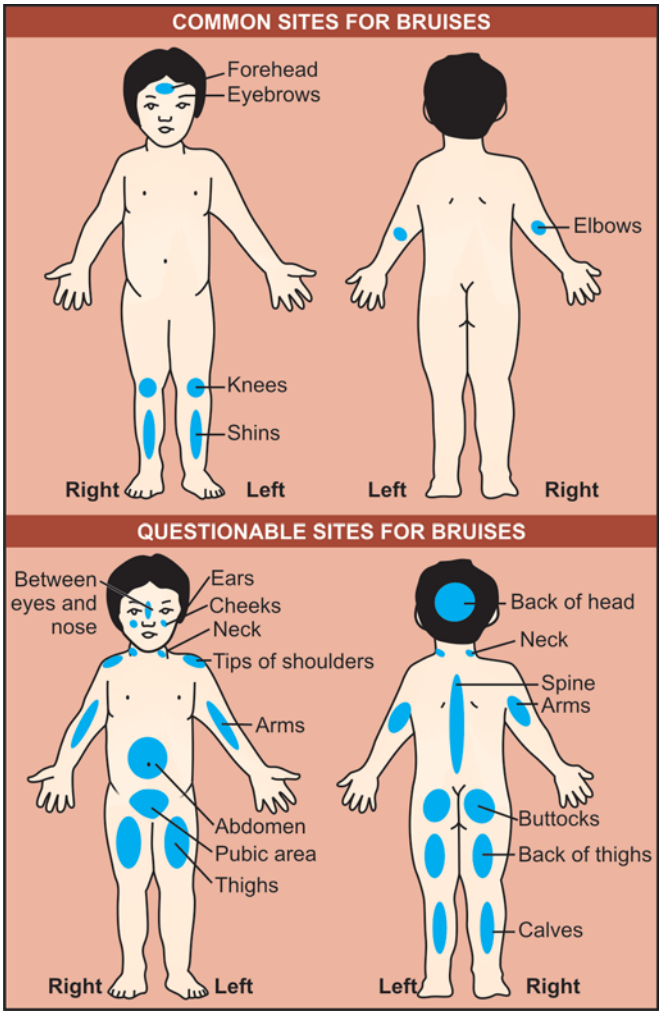

Fig. 26.5: Common sites for bruises—indicators for child abuse

Bite Marks

This type of injury is usually associated with physical or sexual abuse. In such suspected cases, a forensic pathologist or odontologist should be contacted.

Many times misdiagnosed as simple childhood bruises:

- Typically oval or circular configuration
- An area of hemorrhage, representing a "suck" or "thrust" mark, may be found between tooth marks, suggesting physical or sexual abuse
- Although marks may occur anywhere on a child's body, the most common sites are the cheeks, back, sides, arms, buttocks, and genitalia.

Sexual Abuse

While dentists are not as involved as other health professionals in the diagnosis of this type of abuse, they should remain alert for the following signs and symptoms:

Orofacial Manifestations

- **Gonorrhea:** Most commonly sexually transmitted disease in sexually abused children. May appear symptomatically on lips, tongue, palate, face, and especially pharynx ranging from erythema to ulcerations and from vesiculopustular to pseudo membranous lesions.
- **Condylomata acuminata (veneral warts):** Appear as single or multiple raised, pedunculated, cauliflower-like lesions. In addition to the oral cavity, lesions may also be found on the anal or genital areas.
- **Syphilis:** Manifests as a papule on the lip or dermis at the site of inoculation. The papule ulcerates to form the classic chancre in primary syphilis and a maculopapular rash in secondary syphilis.
- **Herpes simplex virus, Type 2(HSV-2):** Herpes simplex virus, type 2 (genital herpes), presents as an oral or perioral painful, reddened area with a grape-like cluster of vesicles (blisters) that rupture to form lesions or sores.

- **Erythema and petechia:** Such trauma at the junction of the hard and soft palate may indicate forced oral sex.

Emotional Abuse

Although difficult to diagnose, a child enduring this form of maltreatment may exhibit the following behavior and physical indicators:

- Lack of self-esteem
- Poor social skills, often antisocial
- Developmentally delayed
- Passive and aggressive-behavioral extremes
- Pronounced nervousness, often manifested in habit disorders such as sucking and rocking. May self-inflict injuries such as lip or cheek biting.

Neglect

Often misunderstood and misdiagnosed. Physical/behavioral indicators include:

General Neglect

Lack of supervision:

- Fatigue or listlessness
- Unattended medical needs
- Poor personal hygiene
- Inappropriate or inadequate clothing

Dental neglect:

- Untreated rampant caries easily detected by a lay person
- Untreated pain, infection, bleeding, or trauma affecting the orofacial region
- History of lack of continuity of care in the presence of identified dental pathology.

Assessment: History Taking and Diagnosis

A key diagnostic feature of abuse or neglect is a discrepancy between the clinical findings and the history given for the problem by the parent or caregiver. Most parents who accompany their injured child to the physician or dentist act in a concerned manner, asking questions regarding the health status of their child. Some parents may even feel an unwarranted guilt that, to some degree, they are responsible for the injury to their child. Abusive adults usually have no questions and may appear withdrawn or unconcerned. Many wait hours or even days before seeking medical or dental attention for their child, even in situations of life-threatening injuries. The following actions outlines what the dentist should do in cases of suspected cases of child maltreatment:

- Before treatment begins, the child should be evaluated for any physical or behavioral signs of maltreatment.
- If abuse is suspected question the child first, away from the parent, about the cause of his or her injury. Seek the same information from the parent's chart with detailed description of the injuries and accounts given for their occurrence.
- The dentist should always document personal opinion why child maltreatment is suspected.
- If a report will be filed with proper authorities, the parent(s) should always be informed.

Prevention (Table 26.1)

Table 26.1: Common features of successful child abuse prevention program

- Strengthen family and community connections and support.
- Treat parents as vital contributors to their children's growth and developement.
- Create opportunities for parents to feel empowered to act on their own behalf.
- Respect the integrity of the family.
- Enhance parent's capability to foster the optimal development of their children and themselves.
- Establish links with community support systems
- Provide setting where parents and children can gather, interact, support and learn from each other.
- Enhance coordination and integration of services needed by families.
- Provide emergency support for parents 24 hours a day.

Dentists, as a member of the health profession team, have the opportunity to assist in the prevention and/or reoccurrence of abuse and neglect of children. This needed assistance is categorized and described below:

Health promotion

- Make sure every member of the dental office team is aware of the signs and symptoms of child maltreatment and committed to recognizing and reporting instance of abuse and neglect.

- Dentists, as health professionals, are mandated to report suspected cases of child maltreatment, with immunity granted to voluntary reporters acting in good faith.
- Reports should be made to local protective services or law enforcement agencies, or through the National Child Abuse Hotline, and should include the name, age, and address of the child, the nature and extent of his or her injury, the person believed to be responsible for the abuse or neglect, and any evidence of previous abuse or neglect.

Health education

Dentists can be a major force in the secondary and tertiary prevention of dental neglect through the effective education of parents and children who are either at risk or where it has been determined that dental neglect of a child exists.

- Counsel parents on the importance of good oral hygiene and routine dental care.
- If financial or transportation obstacles exist, provide information to parents about government-sponsored dental care.

Professional education

The following education or re-education of dental students and dentists, respectively, should be the focus of dental institutions and local and state dental associations:

- Increase exposure of dental students to the issue of child maltreatment in their undergraduate dental curricula.
- Mandate dentists to submit a proof of completion on a child abuse and neglect continuing education course to their respective licensing boards.

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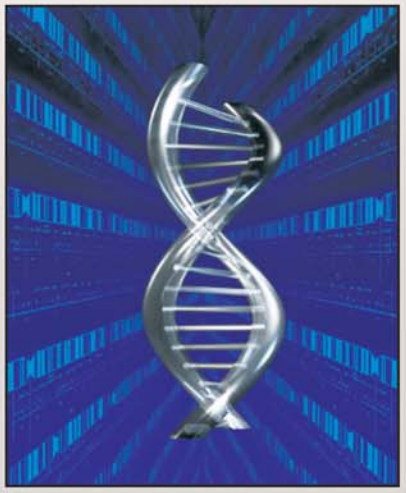

Genetics in Pediatric Dentistry

INTRODUCTION

Genetics is the study of genes at all levels from molecules to population. The term gene is referred to as a basic unit of heredity lying in chromosomes; it is an entire DNA sequence that is necessary for the synthesis of a functional polypeptide or RNA sequence.

Each gene is responsible for a specific trait or a character of an individual; deletion or inclusion of genes gives rise to rise to certain characteristic features resulting either into a character/malformation/disease as per the situation.

There are hundreds and thousands of genes responsible for specific functions in an individual. These genes are located in what is known as 'chromosomes'.

Humans have 46 chromosomes that contain 100,000 genes, including numerous duplicates. Any variation is structure or form of chromosomes results in various disorders and thereby considered as functional units of heredity and evolution.

MODES OF GENETIC TRANSMISSION

1. Mutation
2. Selection
3. Mixture
4. Drift

METHODS AND CRITERIA IN GENETIC IDENTIFICATION

According to Neel and Chull, the roles of genetic factors are as follows:

- Occurrence of the disease in a definite numerical proportion among individuals related by a particular descent.
- Failure of the disease to spread to non-related individuals. The consanguinity effect seen often justifies the role of relatedness.
- Onset of the disease at a characteristic age without a known precipitating event.
- Greater concordance of the disease in identical than in fraternal twins.

CLASSIFICATION OF CHROMOSOMAL ABERRATIONS

Chromosomal aberrations are classified based upon following criterions:

Structural Abnormalities

i. *Deletion:* Breaking away or loss of a portion of chromosome.
For example, Cri-du-chat syndrome.

ii. *Translocation:* Two chromosomes break and exchange their broken segments in reciprocal translocation.
For example, Robertsonian translocations.

iii. *Inversion:* The broken fragment reattaches itself in reverse orientation to the same chromosome.
For example, Increase in miscarriages.

iv. *Duplication:* An over representation of specific chromosomal region.

v. *Transverse centromeric division:*
Instead of dividing longitudinally, centromere divides in transverse plane forming an isochromosome, thereby resulting in duplication of one arm and deletion of another arm of the involved chromosome.

Numerical Abnormalities

i. Involving chromosomes sets:
Monoploid
Euploid
Polyploidy

ii. Involving individual chromosomes
Autosomal derivatives
Monosomy - missing single pair of chromosome
Trisomy
Sex linked derivatives.

Type of Chromosomal Abnormality

Gross chromosomal aberrations
Single gene disorders
Polygenic disorders

GENETIC BASIS OF DENTAL VARIATIONS

The genetic factors cause variations in following features of dental relation. They include:

- Variations in size, shape of jaws
- Variations in size, number, shape, form of teeth
- Malocclusion
- Periodontal conditions
- Incidence of facial clefts
- Growth and development

COMMONLY APPLIED METHODS FOR STUDYING ROLE OF GENETICS IN DENTISTRY

Twin Method

- Monozygotic or identical twins have same genotypes; any difference between them reflects upon the environmental influences; thus if the environmental factors are isolated; it is very much possible to identify the genetic contribution.
- But fraternal twins do not have same genotypes and therefore they have different traits except fore their age.
- Comparison of monozygotic versus dizygotic twins in concordance and discordance of traits permits us to isolate the genetic factors.

Family Line Pedigree

- This is one of the best methods to identify the characteristics or diseases that cannot be explained by any other way can be done by their occurrence between parents and off spring.
- Such studies require large number of families to be included. The genetic traits discussed before can be used in the study; but probability of dominant or recessive traits should be considered in the population.
- The larger the number of generations, the better a trait can be traced by repetitive occurrence.

GENETIC COUNSELING

It is a communication process in which individuals seeking advice are provided with all scientific information enabling them to decide about their current or future pregnancies.

It is a systematic communication mechanism that involves through investigation of the patient's medical; family history and current clinical status to help them in making decisions of their success in reproduction and anticipation of a healthy newborn to enter the world.

Who need's genetic counseling: Basically anyone and everyone can have genetic counseling who are desirous of giving birth to a child free from any disease/deformity. But it is essential for those who are at the risk of certain familial diseases; deformities and other congenital disorders or increased risk of miscarriages.

Genetic counseling is therefore an investigatory and diagnostic process. Genetic counseling is done at different stages.

Gene Mapping Prior to Pregnancy

- It involves genetic investigations of both the partners and their family history evidences to delineate any risk of inheritable diseases or disorders.

Prenatal Stage

- At times life partners undergo genetic counseling after confirmation of pregnancy to ensure the health; safety and disease-free state of the developing embryo or fetus.

Procedures for prenatal diagnosis are:

- Visualization of fetus through:
  - Ultrasonography
  - 3D ultrasonography
  - Fetoscopy, etc
- Analysis of fetal tissue:
  - Amniocentesis
  - Chorionic villus sampling
  - Fetal and maternal blood analysis
  - Fetal live biopsy
  - Fetal skin biopsy

Preimplantation Diagnosis

- In this procedure, 1 or 2 cells are removed from cleavage stage embryos from the patients. The

affected embryos are identified by molecular genetic techniques. Subsequently, healthy genes are re-implanted into the uterus enabling further development till full term.

- Procedures for preimplantation diagnosis are:
  - Polar body biopsy
  - Multi-cell biopsy
  - Blastocyst biopsy

STEPS INVOLVED IN IDENTIFICATION OF GENETIC DISEASE

- Consult the patient and know the reason for consultation
- Explain the principle or basis of genetic counseling and the ways by which the counselor can help the patient
- Build up the family tree "bottom up". Starting from the patient up to the last traceable blood lines of both the partner (for example up to grandparent; cousins; uncles, aunts, etc)
- Fill in all needed details of the family tree systematically including any positive history of abortions, twins, number of pregnancies, consanguinity, congenital diseases, disorders, familial diseases, etc
- Analyze the above details and calculate the risks of recurrence either 'high' or 'low'
- Advice the patient for necessary hematological, genetic, biochemical investigative procedures
- Analyze all the data obtained and arrive at a diagnosis and rate of risks involved
- The final and the most crucial step is decision making. It is briefly explained as follows:
  - Allow the patient and his/her family member to decide on continuation/termination of pregnancy
  - Explain the possible risks involved and any possibilities of correction prior to delivery through intra-uterine fetal surgery or after delivery of the child
  - Possibilities of survival of child after delivery should also be discussed and explained in detail to the patients.

Thus these are the characteristic features of genetics and genetic counseling which is undergoing constant and tremendous transformation into a major field of scientific study and research that is aimed in elimination of various diseases/disorders in the upcoming future.

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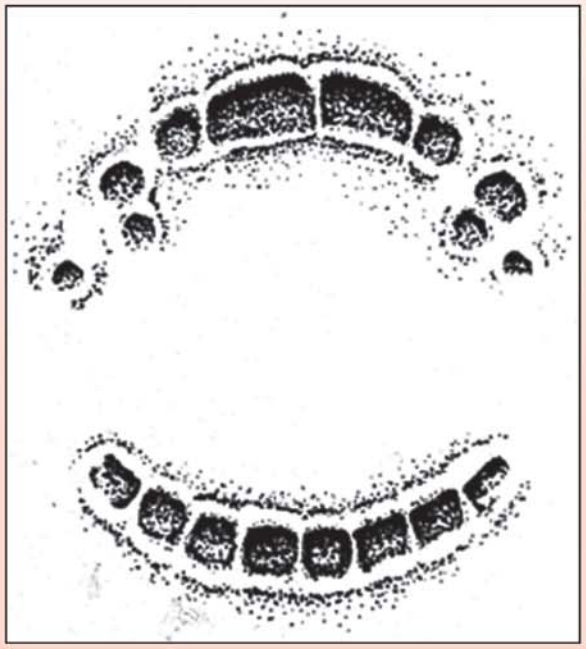

Forensic Pedodontics

INTRODUCTION

Forensic odontology is a branch of forensic medicine and, in the interests of justice, deals with proper examination, handling and presentation of dental evidence in a court of law (Kieser Neilson). It is an investigative aspect of dentistry that analyzes dental evidence for human identification.

Forensic dentistry includes:

a. Forensic odontology
b. Jurisprudence (The science of law
c. Forensic odontologist.

SIGNIFICANCE OF FORENSIC PEDODONTICS

A pedodontist has a spectrum of roles in attending a child patient, complete understanding of this science is one of the chief requisites for the pedodontists as they are often the first ones to interact or deal with children. They have a vital role in assisting forensic experts in cases of crime; mass disasters or child abuse in identifying the affected victim or criminal through dental identification, Bitemark study, etc.

ROLE OF PEDODONTIST

Though a pedodontist does not have a direct role to play, he/she aids the forensic experts in providing all vital information needed. A pedodontist should be equipped with knowledge of dentistry and its application in forensic science.

Child Abuse

A pedodontist can interact better with an abused child and gain his/her confidence; reinforce emotion and motivate the abused child psychologically to healthy state and help to identify any evidences of physical/sexual abuse through any bite marks or finger marks or object marks on the body of the child.

Mass Disaster

In case of mass disasters; a pedodontist's focus is on identification of victims by dental identification of age; dental records can also be traced sometimes from regional dentists.

Age Determination

As already discussed; pedodontist helps in determining the age of the victim from the remains of the victim's dentition. Pedodontist plays a vital role in determining

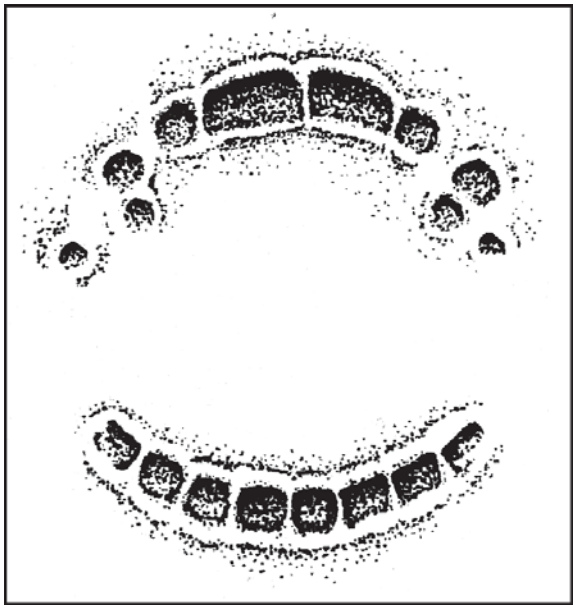

Fig. 28.1: Bite marks

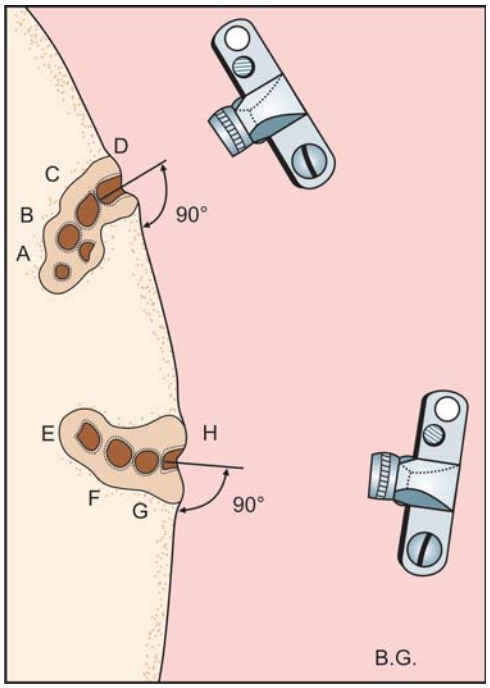

Fig. 28.2: Bite mark measurement

the dental age of the unknown victim based upon factors like dentition; Root morphology and jaw description, etc.

Bite Marks Evidence (Figs 28.1 and 28.2)

Pedodontists should be equipped with knowledge to gather evidence and study the bite marks; collect saliva sample using swabs from bite site to advocate DNA, PCK testing, etc. collection of photographic evidence of bite site, impressions, tissue samples, etc.

Lip Print Identification

Knowledge of cheiloscopy is essential in identifying and collecting evidence from victim's body and possible suspects.

Dental Record Maintenance

Pedodontists should maintain dental records of every child they attend so that they can be retrieved when needed in times of investigation during any incidence of child abuse/murder, etc.

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General Epidemiology and Survey

INTRODUCTION

Epidemiology is an old world, dating back to the third century BC and has its origin in the idea first expressed over 2000 years ago by Hippocrates and others, that environmental factors can influence the occurrence of disease.

The word 'epidemiology' is derived from the Greek word, 'Epidemic'. 'Epi = upon', 'demos = people', 'Logos = science'.

DEFINITIONS

Parkin (1873) defines epidemiology as **"the branch of medical science which deals with the treatment of epidemics".**

John M Last (1988) defined epidemiology as "**the study of the distribution and determinants of health related states or events in specified populations, and the application of this study to the control of health problems".**

COMPONENTS

The three components of epidemiology.

Disease Frequency

Basic measures of disease frequency are ratio or rate which helps to compare disease frequency in different populations in relation to suspected casual factor.

Distribution of Disease

The basic tenet of epidemiology is that the distribution of disease occurs in pattern in a community and that patterns may lead to the generation of hypothesis about causative factors.

These patterns of distribution are best studied in terms of time, place and person.

Determinants of Disease

One of the many functions of epidemiology is to test an etiological hypothesis in identifying the risk factors of the disease. The hypothesis is tested using the principles and methods of epidemiology. This aspect of epidemiology is known as **"Analytical epidemic".**

AIMS OF EPIDEMIOLOGY

The International Epidemiological Association has listed three main aims of epidemiology, which was put forward by '**Lowe and Kostrzewski'** in 1973 is as follows:

- To describe the size and distribution of the disease problems in human populations
- To provide the data essential for the planning, implementation and evaluation of health services for the prevention, control and treatment of diseases and for the setting up of priorities among those services
- To identify etiological factors in the pathogenesis of disease.

PRINCIPLES OF EPIDEMIOLOGY

The principles of epidemiology as a scientific field of science are related to the basic principles of science. The four important principles are:

- Exact observation (strict, vigorous, accurate, precise)
- Correct interpretation (Free from error)
- Rationale explanation (intelligent, sensible reasonable)
- Scientific construction (by expert knowledge and technical skill).

BASIC MEASUREMENTS IN EPIDEMIOLOGY

The most commonly used measurements in epidemiology are:

- Measurement of mortality
- Measurement of morbidity
- Measurement of disability
- Measurement of natality
- Measurement of presence or absence or distribution of characteristic or attributes of disease
- Measurement of medical needs, health care facilities, utilization of health services, etc.
- Measurement of presence or absence or distribution of the environmental and other factors suspected of causing the disease.

Measurement of Mortality

Crude Death Rate

= No of deaths during the year in a population/mid-year population × 1000

Specific Death Rate

Of males = No of deaths from oral cancer during a year/
Mid-year population × 1000

Case Fatality Rate

= Total no of deaths due to particular disease/
Total number of cases due to same disease × 1000

Proportional Mortality Rate

= No. of deaths from specific disease in a year/
Total deaths from all causes in that year

Survival Rate

= Total no of patients alive after 'x' years/
Total no of patients diagnosed or treated

Measurement of Morbidity

Incidence Rate

= Number of new cases of specific disease during a given time period /
Population at risk during that period

Prevalence

= No of all current cases of a specified disease existing at a given point of time /
Estimated population at the same point in time

Relationship between prevalence (P) and incidence (I)

P = I × D
I = P/D or
D = P/I

EPIDEMIOLOGIC METHODS

1. Descriptive epidemiology
2. Analytical epidemiology
3. Experimental epidemiology.

The above three types of epidemiologic methods are briefly explained as follows:

Descriptive Epidemiology

These studies are concerned with the distribution of diseases or health related characteristics in human populations and identifying the characteristics with which the disease in question seems to be associated.

The study describes the disease in terms of time, place and person.

Procedures involved

a. Defining the population to be studied
b. Defining the disease under study
c. Describing the disease by:
   i. Time—short-term fluctuations, Periodic fluctuation, Long-term
   ii. Place—International variations, National variations, Rural-urban differences, Local distributions
   iii. Person—Age, sex, ethnicity, marital status, occupation, social class, behavior, stress, migration, etc.
d. Measurement of disease:
   - Cross-sectional and
   - Longitudinal studies
e. Comparing with known indices
f. Formulation of hypothesis.

Analytical Epidemiology

Its main objective is to test the etiological hypothesis. It consists of two distinct types of observational studies.

a. Case control study or retrospective study
b. Cohort study.

Case Control Study

- It has three distinct features:
  – Both exposure and disease occurred before the start of the study

  - The study proceeds from effect to cause
  - It uses a control group to support or refute an inference.

Basic steps involved are:

- Selection of cases and controls
- Matching
- Measurement of exposure
- Analysis and Interpretation

Cohort Study

The distinct and distinguishing features of cohort study are:

- The cohorts are identified prior to the appearance of the disease under investigations
- The study groups are observed over a period of time to determine the frequency of the disease among them
- The study proceeds forward from cause to effect.

Elements of cohort study:

- Selection of study subjects
- Obtaining data on exposure
- Selection of comparison group
  - Internal/external comparisons and comparison with general population rates
- Follow-up
- Analysis.

Experimental Epidemiology

Aims

- To provide "scientific proof" of etiological factors
- To provide a method of measuring the effectiveness and efficiency of health services for the prevention, control and treatment of disease and improve the health of the community.

Types

Experimental epidemiology is of two types:

- Randomized control trials
- Non-randomized trials.

Basic steps involved in randomized controlled trials:

- Drawing up a protocol
- Selecting reference and experimental populations
- Randomization
- Manipulation or intervention
- Follow-up
- Assessment of outcome.

Types of randomized controlled trials:

- Clinical trials
- Preventive trials
- Risk factor trials
- Cessation experiments
- Trial of etiological agents
- Evaluation of health services.

Uses of Epidemiology

- Study historically the rise and fall of disease in the population
- Community diagnosis
- Planning and evaluation
- Evaluation of individual's risks and chances
- Syndrome identification
- Completing the natural history of disease
- Searching for causes and risk factors.

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