



Introduction to Operative Dentistry

By

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Academic Year 2022-2023



Tokyo Medical and Dental University (TMDU) is ranked 6th in the world and 1st in Japan for dentistry in QS World University Ranking 2022

Definition and History

****Operative dentistry** is the art and science of the diagnosis, treatment, and prognosis of defects of teeth.*

The operative treatment should restore ***proper tooth form, function, esthetics & maintaining the physiologic integrity of the teeth*** in harmonious relationship with the adjacent hard and soft tissues.



Factors affecting operative dentistry

-General indications

(Caries, discolored , fractured and malformed teeth, in addition to the replacement and repair of the restorations)

-General considerations before any treatment

- a. understanding infection control
- b. thorough examination of the teeth, oral cavity, as well as to the systemic health
- c. treatment plan that restore affected area
- d. understanding the materials to be used and their technique
- e. biologic knowledge
- f. understanding biology of the tooth and supporting structure
- g. knowledge of dental anatomy

-Conservative approach

Previously tooth preparations for operative procedures influenced by the concept "extension for prevention" nowadays improvement in restorative materials, and techniques have provided more conservative approaches to restore teeth.

(The primary results of conservative treatment are retention of more intact tooth structure and less trauma to the pulp and soft tissue.)

-Operative dentistry is still recognized as the foundation of dentistry and many of the areas previously included under operative dentistry have become specialty areas such as endodontics, prosthodontics, and orthodontics .

-G.V. Black make significant contributions to the operative dentistry thus he became the foundation of the dental profession.

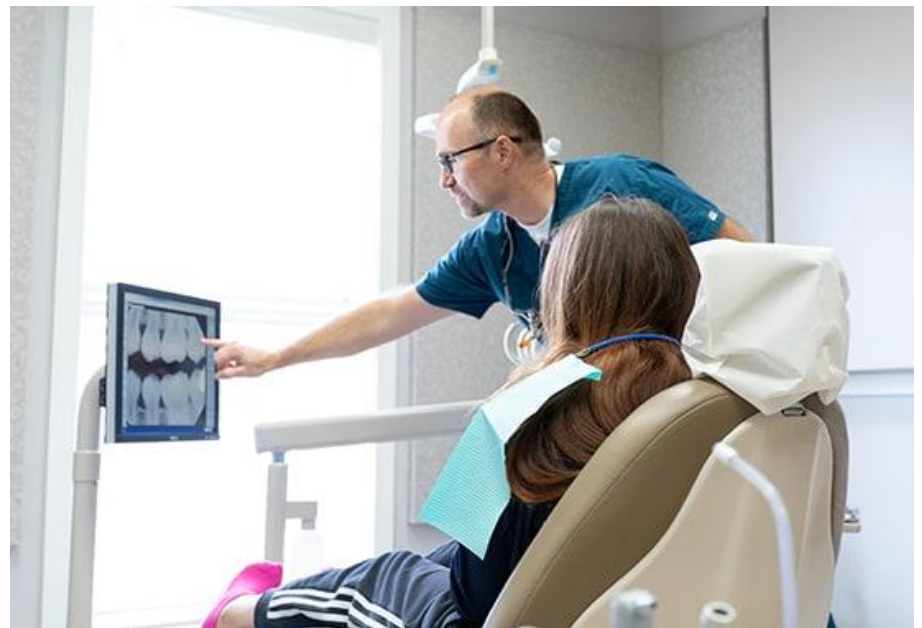


Patient assessment (CH9 in 4th edition)

By

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Pre-operative treatment consideration

I. Patient assessment

1. Infection control

increases in serious transmissible diseases have created great concern and impacted the treatment mode.

Currently COVIT 19 become a serious infectious disease and a great attention should be paid for all dental patients.

Great attention has now offered to assure patients and dentist protection from risks of infectious disease. Use of gloves, masks, protective eyewear, plastic barriers to protect equipment, proper use of disinfectants, and instrument sterilization now essential to provide a professional health care.

Disposable, adjustable plastic ejectors are preferable because of improved infection control.

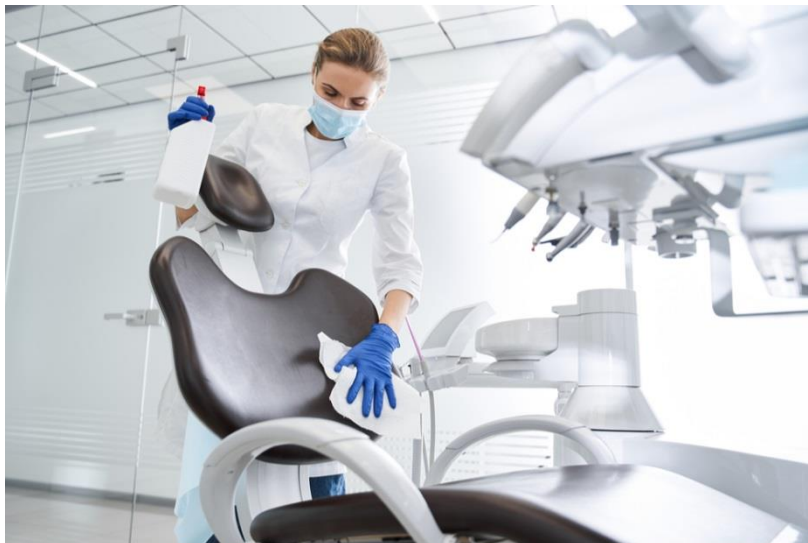


TABLE 9-1 Communicable Diseases of Concern in Dentistry

INFECTIOUS AGENT	ROUTE OF TRANSMISSION	DISEASE
Herpes simplex virus (HSV) types 1 and 2	Congenital; oral (saliva); sexual; direct contact with lesions	Oral/genital herpes Primary herpetic gingivostomatitis Herpes labialis Herpetic whitlow (finger) Keratoconjunctivitis (eye)
Varicella-zoster virus (VZV)	Aerosols; respiratory droplets; direct contact with lesions	Chickenpox, or varicella (primary infection) Shingles, or zoster (reactivated infection)
Human papilloma virus	Direct oral or sexual contacts with lesions	Venereal warts, or <i>condylomata acuminatum</i>
Respiratory viruses (e.g., rhinoviruses, respiratory syncytial virus, influenza viruses)	Direct contact with respiratory droplets; aerosols	Respiratory infections (e.g., cold, flu)
Paramyxoviruses	Direct contact with respiratory droplets; aerosols	Rubeola or measles Mumps
Togavirus	Direct contact with respiratory droplets; aerosols	Rubella, or German measles
Epstein-Barr virus (EBV)	Direct contact with saliva	Infectious mononucleosis
<u>Hepatitis B virus (HBV)</u>	Blood; sexual; perinatal; present in all body fluids, including saliva	Hepatitis, cirrhosis of the liver, hepatocellular carcinoma
<u>Human immunodeficiency virus (HIV)</u>	Blood; sexual; perinatal	Opportunistic infections Neoplastic lesions (e.g., Kaposi's sarcoma) Wasting syndrome Acquired immune deficiency syndrome (AIDS)
<u><i>Mycobacterium tuberculosis</i></u>	Respiratory droplets; aerosols; saliva; ingestion; direct contact	Pulmonary tuberculosis (TB) dissemination to the intestines, kidney, bones, meninges, lymph nodes, and oral structures
<i>Neisseria gonorrhoeae</i>	Sexual contact	Gonorrhea (i.e., oral lesions; gono- coccal arthritis; infections of the skin, eye, heart, and meninges)
<i>Treponema pallidum</i>	Sexual contact, congenital	Syphilis (i.e., oral lesions; dissemi- nated infections to other organs, including the central nervous system [CNS] and heart)

2. Chief complaint

It is important to determine the patient chief complaint (The problem that initiated patient visit or are symptoms or problems expressed by the patient in his own words relating to the condition that prompted the patient to seek treatment.).

All aspects of the patient problem, including (onset, duration, symptoms, and related factors).

The patient should be encouraged to discuss all aspects (symptoms) of the current problem(s), including onset, duration, and related factors they are experiencing.

This information is vital to establishing which specific diagnostic tests are required, determining the cause, selecting appropriate treatment options for the concerns, and building a sound relationship with the patient.



3. Medical review or history:

To identify conditions that could alter, complicate, or contraindicate proposed dental procedures.

(1) communicable diseases that require special precautions (e.g TB, Hepatitis..).

(2) allergies or medications that may contraindicate the use of certain drugs.

(3) systemic diseases and cardiac abnormalities that demand prophylactic antibiotic coverage.

(4) physiologic changes that may influence treatment.

The practitioner also might identify a need for medical consultation or referral before initiating dental care



Comprehensive medical review form is the focus of the patient interview. This form helps the practitioner identify conditions that may affect dental treatment or require referral to a physician. (From The American *Dental* Association.)

ADA. Health History Form

Medical Alert	Condition	Premedication	Allergies	Anaest.	Date
---------------	-----------	---------------	-----------	---------	------

Name _____ Home Phone () _____ Business Phone () _____
Last First Middle
 Address _____ City _____ State _____ Zip Code _____
P.O. Box or Mailing address
 Occupation _____ Height _____ Weight _____ Date of Birth ____/____/____ Sex ☐ M ☐ F
 SS# _____ Emergency Contact _____ Relationship _____ Phone () _____

If you are completing this form for another person, what is your relationship to that person? _____
Name Relationship

For the following questions, please (X) whichever applies, your answers are for our records only and will be kept confidential in accordance with applicable laws. Please note that during your initial visit you will be asked some questions about your responses to this questionnaire and there may be additional questions concerning your health. This information is vital to allow us to provide appropriate care for you. This office does not use this information to discriminate.

Dental Information

Yes	No	Don't Know		Yes	No	Don't Know	
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	Do your gums bleed when you brush?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	Have you ever had orthodontic (braces) treatment?
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	Are your teeth sensitive to cold, hot, sweets or pressure?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	Do you have headaches, earaches or neck pains?
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	Have you had any periodontal (gum) treatments?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	Do you wear removable dental appliances?
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	Have you had a serious/difficult problem associated with any previous dental treatment? If so, explain _____				

How would you describe your current dental problem? _____

Date of your last dental exam _____ Date of last dental x-rays _____

What was done at that time? _____

How do you feel about the appearance of your teeth? _____

Medical Information

Yes	No	Don't Know	
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	Are you in good health?
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	Has there been any change in your general health within the past year?

Do you have any of the following diseases or problems: If you answer yes to any of the 3 items below, please stop and return this form to the receptionist.

<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	Active Tuberculosis
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	Persistent cough greater than a 3 week duration

Yes No Don't Know

(Women Only)

- ☐ ☐ ☐ Are you pregnant?
☐ ☐ ☐ Nursing?
☐ ☐ ☐ Taking birth control pills?

- ☐ ☐ ☐ Have you had an orthopedic total joint (hip, knee, elbow, finger) replacement? If so when was this operation done? _____
☐ ☐ ☐ Have you had any complications or difficulties with your prosthetic joint?
☐ ☐ ☐ Has a physician or previous dentist recommended that you take antibiotics prior to your dental treatment? If so, what antibiotic and dose?

Name of physician or dentist* _____ Phone _____

NOTE TO PATIENT: A new report (July 1997) prepared and endorsed by the American Dental Association and the American Academy of Orthopaedic Surgeons has recommended that antibiotic prophylaxis before dental treatment is not indicated for most dental patients with artificial orthopedic prosthetic joints. This office will be glad to discuss this report with you and provide a copy of it to you and your orthopedic surgeon/physician.

Please (X) if you have or had any of the following diseases or problems.

Yes No Don't Know

- ☐ ☐ ☐ Abnormal bleeding
☐ ☐ ☐ AIDS or HIV infection
☐ ☐ ☐ Anemia
☐ ☐ ☐ Arthritis
☐ ☐ ☐ Rheumatoid arthritis
☐ ☐ ☐ Asthma
☐ ☐ ☐ Blood transfusion
 If yes, date _____
☐ ☐ ☐ Cancer/chemotherapy/radiation treatment
☐ ☐ ☐ Cardiovascular disease.
 If yes, specify below:
☐ Angina
☐ Arteriosclerosis
☐ Artificial heart valves
☐ Coronary insufficiency
☐ Coronary occlusion
☐ Damaged heart valves
☐ Heart attack
☐ Heart murmur
☐ High blood pressure
☐ Inborn heart defects
☐ Mitral valve prolapse
☐ Pacemaker
☐ Rheumatic heart disease
☐ ☐ ☐ Chest pain upon exertion
☐ ☐ ☐ Chronic pain
☐ ☐ ☐ Persistent diarrhea

Yes No Don't Know

- ☐ ☐ ☐ Disease, drug, or radiation-induced immunosuppression
☐ ☐ ☐ Diabetes. If yes, specify below:
☐ Type I (Insulin dependent)
☐ Type II
☐ ☐ ☐ Dry mouth
☐ ☐ ☐ Eating disorder.
 If yes, specify _____
☐ ☐ ☐ Epilepsy
☐ ☐ ☐ Fainting spells or seizures
☐ ☐ ☐ G.E. reflux
☐ ☐ ☐ Glaucoma
☐ ☐ ☐ Hemophilia
☐ ☐ ☐ Hepatitis, jaundice or liver disease
☐ ☐ ☐ Recurrent infections
 Indicate type of infection _____
☐ ☐ ☐ Kidney problems
☐ ☐ ☐ Low blood pressure
☐ ☐ ☐ Mental health disorders.
 If yes, specify below: _____
☐ ☐ ☐ Malnutrition
☐ ☐ ☐ Migraines
☐ ☐ ☐ Night sweats

Yes No Don't Know

- ☐ ☐ ☐ Neurological disorders.
 If yes, specify _____
☐ ☐ ☐ Osteoporosis
☐ ☐ ☐ Persistent swollen glands in neck
☐ ☐ ☐ Respiratory problems.
 If yes, specify below:
☐ Emphysema,
☐ Bronchitis, etc.
☐ ☐ ☐ Severe headaches
☐ ☐ ☐ Severe or rapid weight loss
☐ ☐ ☐ Sexually transmitted disease
☐ ☐ ☐ Sinus trouble
☐ ☐ ☐ Sleep disorder
☐ ☐ ☐ Sores or ulcers in the mouth
☐ ☐ ☐ Stroke
☐ ☐ ☐ Systemic lupus erythematosus
☐ ☐ ☐ Thyroid problems
☐ ☐ ☐ Tuberculosis
☐ ☐ ☐ Ulcers
☐ ☐ ☐ Excessive urination
☐ ☐ ☐ Do you have any disease, condition, or problem not listed above that you think I should know about? Please explain:

NOTE: Both doctor and patient are encouraged to discuss any and all relevant patient health issues prior to treatment.

I certify that I have read and understand the above. I acknowledge that my questions, if any, about inquiries set forth above have been answered to my satisfaction. I will not hold my dentist, or any other member of his/her staff, responsible for any action they take or do not take because of errors or omissions that I may have made in the completion of this form.

4. Dental history

A brief history of past dental treatment can provide useful information about patient's tolerance for dental treatment. Questions about previous episodes of fractured or lost restorations, trauma, infection, sensitivity and pain can give information that will alter the dentist to possible problems and guide him to clinical and radiographic examination. Patients may not volunteer this information; hence specific questions regarding thermal sensitivity, discomfort during chewing, gingival bleeding and pain are warranted. When there is a history of symptoms indicative of pulpal damage or incomplete tooth fracture, specific diagnosis tests should be performed during the examination.

Review of the dental history reveals information about past dental problems and previous treatment.

Such information help dentist to deal with patient problem as well as to guide him for necessary clinical and radio-graphical examination or other tests.

Also, this discussion may lead to identification of other problems such as areas of food impaction, inability to floss, areas of pain, and broken restorations and/or tooth structure.

it is important to know the date and type of available radiographs to ascertain the need for additional radiographs and minimize the patient's exposure to unnecessary ionizing radiation.

5. Risk assessment

Most diseases have been shown to be associated with numerous behavioral/ social, physical/ environmental, microbiologic, or host factors. To formulate an caries risk assessment dentist must gather all appropriate data from both

1. The interview with the patient and
2. The clinical examination for caries detection

A patient at high risk for dental caries should receive aggressive intervention to remove or alter as many risk factors as possible.

Objective of caries risk assessment is determining the current caries lesion activity so we can know if actively progressing.

An inactive lesion may be visible clinically or radio-graphically; however, it may not be active or progressing over time.

Inactive lesions are arrested and may not require operative intervention.

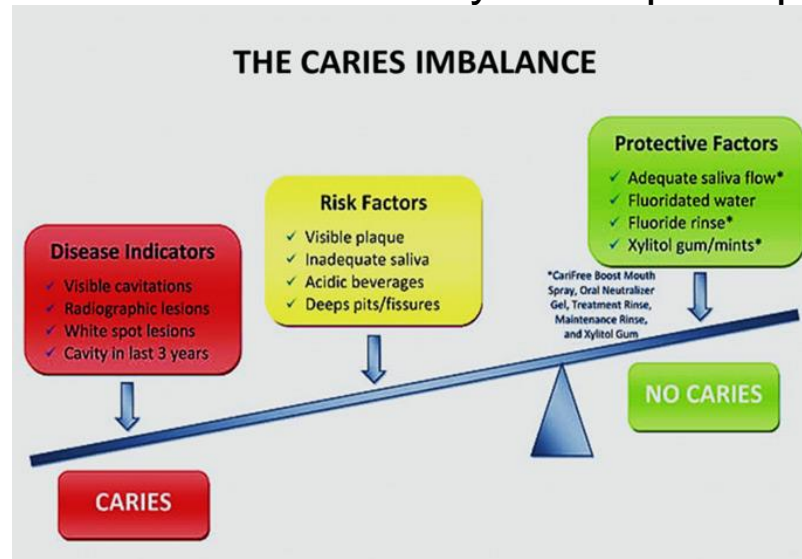
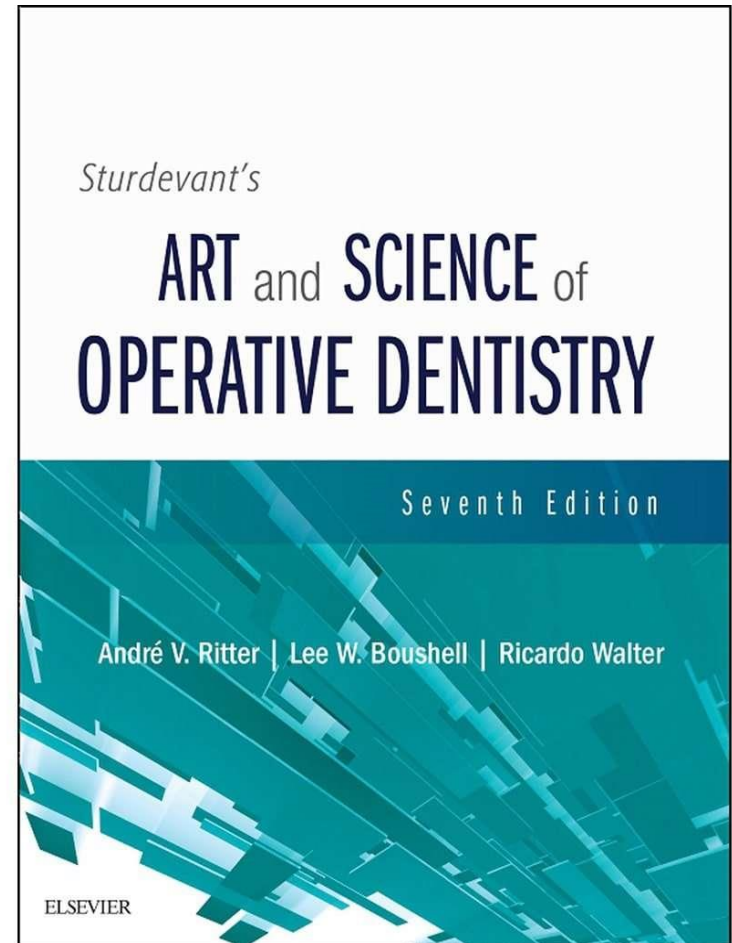
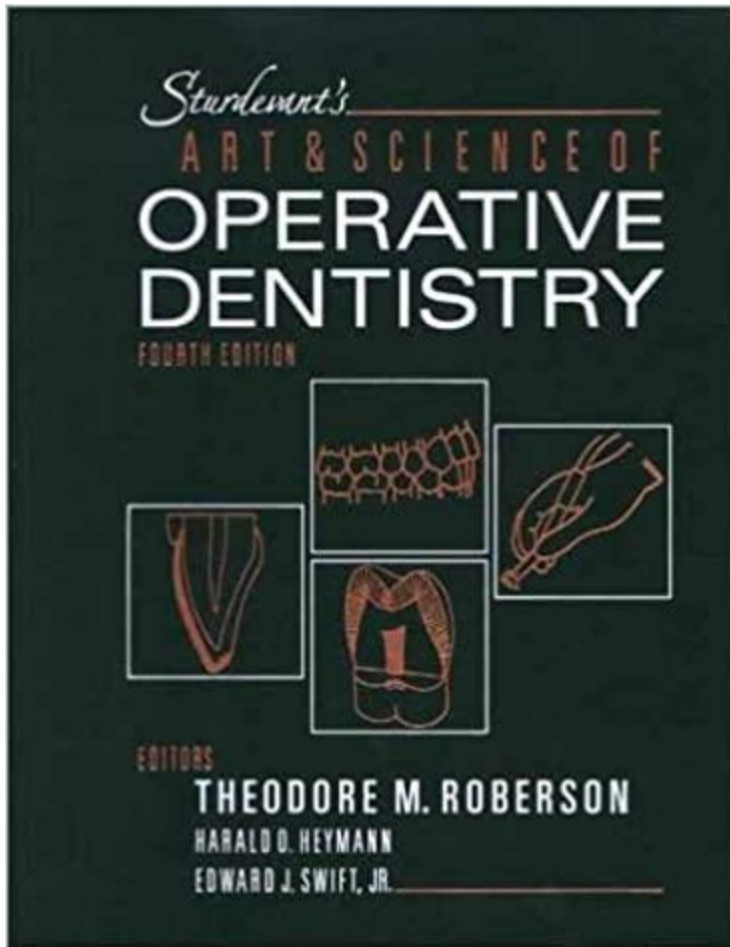


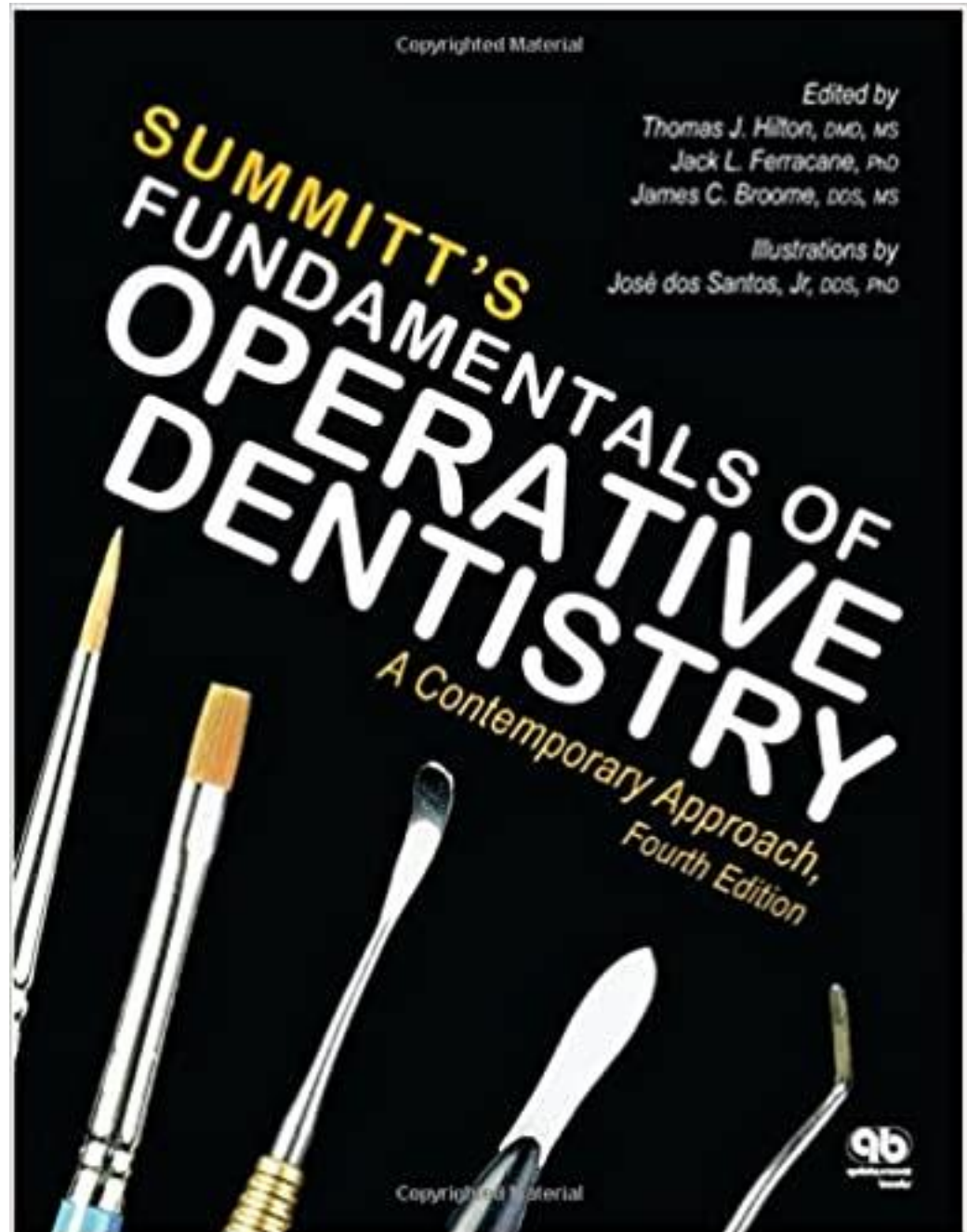
TABLE 9-3 Risk Factors for Caries

FACTORS	HIGH-RISK CHARACTERISTICS
NON-ORAL	
Age	Less than 18 or more than 65 years old
Socioeconomic status	Lower status
Medical condition	Reduced salivation
Medications	Reduced salivation
Fluoride history	Lack of fluoride during tooth development
Dietary habits	High intake of refined carbohydrates; tobacco and alcohol use
Genetic predisposition	Family history of disease
General health	Debilitation and decreased ability to give self-care
ORAL	
Tooth anatomy and composition	Development fissures and low fluoride content
Oral flora/plaque	High levels of mutans streptococci (see Chapter 3)
Previous infections and restorations	History of extensive restoration
Restorations	Defective restorations
Oral hygiene (e.g., skills, knowledge, motivation)	Poor oral hygiene

PRIMARY REFERENCES



ASSISTANT REFERENCE





Examination, diagnosis & treatment plane

(CH9 in 4th edition)

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II. Examination & Diagnosis

Clinical *examination*:

is the process of observing both normal and abnormal conditions.

Preoperative examination of the occlusion, dentition, & periodontium is essential.

-*Centric occlusion.*

-Retruded contact position (RCP).

-Excursion/excursive movements.

-Working side.

-Non-working side.

Diagnosis is a determination of variations from normal condition.



A. General considerations of examination: Includes chart recording and preparation for dental examination.

1- Charting and record :

A dental charting is a stylised record of the patient's current dental status. Thus forming part of the medico-legal record.

The object of a dental chart is to record:

- All the present teeth.
- Teeth that are absent or unerupted.
- Presence and condition of existing restorations (including dentures and bridgework).
- Presence and extent of dental caries and other dental abnormalities.
(e.g. non-carious tooth tissue loss, fractures, developmental defects and discoloration).

2- Preparation for clinical examination

The clinical examination is performed systematically in a clean, dry, well-illuminated mouth.

Proper instruments including clean and clear dental mirror, sharp explorer, and periodontal probe are required.

A routine for charting should be established such as starting in the upper right quadrant with the most posterior tooth and progressing around the maxillary and mandibular arches.

B. Soft tissue examination

Evaluation of oral structures is necessary before operative care is initiated and to determine an oral manifestations of contagious diseases as well.

Begin by examining the submandibular glands and cervical nodes for abnormalities in size, texture, mobility, and sensitivity to palpation. Then, palpate the masticatory muscles for pain or tenderness.

Next, start in one area of the mouth and follow a routine pattern of visual examination and palpation of the cheeks, vestibules, mucosa, lips, lingual and facial alveolar mucosa, palate, tonsillar areas, tongue, and floor of the mouth.

C. Teeth and restoration examination:

(caries, amalgam, composite, cast restorations)

1- Caries examination:

Caries can be examined with the aids of

- (1) visual changes in tooth surface texture or color,
- (2) tactile sensation when an explorer is used correctly.
- (3) Radiographs.
- (4) Transillumination.



2- Amalgam and composite examination: Clinical evaluation of *amalgam restorations* requires visual observation, application of tactile sense with the explorer, use of dental floss, interpretation of radiographs, and experience. Defects in restorations includes (*Proximal overhangs, Marginal gap or ditching, Voids, fracture, proximal contact area, Recurrent caries....*)

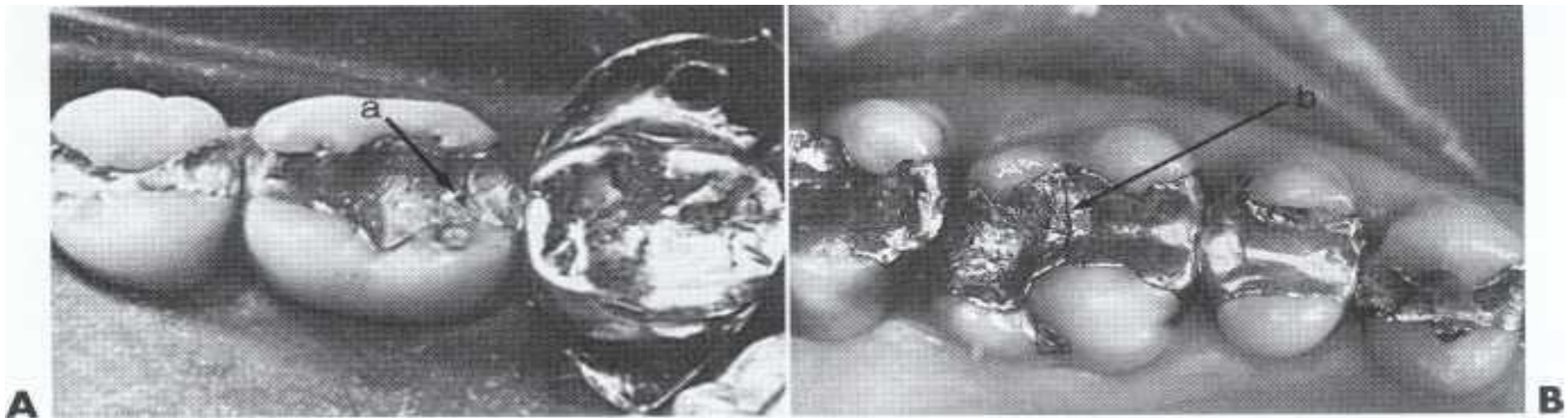


FIG . 9-10 Lines across the occlusal surface of an amalgam restoration may be: A, a fracture line (a) that indicates replacement, or B, an interface line (b) that indicates two restorations placed at separate appointments, which by itself is not sufficient indication for replacement.

3- Cast restoration Cast

Restorations should be evaluated clinically in the same manner as amalgam restorations. If any aspect of the restoration is not satisfactory or is causing tissue harm, it should be classified as defective and considered for recontouring, repair, or replacement

4. Radiographic examination of teeth & restorations

Exposure to **any amount** of radiation can result in adverse affects. Digital radiography are now available and are designed to enhance diagnostic yield and reduce radiation exposure. Radiographs are excellent diagnostic media, they do have limitations. **The only way to guard against these limitations is to continually correlate clinical and radiographic findings.**

Guidelines for Prescribing Dental Radiographs for Dentate Adults

NEW PATIENTS		RECALL PATIENTS	
ALL NEW PATIENTS TO ASSESS DENTAL DISEASES	CLINICAL CARIES OR HIGH-RISK FACTORS FOR CARIES*	NO CLINICAL CARIES AND NO HIGH-RISK FACTORS FOR CARIES*	PERIODONTAL DISEASE OR A HISTORY OF PERIODONTAL TREATMENT
Individualized radiographic examination consisting of posterior bitewings and selected periapicals; a full-mouth intraoral radiographic examination is appropriate when the patient has clinical evidence of generalized dental disease or a history of extensive dental treatment	Posterior bitewing examination at 12- to 18-month intervals	Posterior bitewing examination at 24- to 36-month intervals	Individualized radiographic examination consisting of selected periapical and/or bitewing radiographs for areas where periodontal disease (other than nonspecific gingivitis) can be demonstrated clinically

5. Adjunctive aids for examination of teeth :

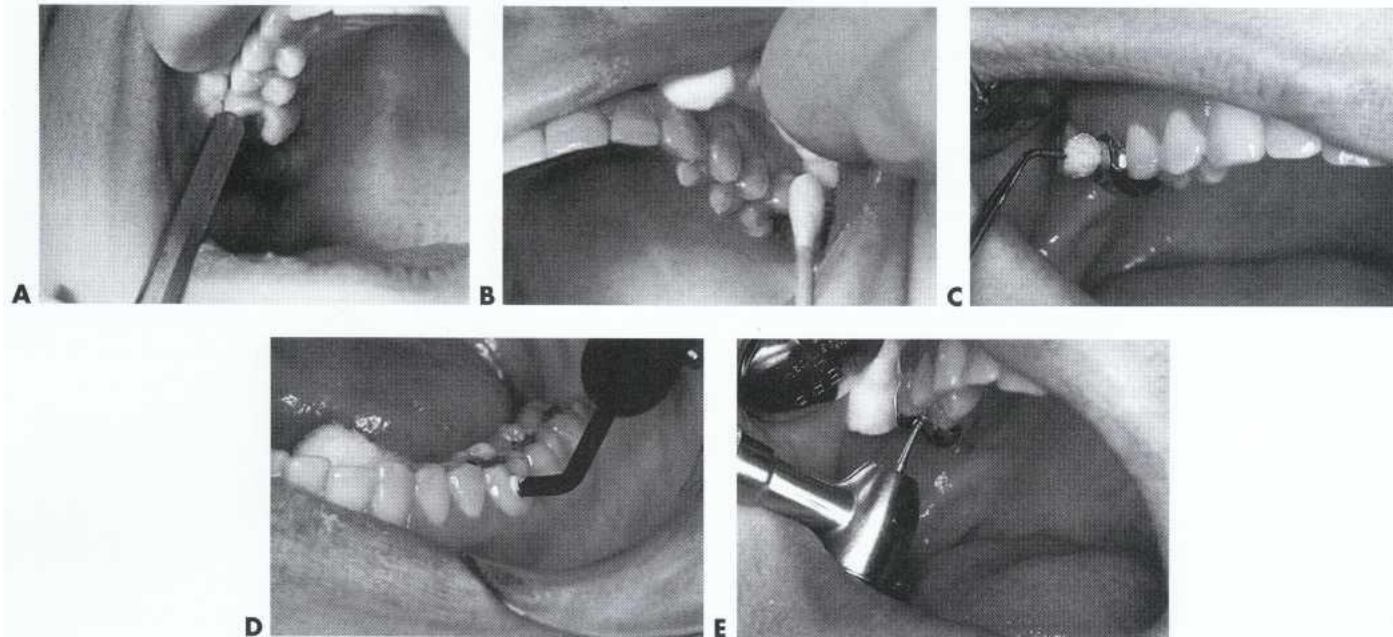
Percussion test: is performed by gently tapping the occlusal or incisal surfaces of the suspected tooth and adjacent teeth with the end of the handle of a mouth mirror to determine the presence of tenderness

Palpation: also can reveal nontender swellings that may be overlooked otherwise.

Pulp vitality: thermal tests, and electric pulp test.

Study casts: are helpful in providing an understanding of occlusal relationships, developing the treatment plan, and educating the patient.

Additional aids: transillumination, the biting test, mobility testing, the anesthetic test, and occlusal analysis.



Diagnostic aids for examining teeth show

A, Percussion.

B, Cold test.

C, Heat test with hot gutta-percha.

D, Electric pulp test.

E, Test preparation.

D. Periodontium examination

The examination of periodontium includes

(clinical , radiographic , occlusion , and patient in pain)

EXAMINATION OF THE PATIENT IN PAIN

One of the most challenging problems a dentist encounters is the treatment of patients who have pain in the jaws or teeth.

Such problems often can test the dentist's diagnostic skills.

The cause of discomfort must be determined before relief can be provided.

The problem can be identified and treated by gathering information from the patient and objective information from the clinical examination supplemented with appropriate diagnostic tests.

The patient is then asked to describe various characteristics of the pain, particularly:

- (1) the onset and duration,
- (2) stimuli,
- (3) spontaneity,
- (4) intensity,
- (5) factors that relieve it (Box 9-2).

After assessing the **subjective** symptoms described by the patient and developing a preliminary diagnosis, the dentist should apply **objective tests** to confirm the diagnosis.

These include a *percussion test* to determine possible inflammation in the periodontal ligament (PDL), *palpation* to examine for any tenderness in the apical region, and *transillumination* to check for cracks or caries in the tooth as well as for tooth color changes that may indicate loss of vitality.

If a tooth is suspected of having a pulpal problem, *electric pulp testing* combined with *thermal testing* may assist in the diagnosis.

Periodontal probing helps rule out periodontal abscess.

Clinical Appearance and Diagnosis

Dentist must recognize any pathological changes or traumatic condition on the enamel surface that different from normal characteristic of the surface.

Key diagnostic sings may include:

1. Color changes associated with demineralization

Color changes related to enamel demineralization and caries are critical diagnostic observation because translucency is directly related to degree of mineralization. Subsurface enamel porosity from carious demineralization is manifested clinically by a milky white opacity called (white spot lesion); when located on smooth surfaces. In later stages of caries, internal demineralization of enamel at the DEJ, subsurface cavitation imparts a blue or gray color to the overlying enamel.

Care must be paid for other anomalies development like extrinsic stains, excessive fluorosis which can also change color of enamel surface.



2. Cavitation

The dentin is affected until enamel breaks away to create a cavity, a restoration must then be placed. If untreated the cavitation expands to compromise the structural strength of the crown and microorganisms infiltrate into deep dentin to affect the vitality of the tooth.

When the carious lesion extends gingival to CEJ as in root caries, isolation, access and gingival tissue response complicate the restorative procedure.



3. Excessive Wear

Although the enamel is considered the most hard structure but it still liable for wear when become in contact with opposing hard restoration or porcelain or even with opposing enamel.

The clear manifestation of heavy occlusal wear is demonstrated when rounded cuspal contacts are ground to flat facets. Depending on factors such as bruxism, malocclusion, age and diet; cusps may be completely lost and enamel abraded away so that dentin is exposed.

Attention should be considered during cavity preparation therefore the outline form should be designed so that the margins of restorative materials avoid critical high stress areas of occlusal contact to avoid any future complication like fracture of tooth and/or restoration.



Attrition



Abfraction



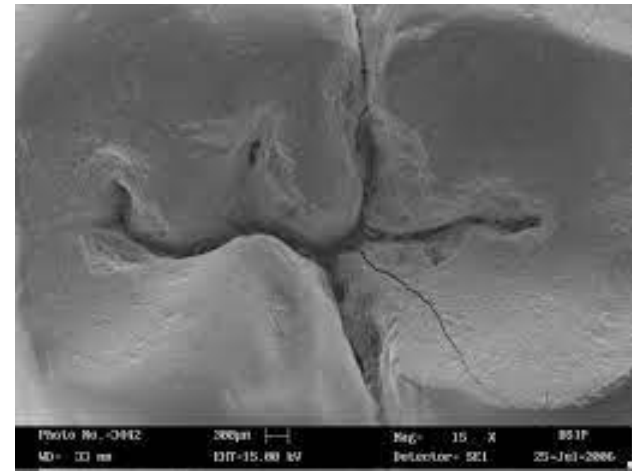
Erosion



Abrasion

4. Morphological Faults and fissures:

A deep fissure is formed by incomplete fusion of lobes of cuspal enamel in the developing tooth. Pits and fissures defects are eight times more vulnerable to caries than are smooth surfaces. Careful observation of enamel surrounding fissures for evidence of demineralization or cavitations' is necessary to determine the need for restorative intervention.



5. Cracks

Pronounced crack extends from developmental grooves across marginal ridges to axial walls or from the margins of large restorations may cause cuspal fracture. When this crack extends through dentin or when the patient has pain when chewing; the tooth requires a restoration that provide complete cuspal coverage.

III. Treatment planning

1. General consideration

A treatment plan is a carefully sequenced series of services designed to eliminate or control etiologic factors, repair existing damage, and create a functional, maintainable environment.

Treatment plans are influenced by patient preferences, motivation, systemic health, emotional status, and financial capabilities.

2. Sequence of treatment plane

Treatment plan sequencing is the process of scheduling the needed procedures into a time frame.

Complex treatment plans often should be sequenced in phases, including an urgent phase, a control phase, a reevaluation phase, a definitive phase, and a maintenance phase.

However, for most patients, the first three phases are accomplished as a single phase.

Urgent : a patient presenting with swelling, pain, bleeding, or infection should have these problems managed as soon as possible and certainly before initiation of subsequent phases.

Control Phase :

- (1) eliminate active disease such as caries and inflammation,
- (2) remove conditions preventing maintenance,
- (3) eliminate potential causes of disease, and
- (4) begin preventive dentistry activities.

Reevaluation: The holding phase is a time between the control and definitive phases that allows for resolution of inflammation and time for healing.

Definitive or Corrective phase of treatment: may include endodontic, periodontic, orthodontic.

Maintenance: This phase includes regular recall examinations

3. Indications for operative treatment

1. Operative preventive treatment (*primary goal of dentistry is to prevent disease*)
2. Restoration of incipient lesions (*Poor oral hygiene and a low frequency of routine dental care in unmotivated patients*)
3. Esthetic treatment
4. Treatment of abrasion, erosion, and attrition
5. Treatment of root-surface caries
6. Treatment of root-surface sensitivity
7. Repairing and resurfacing existing restorations
8. Replacement of existing restorations
9. Indications for (direct composite, tooth-colored restorations, amalgam restorations)



**YOUR HARD STUDY WILL BE
APPRECIATED**



INFECTION CONTROL & STERILIZATION

(CH8 in 4th edition)

By

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Increases in serious transmissible diseases over the last decades have created global concern and impacted the treatment mode in dentistry.

Microbial exposures in the dental practice involve both:

Airborne contamination

-A high-speed handpiece is capable of creating airborne contaminants from bacterial residents in the (dental unit water spray system and from microbial contaminants from saliva, tissues, blood, plaque and fine debris from carious teeth.

-Aerosols consist of invisible particles ranging from 50-5 μm that can remain suspended in the air and breathed for hours.

-Aerosols and larger particles may carry agents of any respiratory infection borne by the patient.

-Barrier protection of personnel using masks, protective eyewear, gloves, and gowns is now a standard requirement for dental procedures.

- A pretreatment mouth rinse, rubber dam, and high velocity air evacuation also can reduce risk.

- Adequate air circulation should be maintained.



Digital contamination of surfaces or (Hand surface contamination)

This can impact and exchanged between both patients and dentist. This may encountered by different ways (i.e., hands soiled with saliva that repeatedly contact operator equipment and surfaces and return to the patient's mouth during treatments).

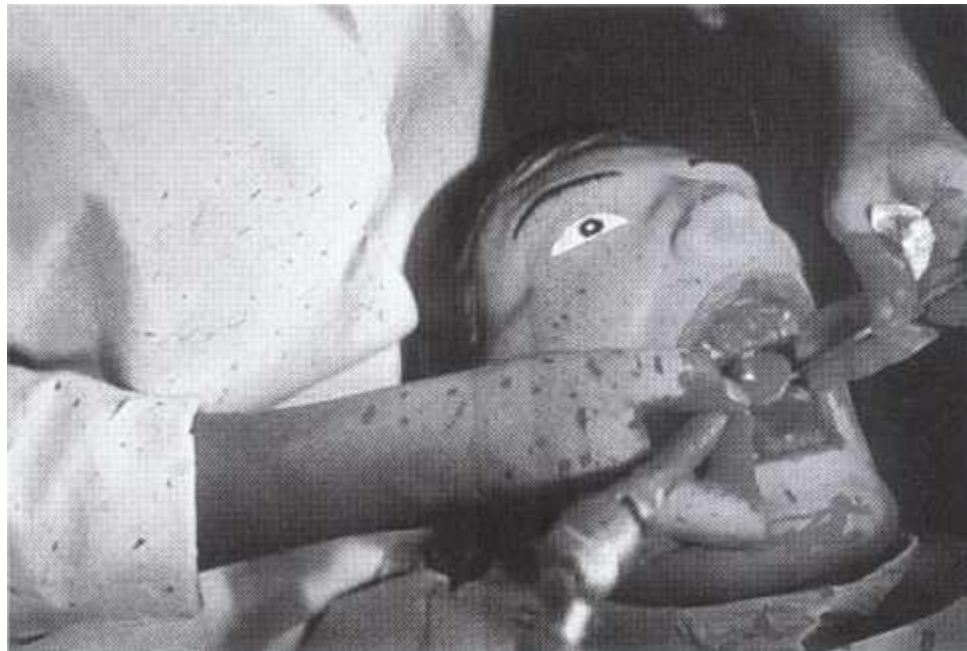
With saliva-contaminated hands, the hygienist, dentist, and assistant could repeatedly contact or handle unprotected operatory surfaces during treatments if not careful.

Sampling by some investigation have confirmed widespread residual contamination with oral bacteria. Thus contamination was not always controlled by use of cleaning and disinfecting procedures.

The following items or areas that may still be contaminated after cleaning included

handpieces; unprotected lamp handles; air-water syringe handles; control switches on the patient's chair; seat edges and rests of the dentist's and assistant's chairs; faucet knobs; cabinet, drawer, and operatory tray handles; room light switches, amalgam mixing equipment, light-curing units and telephones.

Special attention should be given to mobile phone nowadays it is considered a big source of contamination.



Cross Infection

Patients infected usually are not aware of the source of their infection and go elsewhere for diagnosis and treatment of non-oral infections.

The current pandemic disease of COVIT19 become a serious infection worldwide.

Hepatitis B and AIDS were the infectious disease to gain attention as a risk for health care personnel who have blood and body-fluid contact therefore cross-infection should be prevented.

In dentistry saliva considered a potentially infectious material, since oral manipulations and dental treatments routinely cause saliva to become contaminated with the patient's blood.

All patients and blood contaminated body fluids are treated as infectious.

When dental personnel experience exposure to saliva, blood, and possible injury from sharp instrumentation while treating patients, they are more vulnerable to infections if they have not had the proper immunizations or used the proper protective barriers.

Importance of Medical History :

- (1) To detect any unrecognized illness that requires medical diagnosis and care;
- (2) To identify any infection or high risk that may be important to a clinical person exposed during examination, treatment, or cleanup procedures;
- (3) To assist in managing and caring for infected patients.
- (4) To reinforce use of adequate INFECTION CONTROL procedures.

BARRIER PROTECTION

Gloves. all clinical personnel must wear treatment gloves during all treatment procedures. After each appointment, or if a leak is detected, remove gloves, wash hands, and put on fresh gloves.

Instructions for Handwashing. At the beginning, remove watches, jewelry, and rings, or at least those with enlarged projections or stones that can penetrate gloves; then wash hands with a suitable cleanser (4% chlorhexidine).

Eyewear. like goggle or glasses and when are removed they should be cleaned and disinfected.

Masks. Wear a mask to protect against aerosols. *Change the mask between every patient or whenever it becomes moist or visibly soiled.*

Hair Protection. Hair can trap heavy contamination that, if not washed away, can be rubbed back from a pillow onto the face at night.

White prone. Operatory clothing becomes highly spattered with invisible saliva and traces of blood throughout the day.

DISPOSAL OF CLINICAL WASTE

Contaminated materials such as used masks, gloves, blood/saliva-soaked sponges, cotton rolls, and so on, must be discarded safely.

Special care should be given to needle.

Separating needles and sharps into hardwalled, leak-proof, and sealable containers and out of soft trash has provided adequate safety.

The concept of asepsis is to prevent cross contamination.

All items that are touched with saliva-coated hands must be rendered free of contamination before treating the next patient.

These contaminated items can be discarded; protected by disposable covers; or removed, cleaned, and sterilized.

STERILIZATION

killing all forms of life, including the most heat-resistant forms, bacterial spores.

- For instruments that can penetrate tissues, this provides control of spore-forming tetanus and gas gangrene species, as well as all pathogens borne by blood and secretions.
- Sterilization necessary because Infectious dental patients are often undetected.
-
- Sterilization provides a method of instrument recycling that can be monitored and documented to show that conditions for control of disease transmission were indeed established.
- Because most instruments contact mucosa and/or penetrate oral tissues, it is essential that reused instruments be thoroughly cleaned and sterilized by accepted methods that can be routinely tested and monitored.

Four accepted methods of sterilization are:

- A. Steam pressure sterilization (autoclave)
- B. Chemical vapor pressure sterilization (chemiclave)
- C. Dry heat sterilization (dryclave)
- D. Ethylene oxide sterilization

Each method and each commercial modification has very specific requirements regarding timing, temperature suitable packaging of materials, and kinds of items and materials that can be safely and effectively sterilized **Ignoring any of these specifications can prevent sterilization or damage materials or instruments.**

Stainless steel instruments and mirrors used for operative, endodontic, periodontics, or dental hygiene procedures can be sterilized by any accepted method.

Both high- and low-speed handpieces are best autoclaved.

Burs, can be safely sterilized by dry heat or chemical vapor in a chemiclave or in a gas sterilizer, but they may rust or corrode if not protected from steam in the autoclave.

A- STEAM PRESSURE STERILIZATION (AUTOCLAVING)

- Sterilization with steam under pressure is performed in a steam autoclave .
- For a light load of instruments, the time required at (121° C) for at least of 15 minutes at 15 lbs of pressure. Time for wrapped instruments can be reduced to 7 minutes if the temperature is raised to approximately (134° C) to give 30 pounds of pressure.
- **Advantages of Autoclaves.**
 1. Autoclaving is **the most rapid and effective method** for sterilizing cloth surgical packs and towel packs.
 2. Most spores & viruses can be destroyed .
- **Disadvantages of Autoclaves.**
 1. Items sensitive to the elevated temperature cannot be autoclaved.
 2. Autoclaving tends to rust carbon steel instruments and burs.

B- CHEMICAL VAPOR PRESSURE STERILIZATION (CHEMICLAVING)

Chemical vapor pressure sterilizers operate at (131° C) and 20 pounds of pressure. They are similar to steam sterilizers and have a cycle time of approximately 30 min.

Advantages of Chemiclaves.

Carbon steel and other corrosion-sensitive burs, instruments, and pliers are said to be sterilized without rust or corrosion.

Disadvantages

1. Items sensitive to the elevated temperature will be damaged.
2. Towels and heavy cloth wrappings of surgical instruments may not be penetrated to provide sterilization.

C- DRY HEAT STERILIZATION

•Conventional Dry Heat Ovens.

•Dry heat sterilization is readily achieved at temperatures above (160° C). Individual instruments must actually be heated for 30 minutes to achieve sterilization.

•Advantages of Dry Heat Sterilization.

1. Carbon steel instruments and burs do not rust, corrode, or lose their temper or cutting edges if they are well dried before processing.
2. rapid cycle is possible at high temperature.
3. Efficient and economic.

•Disadvantages of Dry Heat Sterilization.

High temperatures may damage more heat-sensitive items, such as rubber or plastic goods and affect sharpness of the instrument.

Short Cycle high temperature sterilization

It reduces total sterilization time to 6 minutes for unwrapped and 12 minutes for wrapped instruments.

D- ETHYLENE OXIDE STERILIZATION

- Ethylene oxide sterilization is the best method for sterilizing complex instruments and delicate materials.
- **Advantages**
less expensive and can operate over night to produce sterilization.

Disadvantages

Porous and plastic material absorb gas and therefore require time for elimination before contact tissue.

BOILING WATER

Instrument should be cleaned and boiled at 100 C for 10-30 min.

- Boiling is a method of high-level disinfection that has been used when actual sterilization cannot be achieved.
- **Advantages of boiling water sterilization**
- Rapid and cheap.
- **Disadvantages**
 1. Boiling water does not kill spores and cannot sterilize instruments.
 2. rusting of the instrument and lose their sharpness.

Uses of High-Level Disinfection.

- instruments now exist that cannot be heat sterilized. High-level disinfection is used mainly for plastic items that enter the mouth and that cannot withstand heat sterilization.
- Periodontal, restorative, and endodontic instruments are readily processed by autoclave or chemical vapor pressure sterilization. Carbon steel instruments and burs, if dried well before sterilizing, are best sterilized by dry heat and chemical vapor pressure sterilizers because these methods reduce the risk of rust.

- Oral fluid contamination of rotary equipment and especially the high speed handpiece involve:
 - (1) contamination of handpiece external surfaces and crevices,
 - (2) turbine chamber contamination that enters the mouth,
 - (3) water spray retraction and aspiration of oral fluids into the water lines of older dental units,
 - (4) growth of environmental aquatic bacteria in water lines,
 - (5) exposure of personnel to spatter and aerosols generated by intraoral use of rotary equipment.
- Therefore *Sterilization of handpieces must be monitored and documented* .Otherwise scrub and disinfect the motor-end for each reuse if it cannot be sterilized.



Cariology: Lesion, Diagnosis, Etiology, Classification, and Risk assessment

By

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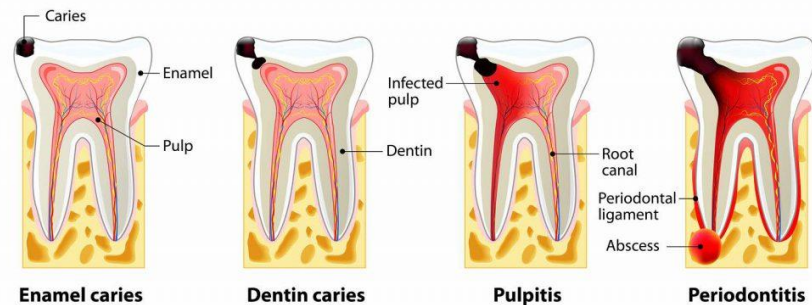
Dental caries is an infectious microbiologic disease of the teeth that results in localized dissolution and destruction of the calcified tissues.

It is a preventable disease and can be arrested or reversed in its early stages.

Caries lesion. Tooth demineralization as a result of the caries process.



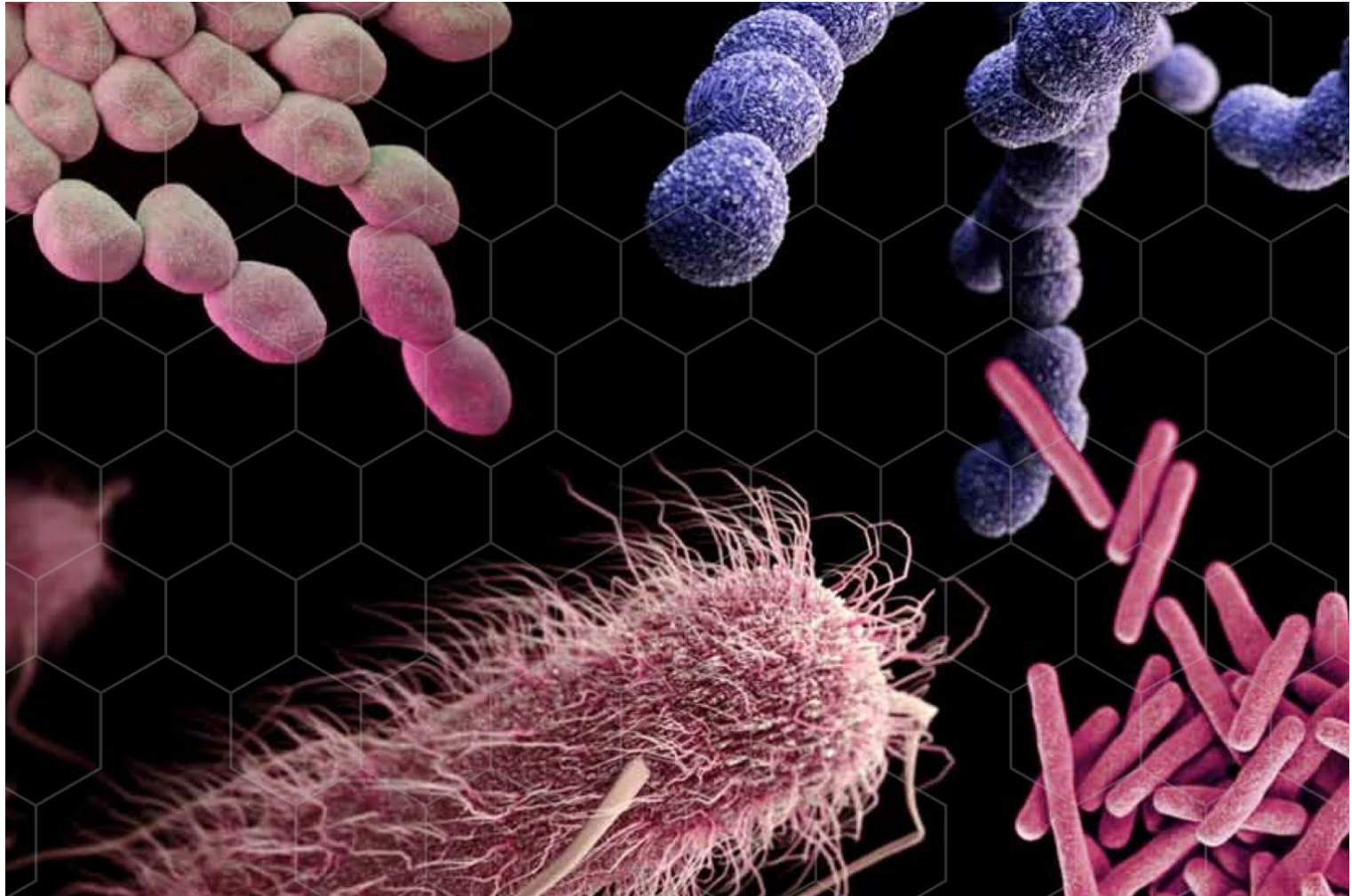
THE STAGES OF CARIES DEVELOPMENT



Dental caries is one of the multifactorial oral disease is caused primarily by an imbalance of the oral flora due to the presence of fermentable dietary carbohydrates on the tooth surface over time.

At the tooth level, dental caries activity is characterized by localized demineralization and loss of tooth structure.

Over 300 species of bacteria present in the oral cavity, only mutans streptococci, are caries causing (**cariogenic**) organisms.

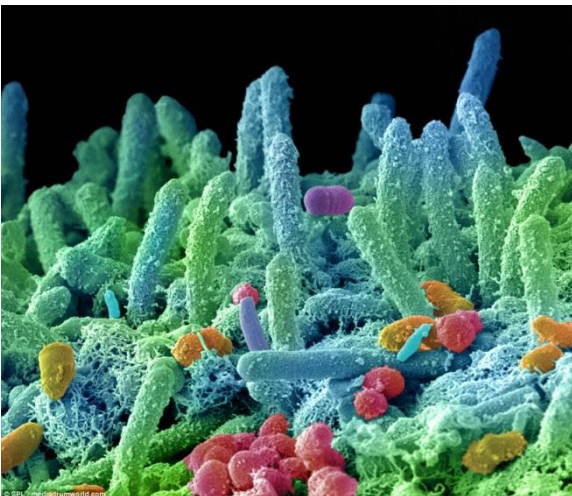


MS are the primary causative agents of **initial coronal caries** because they

- (1) adhere to enamel.
- (2) produce and tolerate acid.
- (3) Live and grow in a sucrose-rich environment.
- (4) produce bacteriocins, substances that kill off competing organisms.

Mutans Streptococci and lactobacilli can produce great amounts of acids (acidogenic), and appear to be the primary organisms associated with caries in man.

Mutans Streptococci are most strongly associated with the **onset of caries** while lactobacilli are associated with **active progression of cavitated lesions**.



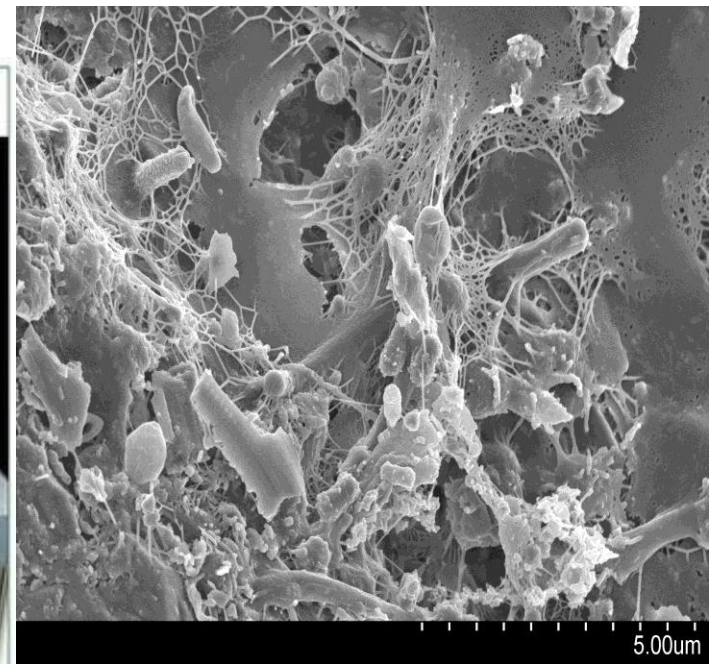
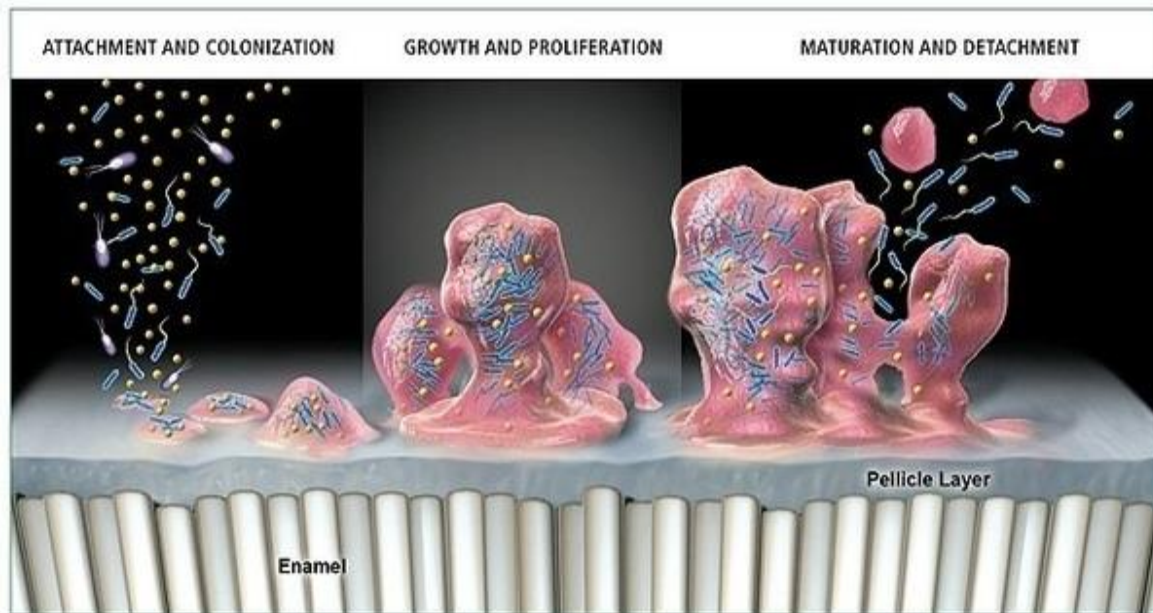
Biofilm

Dental plaque is a term historically used to describe the soft, tenacious film accumulating on the surface of teeth.

Dental plaque has been more recently referred to as the *dental biofilm* or simply the *biofilm*, which is a more complete and accurate description of its composition (bio) and structure (film).

The biofilm is composed mostly of bacteria, their by-products, extracellular matrix, and water.





Dental Plaque (Biofilm) : *A gelatinous mass of bacteria adhering to the tooth surface.*

Carious lesions occur under a mass of bacteria producing acidic environment to demineralize tooth structure after metabolization of refined carbohydrates .



Etiology of Dental Caries:

Dental caries has a multifactor etiology; however four principle factors are necessary for the production of a carious lesion:

- **Dental Plaque (Bacteria)**

Cariou lesions only occur under a mass of bacteria capable of producing a sufficiently acidic environment to demineralize tooth structure.

- **Diet**

(Substrate such as a fermentable carbohydrate or dietary sugars) which is necessary for microorganesim to as nutrition with subsequent production of acid as byproduct that would initiate the demineralization.

- **Tooth**

(A susceptible tooth) as teeth composed of minerals (calcium and phosphate) therefore low pH will create environment for demineralization of the minerals resulting destruction of hard tissue.

pH is a measure of how acidic/basic water is. The range goes from 0 to 14, with 7 being neutral. pHs of less than 7 indicate acidity, whereas a pH of greater than 7 indicates a base.

- **Time**

To produce demineralization process sufficient time at low pH is required. It is difficult to determine the exact time for demineralization inside patient mouth.

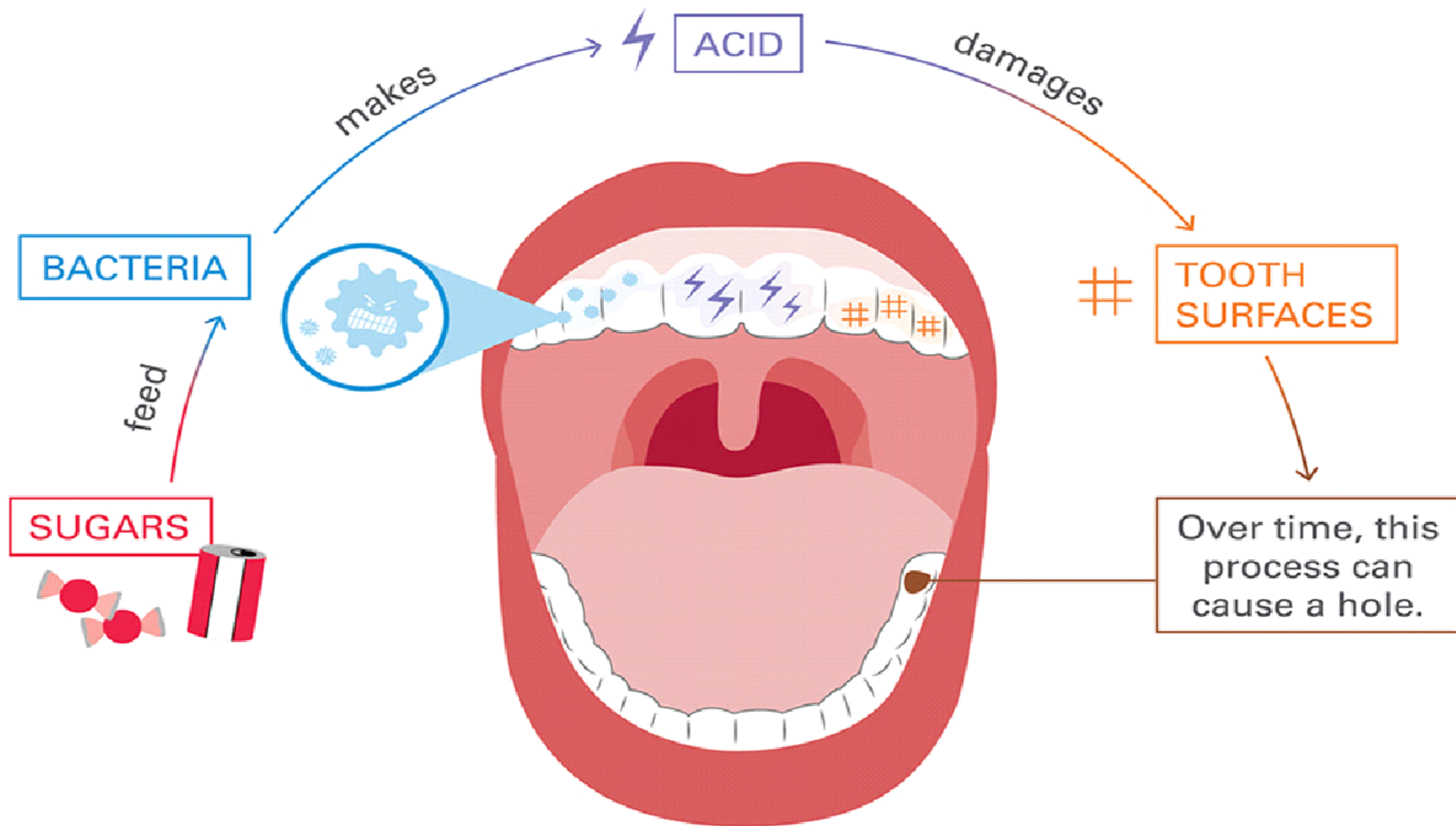
Diet and Dental Caries:

High-frequency exposure to fermentable carbohydrates may be the most important factor in producing cariogenic biofilm and ultimately caries lesions.

Frequent ingestion of **fermentable carbohydrates** begins a series of changes in the local tooth environment, essentially changing the composition of the biofilm, thus favoring the growth of highly acidogenic bacteria that eventually leads to caries lesion formation.

In contrast, when ingestion of fermentable carbohydrates is severely restricted or absent, biofilm growth typically does not lead to caries lesions.

Dietary sucrose plays a leading role in the development of pathogenic biofilms and may be the most important factor. Sucrose in particular allows the formation of extracellular polysaccharides, which render the biofilm viscous and sticky.



Process of Demineralization and Remineralizaion:

Repeated demineralization events may result from a predominantly pathologic environment causing the localized dissolution and destruction of the calcified dental tissues, evidenced as a caries lesion.

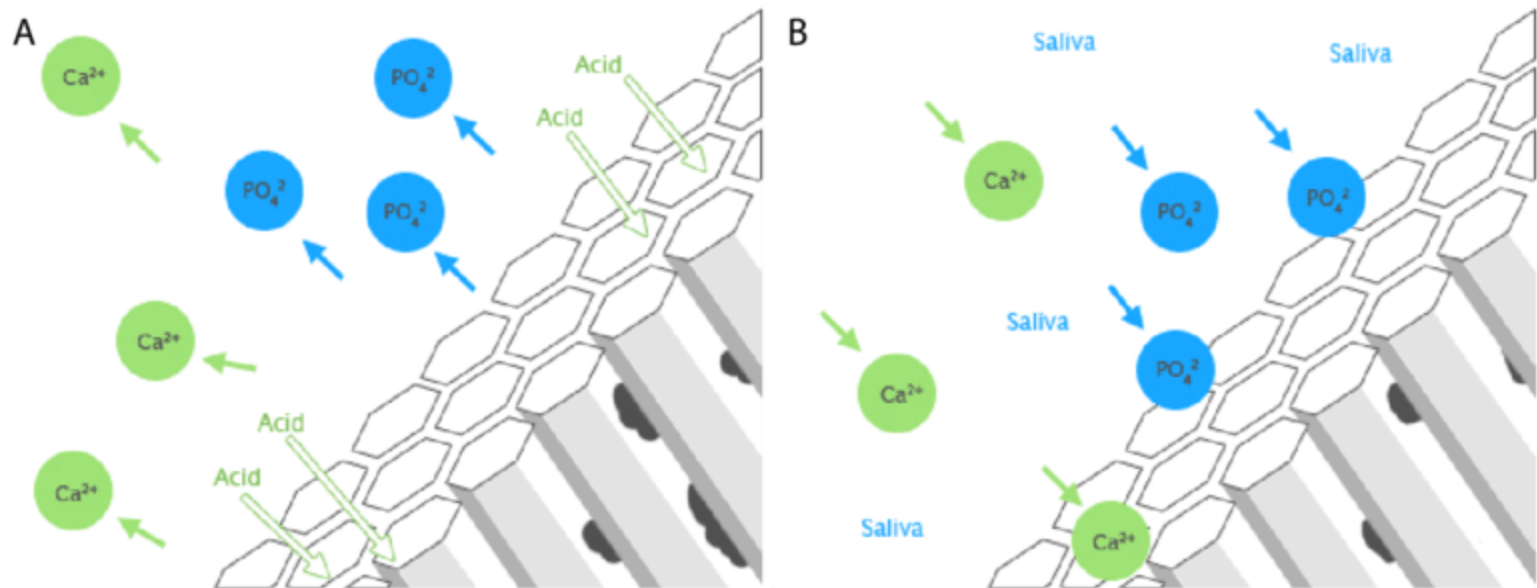
The organic acids produced by bacteria, if present in the biofilm for extended periods, can lower the pH in the biofilm to below a critical level (5.5 for enamel, 6.2 for dentin).

This low pH has effects both on the biofilm composition and at the tooth surface level , in turn will lead to further acidification of the environment.

Mechanism of demineralization and remineralization:

The low pH drives calcium and phosphate from the tooth to the biofilm in an attempt to reach equilibrium, hence resulting in a net loss of minerals by the tooth, or *demineralization*.

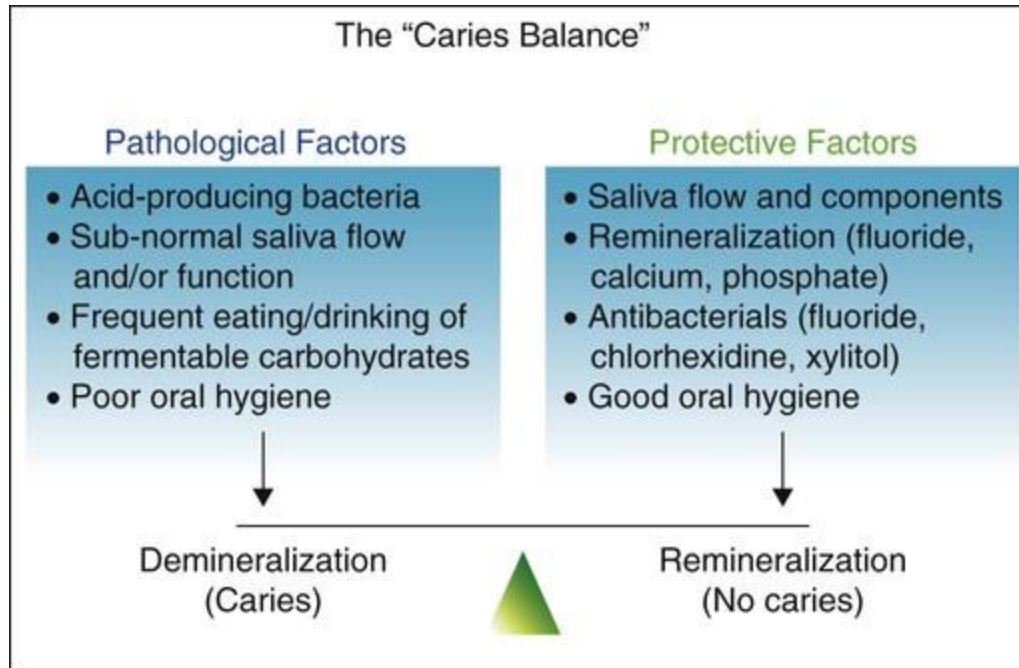
When the pH in the biofilm returns to neutral and the concentration of soluble calcium and phosphate is supersaturated relative to that in the tooth, mineral can then be added back to partially demineralized enamel in a process called *remineralization*.



Thus at the tooth surface and subsurface level, dental caries lesions result from a dynamic process of damage (demineralization) and restitution (remineralization) of the tooth matter.

These events take place several times a day over the life of the tooth and are modulated by many factors including number and type of microbial flora in the biofilm, diet, oral hygiene, genetics, dental anatomy, dentin and enamel composition, use of fluorides and other chemotherapeutic agents, saliva composition, salivary flow, and buffering capacity.





Understanding the balance between demineralization and remineralization is the key for caries management.

CARIES DIAGNOSIS

The process of caries diagnosis involves both risk assessment and the application of diagnostic criteria to determine the disease state.

*The primary objectives of caries diagnosis are to identify those lesions that **require surgical (restorative) treatment**, those that require non-surgical treatment, and those persons who are at high risk for developing carious lesions.*

Knowing which patients are at high risk for developing caries provides an opportunity to implement specific preventive strategies that may prevent caries. These strategies are specific to high-risk individuals and are not intended for all patients. However, for patients at low risk for caries, preventive measures may be limited to oral hygiene.

Assessment tools for caries diagnosis:

Patient history:

knowing factors through patient history can assist in caries diagnosis such factors include age, gender, fluoride exposure, smoking habits, alcohol intake, medications, dietary habits, economic and educational status, and general health (case sheet).

Clinical Examination or Visual examination:

General information regarding inadequate salivary functioning, plaque accumulation, inflammation of soft tissues, poor oral hygiene, cavitated lesions, and existing restorations also are instructive in determining potential risk to caries development (light source, mirror, and explorer). The tooth must be clean, dry and well illuminated when carrying out a visual examination.

Enhanced visual examination:

(Transillumination, Fibre-optic transillumination, Magnification, Dyes)

Radiographic Assessment:

Dental radiographs provide useful information in diagnosing carious lesions (interproximal caries).

Nutritional Analyses:

Frequent exposure to sucrose increases the likelihood of plaque development by the more cariogenic MS organisms.

Salivary Analyses:

Analyzing saliva may provide important information about appropriateness of secretion rates and buffering capacity as well as numbers of both MS and lactobacilli.

Caries can be classified according to **location, extent, and rate** .

Location :

(1) Pits and fissures of enamel,.

Which is the most susceptible site because the pits and fissures of newly erupted teeth are rapidly colonized by bacteria.

Pit-and-fissure caries expands as it penetrates into the enamel. Thus, the entry site may appear much smaller than the actual lesion, making clinical diagnosis difficult.

Detection of the lesion by explorer might be destructive method as the lesion could be treated conservatively through the remineralization process, however explorer might breakdown the dental tissue and create cavity that will act as harbor for dental plaque and debris that initiate caries progression. In addition the remineralization process will be more difficult to achieve in cavitated lesion than white spot lesion.

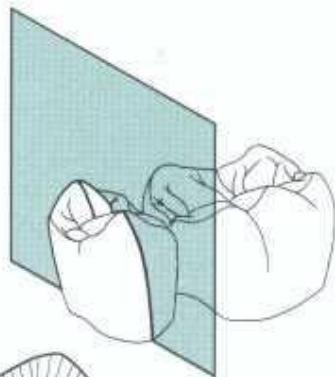
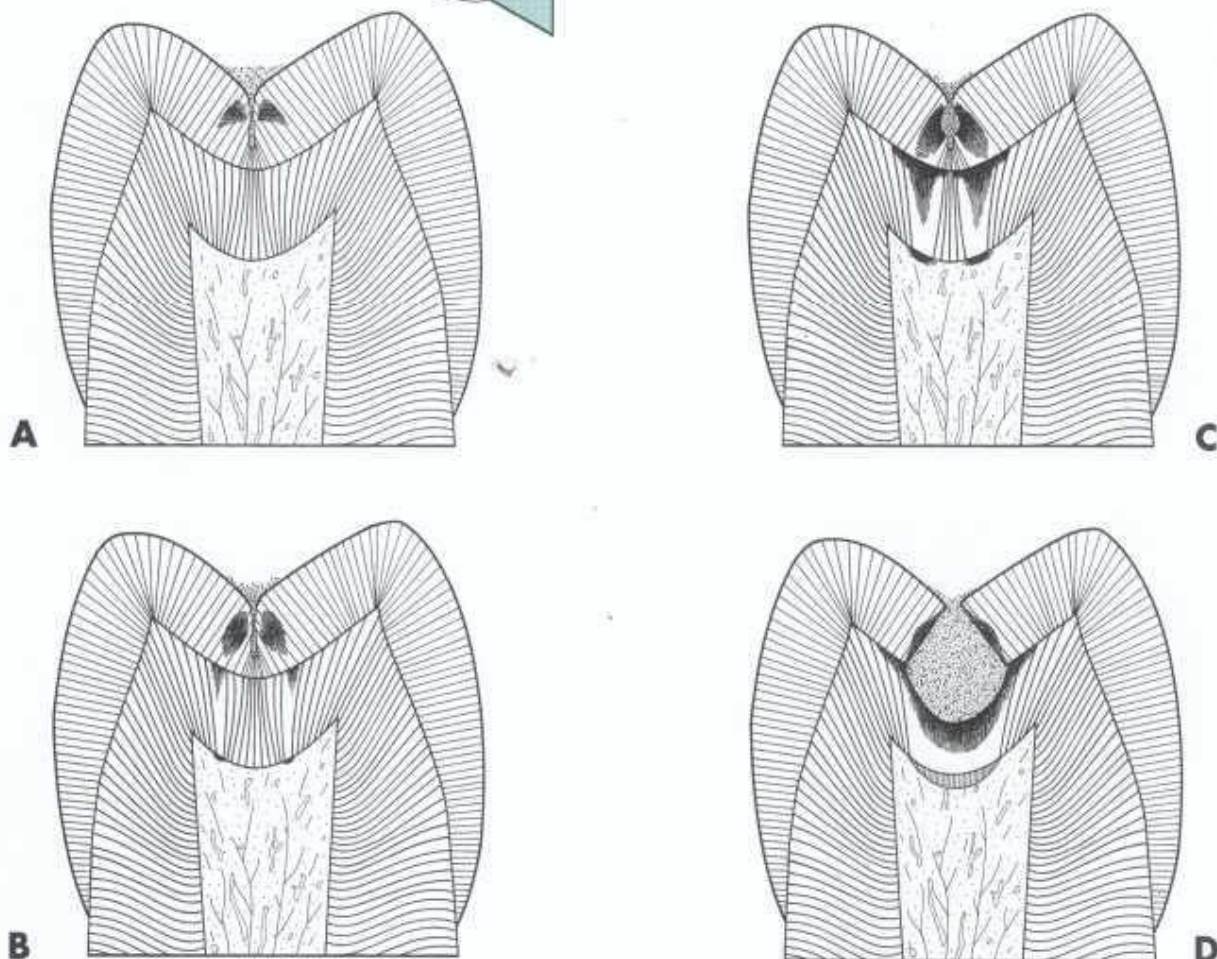


Fig 2-1a Occlusal caries. The shadowing around the stained pits in the second molar indicates the presence of carious dentin at the base of the fissure.



Progression of caries in pits and fissures.

A, The initial lesions develop on the lateral walls of the fissure. Demineralization follows the direction of the enamel rods

B, Forceful probing of the lesion at this stage can result in damage to the weakened porous enamel and accelerate the progression of the lesion.

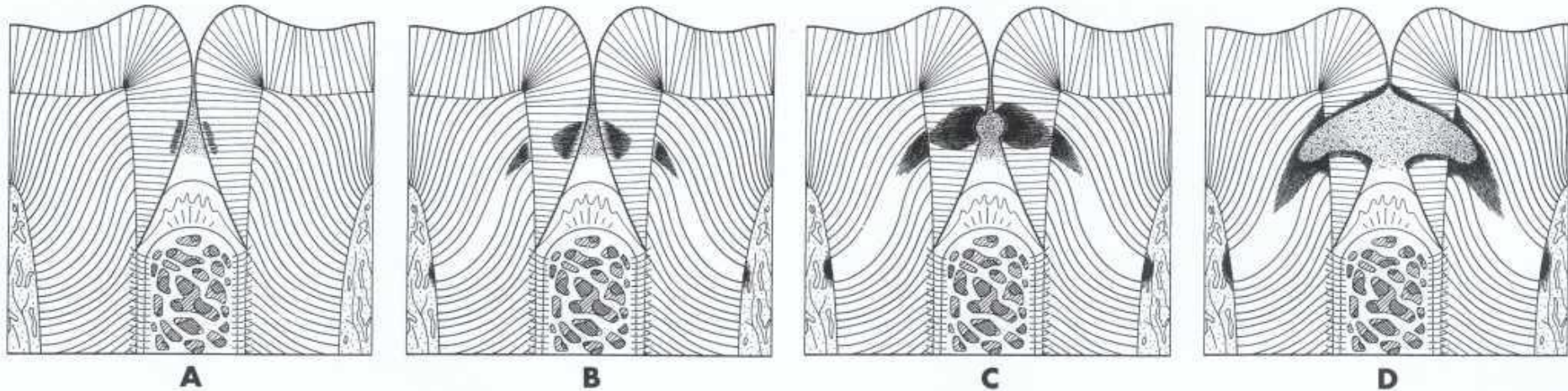
Clinical detection at this stage should be based on observation of discoloration and opacification of the enamel adjacent to the fissure. These changes can be observed by careful cleaning and drying of the fissure.

C, Initial cavitation of the opposing walls of the fissure cannot be seen on the occlusal surface.

D, Extensive cavitation of the dentin and undermining of the covering enamel will darken the occlusal surface

(2) Smooth enamel surfaces & Interproximal region:

The smooth enamel surfaces of the teeth present a less favorable site for plaque attachment. Lesions starting on smooth enamel surfaces have a broad area of origin and a conical, or pointed, extension toward the DEJ. Smooth surface lesion shows a V shape with a wide area of origin and the apex of the V directed toward the DEJ. The caries again spreads at this junction in the same manner as in pit-and-fissure caries. Thus, the apex of the cone of caries in the enamel contacts the base of the cone of caries in the dentin.



A, Initial demineralization on the proximal surfaces is not detectable clinically or radiographically.

B, When proximal caries first becomes detectable radiographically, the enamel surface is likely to still be intact. An intact surface is essential for successful remineralization and arrest of the lesion.

C, Cavitation of the enamel surface is a critical event in the caries process in proximal surfaces. Cavitation is an irreversible process and requires restorative treatment / correction of the damaged tooth surface. Cavitation can only be diagnosed by clinical observation. The use of a sharp explorer to detect cavitation is problematic because excessive force in application of the explorer tip during inspection of the proximal surfaces can damage weakened enamel and accelerate the caries process by creating cavitation.

D, Advanced cavitated lesions require prompt restorative intervention

What are the following terms mean ?
Backward Caries.
Forward Caries.
Residual Caries.

(3) Root surface.

Recently root caries get special attention due to the increase in the old age population. The cementum covering the root surface is extremely thin and provides little resistance to caries attack. Root caries lesions have less well defined margins, tend to be U-shaped in cross-section, and progress more rapidly because of the lack of protection from an enamel covering.

Characteristic of root caries :

1. rapidly progress.
2. a symptomatic
3. close to the pulp.
4. more difficult to restore.

Secondary (Recurrent) Caries. Secondary caries occurs at the junction of a restoration and the tooth and may progress under the restoration.

Extent of Caries

Incipient Caries (Reversible or remineralizable).

Incipient caries is the first evidence of caries activity in the enamel.

On smooth surface enamel, the lesion appears opaque white when air-dried, and will seem to disappear (not distinguishable from contiguous unaffected enamel) if wetted.

This lesion of demineralized enamel has not extended to the DEJ, and the enamel surface is fairly hard and still intact (smooth to the touch).

The lesion can be *remineralized* if immediate corrective measures alter the oral environment, including plaque removal and control.

Cavitated Caries (Nonreversible or non remineralizable).

In cavitated caries, the enamel surface is broken (not intact), and usually the lesion has advanced into dentin. Usually remineralization is not possible and treatment by tooth preparation and restoration is often indicated.

Rate (Speed) of Caries

Acute (Rampant) Caries.

Acute caries, often termed *rampant caries*, is when the disease is rapid in damaging the tooth. It is usually in the form of many, soft, light-colored lesions in a mouth and is infectious. Less time for extrinsic pigmentation explains the lighter coloration.

Chronic (Slow or Arrested) Caries.

Chronic caries is slow, or it may be *arrested* following several active phases. The slow rate results from periods when demineralized tooth structure is almost remineralized (the disease is episodic over time because of changes in the oral environment). The condition may be in only a few locations in a mouth, and the lesion is discolored and fairly hard. The slow rate of caries allows time for extrinsic pigmentation.

Progression of caries:

Decrease in caries progression have been reported recently possibly due to improvement in the preventive measures like using of fluoride.

The progression of the carious lesion depending on the site of origin (pit and fissure or smooth surface) and the conditions in the mouth (poor oral hygiene and frequent sucrose-containing food, dry mouth).

Early detection of caries lesion is important to take necessary preventive measures to arrest the progression and initiate the remineralization process, this will imply us to apply what is call (Minimum intervention)

Caries risk assessments :

During the initial history, examination and treatment planning for every patient, it is important to assess the risk of developing further carious lesions or progression of existing lesions. This procedure is termed ***caries risk assessment*** which is based on the following factors:

1• Caries experience

- the extent and number of previous restorations (indicator of past disease)
- the extent and number of new lesions
- the progression of new lesions.

2• Fluoride use – type and frequency. • Oral hygiene and the extent of plaque.

3• Dietary factors – eating habits, number of main meals, snacks, frequency of carbohydrate intake.

4• Bacterial activity – the presence and amount of cariogenic bacteria, specifically *Lactobacillus* and *Streptococcus mutans*.

5• Saliva – both the amount (quantity) and buffering capacity (quality).

6• Socio-economic status – to evaluate the patient for compliance. Caries tends to be a disease of deprivation and is more prevalent in patients with lower socio-economic status.

CARIES CONTROL

The treatment objective for caries control is to remove the decay from all of the advanced carious lesions, place appropriate pulpal medication, and restore the lesions in the most proper manner.

The caries control procedure allows quick removal of the caries, placement of a temporary restoration ,and the rescheduling of the patient for a more time consuming, permanent restoration. Before placement of a permanent restoration, a caries control procedure also provides a suitable delay that gives the pulp time to recover, allowing a better assessment of the pulpal status.

A caries control procedure is indicated when:

- (1) the caries is extensive enough that adverse pulpal sequelae are soon likely to occur,
- (2) the goal of treatment is to remove the nidus of caries infection in the patient's mouth, or
- (3) a tooth has extensive carious involvement that cannot or should not be permanently restored because of inadequate available time or questionable pulpal prognosis.

Usually all soft infected dentin should be removed, in a symptomatic deep soft dentin may be left and medication applied like calcium hydroxide (**Indirect pulp capping**) . The goals of the caries control procedure are to prevent pulp exposure and aid pulpal recovery by medication. Such repair usually occurs in 6 to 8 weeks and may be evident radiographically in 10 to 12 weeks.

* If pulp penetrated by instrument during operative procedure decision made with either **RCT** or **direct capping** to promote reparative dentin formation.

However if exposure is consequence of infected dentin extending into pulp termed carious pulpal exposure **RCT** indicated.

* Temporary restoration depend on remaining tooth structure.

MOISTURE CONTROL

(Rubber Dam, Saliva ejector, Aspiration, Cotton wool rolls, Matrix bands, Pharmacological).

The presence of oral fluids (saliva, blood, gingival crevicular fluid and water coolant spray) on the surface of a preparation is likely to:

- Dilute or displace etchant or bonding materials.
- Impair the creation of a bond between tooth and restoration.
- Interfere with cohesion of successive increments of restorative material.
- React with restorative material and thus impair its strength or dimensional stability, e.g. with zinc containing amalgams leading to porosity and expansion.
- Discolour tooth-coloured resin restorations, e.g. with blood contamination.
- Prevent the creation of a marginal seal where a cement lute is employed, e.g. for an indirect restoration.
- Contaminate a site that should preferably have as low a bacterial load as possible, e.g. pulp exposures and root canal therapy.

For these reasons it is necessary to isolate a preparation from moisture, especially when placing restorative materials and undertaking endodontic therapy.





Enamel and Dentin Caries (Histopathology overview)

(CH3 in 4th edition)

By

Assistant Prof. Dr. Maan M. Nayif

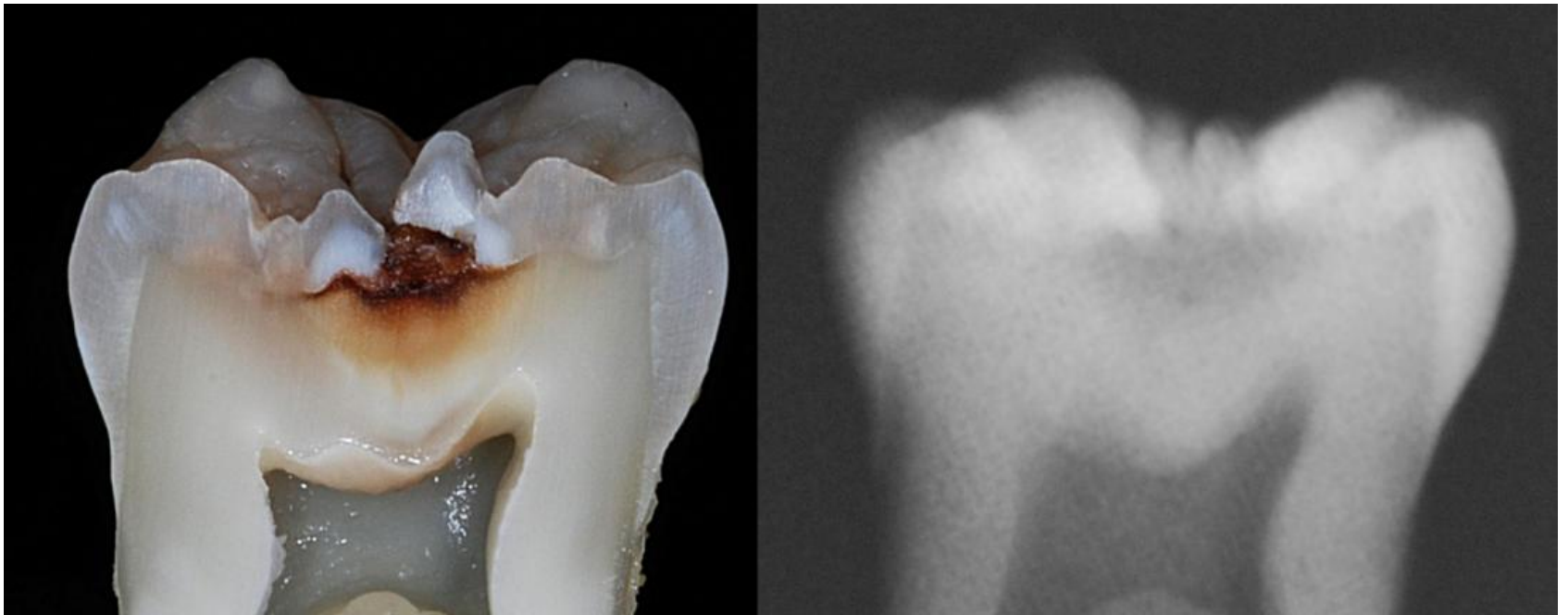
**PhD in Restorative Dentistry, Tokyo Medical
and Dental University, Japan**

Enamel caries

Histology of enamel

Clinical characteristic of enamel caries

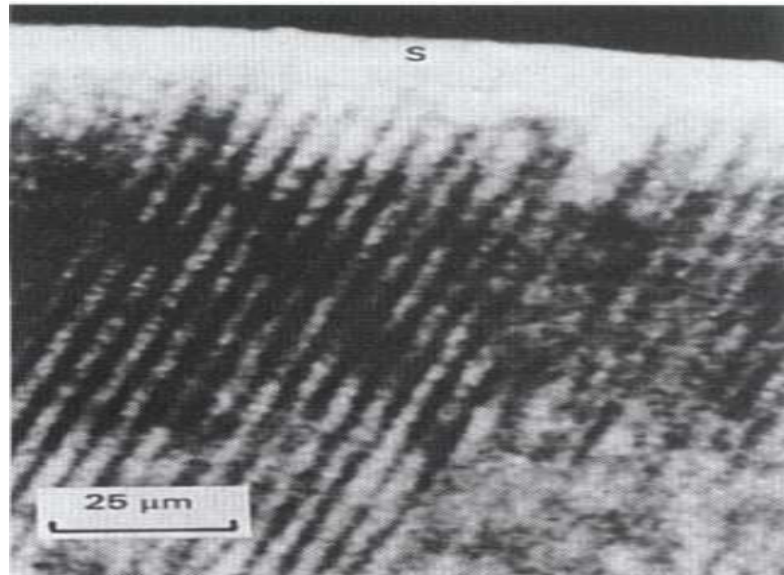
Zones of the incipient lesion



Histology of enamel:

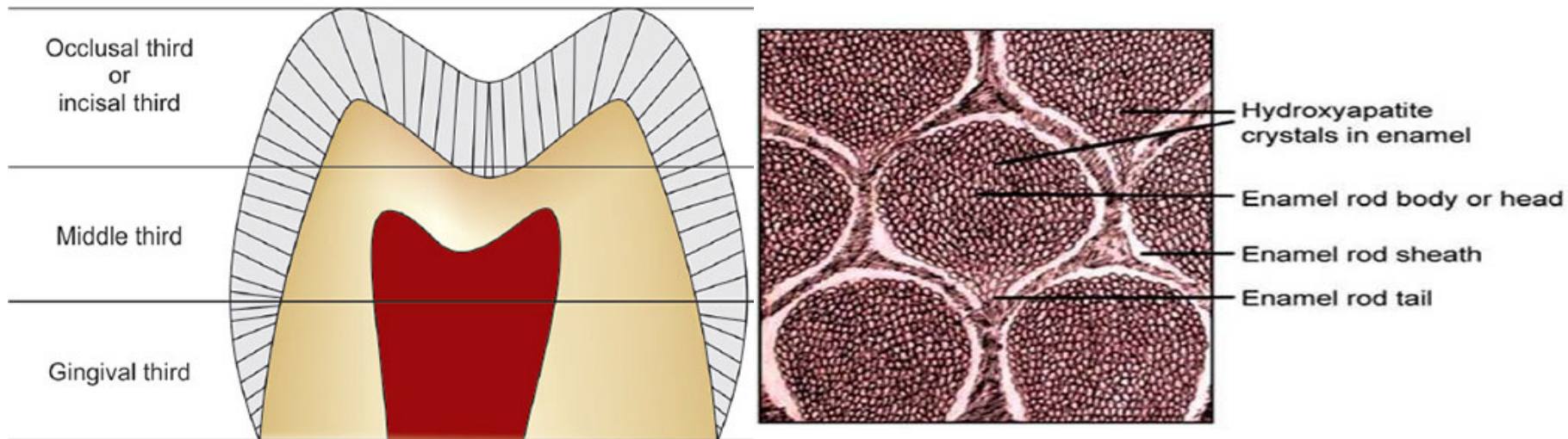
Enamel is composed of very tightly packed hydroxyapatite crystallites, organized into long columnar rods (prisms). The rods are somewhat key-shaped in cross-section.

Both the striae of Retzius and the inherent spaces in prism boundaries provide sufficient porosity to allow movement of water and small ions, such as hydrogen ions.



Microradiograph of cross-section of small carious lesion in enamel. Well-mineralized surface (s) is evident. Alternating radiolucent and radiopaque lines indicate demineralization between enamel rods.

Structurally, enamel is composed of millions of enamel rods (or “prisms”), rod sheaths, and a cementing inter-rod substance. Rods vary in number from approximately 5 million for a mandibular incisor to about 12 million for a maxillary molar.



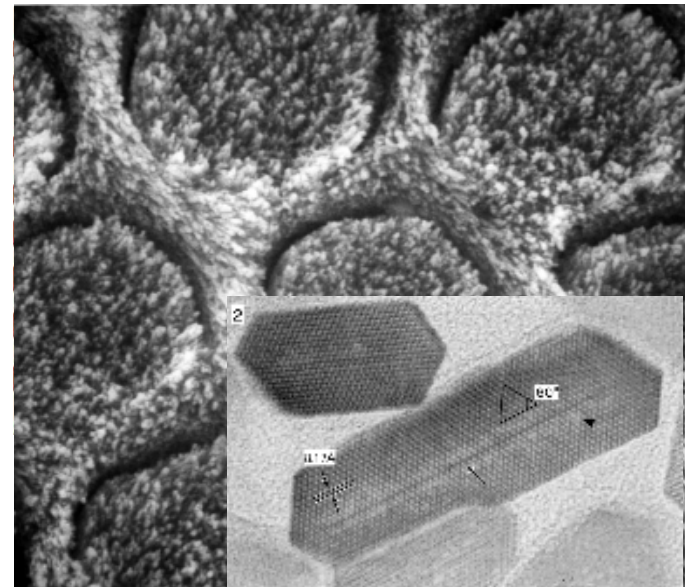
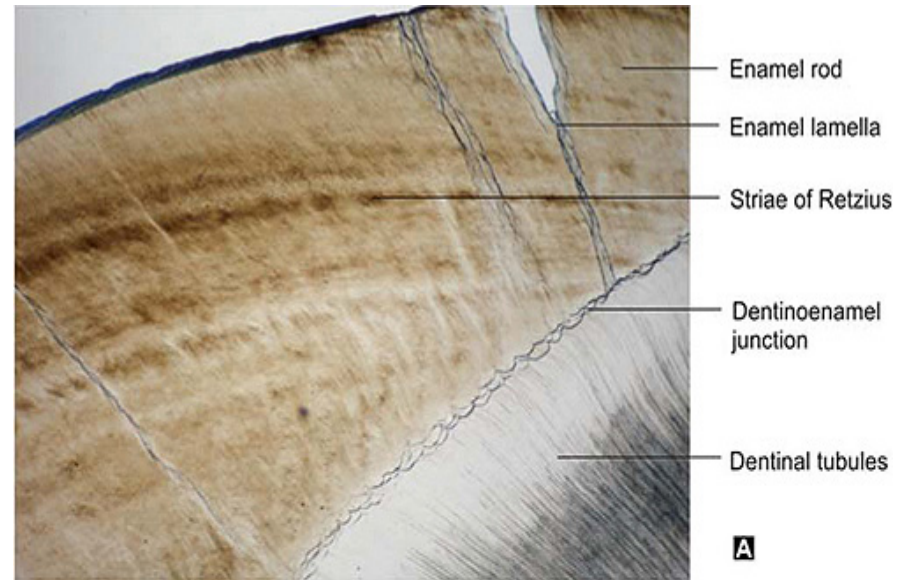
Orientation of rods In general, the rods are aligned perpendicularly to the DEJ and the tooth surface except in the cervical region of permanent teeth, where they are oriented outward in a slightly apical direction. Starting at 1mm from the CEJ, the rods on the vertical surfaces run occlusally or incisally at approximately a 60 degrees inclination and progressively incline approaching the marginal ridges and cusp tips, where the rods are parallel to the long axis of the crown. The rods beneath the occlusal fissures are also parallel to the log axis, but rods on each side of the fissure vary up to 20 degrees from the long axis.

Chemically, enamel is a highly mineralized crystalline structure of hydroxyapatite vary in size and shape in the form of a crystalline with mineral constituent (90%–92% by volume). Other minerals and trace elements are present in smaller amounts.

The remaining constituents of tooth enamel include organic matrix proteins (1%–2%) and water (4%–12%) by volume.

The crystallites are tightly packed in a distinct pattern of orientation that gives strength and structural identity to the enamel rod.

Enamel lamellae are thin, leaf-like faults between the enamel rod groups that extend from the enamel surface toward the DEJ, sometimes extending into dentin. **They contain mostly organic material and may predispose the tooth to the entry of bacteria and subsequent development of dental**



Properties of Enamel:

1. *Hardness*

Enamel is the hardest substance of the human body.

Hardness may vary over the external tooth surface according to the location; also, it decreases inward, with hardness lowest at the DEJ.

The density of enamel also decreases from the surface to the DEJ.

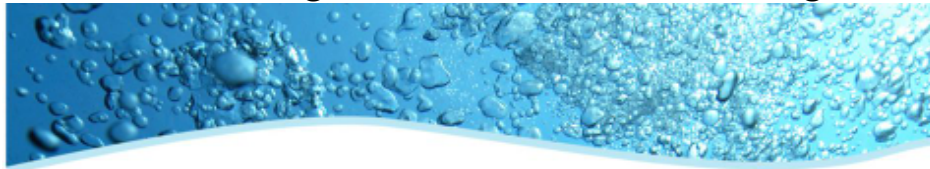
It is as hard as steel with a Knoop Hardness Number (KHN) of 343 (compared to 68 for dentin). However, enamel will wear because of attrition or frictional contact against opposing enamel or harder restorative materials such as porcelain.

2. **Brittleness**

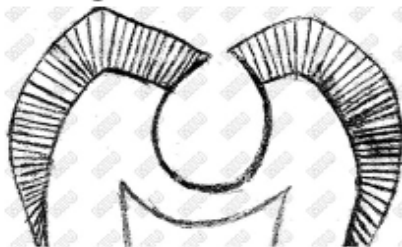
Enamel is very brittle (Tensile strength of enamel rods is low as 1.25 Mpa); thus it requires a supporting base of dentin to withstand the masticatory stress.

Enamel rods that fail to possess a dentin base because of caries or improper cavity design are easily fractured away from neighboring rods.

Chipping of enamel rods at the cavo-surface margin of restored cavity creates a gap defect, leakage of bacteria and their products that may lead to secondary caries. Therefore, a basic principle of cavity wall preparation is to cut enamel parallel the direction of enamel rods or with beveling and avoid undercutting them.



2. **All unsupported or undermined enamel** must be eliminated; otherwise, it will break easily under mastication forces leaving a marginal “ditch”



3. *Permeability*

Although enamel is a hard, dense structure, it is permeable to certain ions and molecules. The route of passage may be through structural units such as rod sheaths, enamel cracks, and other defects that are hypomineralized and rich in organic content. Water plays an important role as a transporting medium through the small intercrystalline spaces. Enamel tufts are hypomineralized structures of inter-rod substance between adjacent groups of enamel rods that project from the DEJ .

Enamel permeability decreases with age because of changes in the enamel matrix, a decrease referred to as *enamel maturation*. The micro-pores form a dynamic connection between the oral cavity and the pulpal and dentinal tubule fluids.

Various fluids, ions and low molecular weight substance can diffuse through the semipermeable enamel. Therefore, the dynamics of acid demineralization, caries, remineralization, fluoride uptake are not limited to the surface but are active in three dimensions.

4. *Solubility to acids*

Enamel is soluble when exposed to acid medium, although the dissolution is not uniform. Solubility of enamel increases from the enamel surface to the dentinoenamel junction.

Role of fluoride in enamel solubility:

When fluorides are present during enamel formation or are topically applied to the enamel surface, the solubility of surface enamel is decreased.

Fluoride additions can affect the chemical and physical properties of the apatite mineral and influence the hardness, chemical reactivity, and stability of enamel while preserving the apatite structures. Trace amounts of fluoride stabilize the enamel by lowering acid solubility, by decreasing the rate of demineralization, and by enhancing the rate of remineralization.

Enamel acid etching:

Acid-etching is a technique used for modification of enamel for improving adhesive restoration retention. This will provides a conservative, reliable, alternative to traditional methods of tooth preparation and restorations (retentive grooves, pins, extension for prevention...).

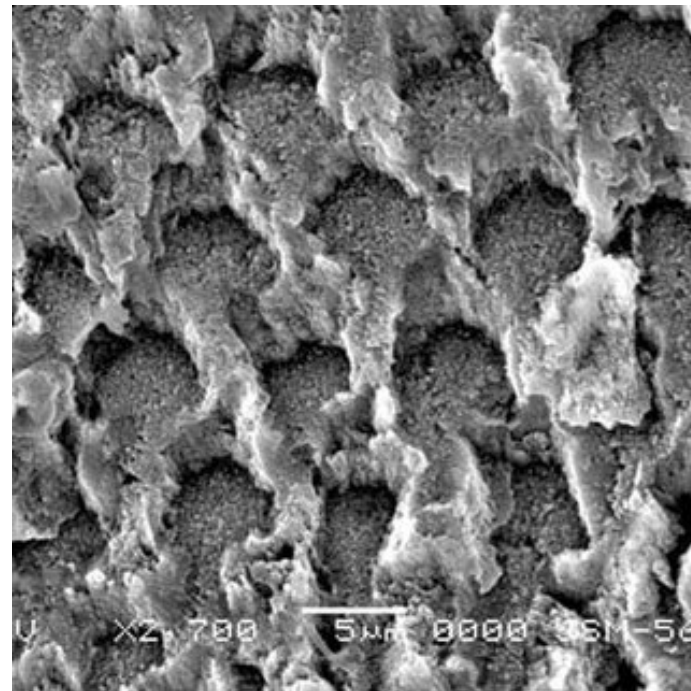
The susceptibility of these crystallites (HAp) to acidic conditions, from the caries process or as a result of an etching procedure, may be correlated with their orientation.

Acid-induced mineral dissolution (demineralization) occurs more in the head region of each rod. The tail region and the periphery of the head region are relatively resistant to acidic demineralization.



Acid etching remove about 10 micron of enamel surface which dissolve either rod core or periphery to form three dimension irregular and pitted surface with numerous microscopic undercuts of about 20 micron in depth.

The etched enamel has a higher surface energy, so resin monomer flows into and adheres to the etched depressions to polymerize and form retentive resin tags.



5. Color:

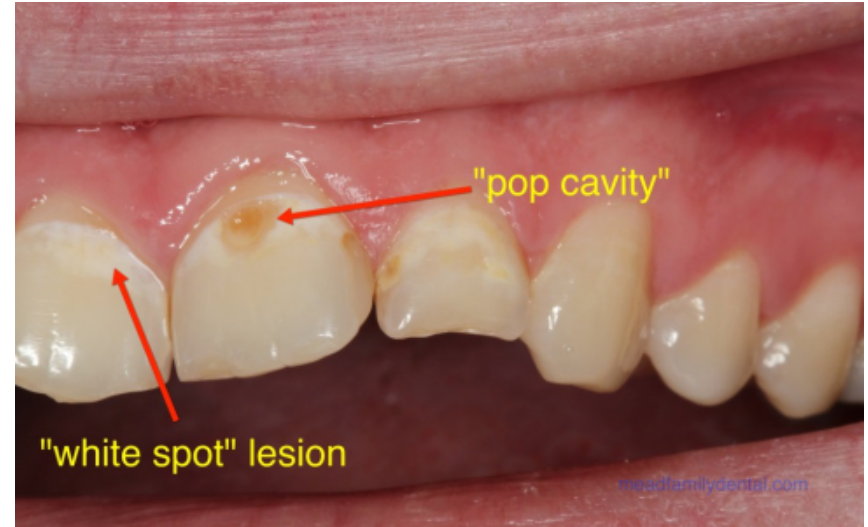
Enamel is relatively translucent. The amount of translucency of enamel is related to the variation in the degree of calcification and homogeneity. It has a glossy surface and varies in color from light yellow to grayish white. Therefore, the color of the tooth is primarily a function of the color of underlying dentin, enamel thickness and the amount of the stain in the enamel.

The thickness of enamel is greater at the cusps tips (2.5mm) and incisal edges (2mm) and decreases below deep fissures and become thin at the junction with cementum.

Enamel becomes temporary whiter within minutes when the tooth is isolated from the moist oral environment by rubber dam or absorbents. Thus, the shade must be determined before isolation and preparation for a tooth - colored restoration.



Care must be exercised to distinguish white spots of incipient caries from developmental white spot hypocalcifications of enamel .



Incipient caries will partially or totally disappear visually when the enamel is hydrated (wet), while hypocalcified enamel is relatively unaffected by drying and wetting.

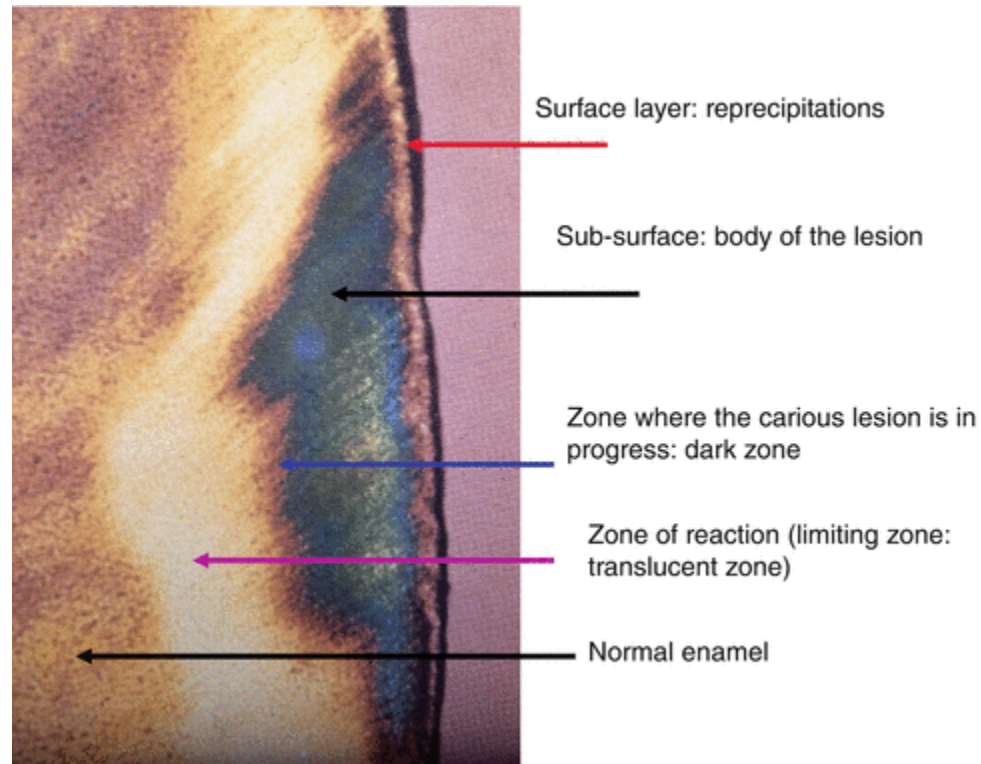
It has been shown that incipient caries of enamel can remineralize.

Calcium and phosphate ions from saliva can then penetrate the enamel surface and precipitate in the enamel lesion.

Remineralized (arrested) lesions can be observed clinically as intact, but discolored, usually brown or black spots **These discolored, remineralized, arrested caries areas are** intact and are more resistant to subsequent caries attack than the adjacent unaffected enamel.

Zones of the enamel lesion :

- (1) translucent zone.
- (2) dark zone.
- (3) body of the lesion.
- (4) surface zone.



Dentinal caries

Histology of dentin

Clinical and histological characteristic of dentinal caries

Zones of the lesion



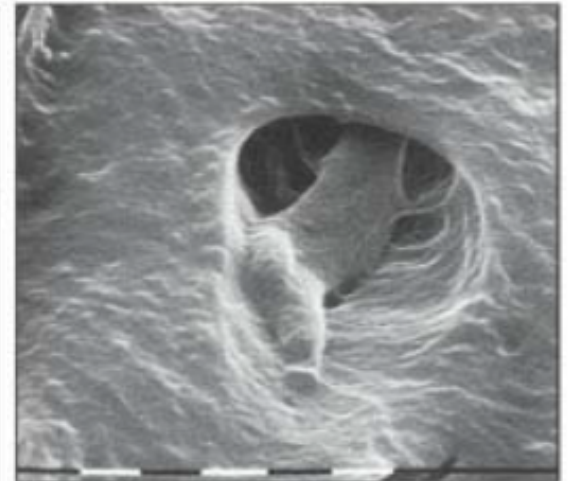
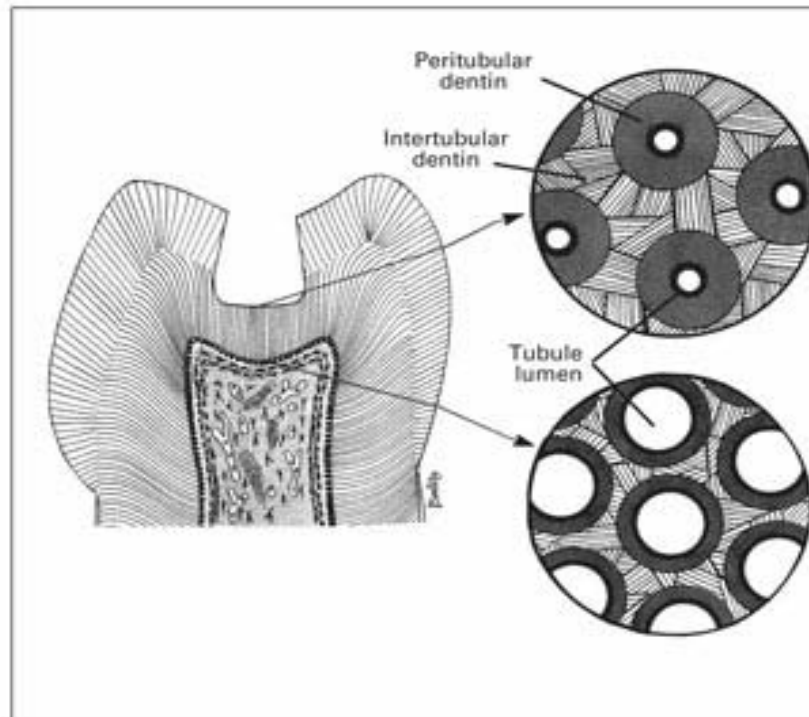
Dentin formation (Dentinogenesis):

is accomplished by cells called **Odontoblasts** that are considered as a part of pulp and dentin tissues because their cell bodies are in the pulp cavity, but their long, slender cytoplasmic cell processes (Tomes fibers) extend well (100–200 μm) into the tubules in the mineralized dentin.

In contrast to enamel formation, dentin formation continues after tooth eruption and throughout the life of the pulp.

The course of the dentinal tubules is a slight S-curve in the tooth crown, but the tubules are straighter in the incisal ridges, cusps, and root areas . Tubules are generally oriented perpendicular to the DEJ.

Human dentin is composed of approximately 45% - 50% inorganic material and 30% organic material and 25% water



Dentin is less mineralized than enamel but more mineralized than cementum or bone.

The mineral content of dentin increases with age composed primarily of hydroxyapatite crystallites (Calcium hydroxyapatite with the chemical formula of $\text{Ca}_{10}(\text{PO}_4)_6(\text{OH})_2$), which are arranged randomly in a less systematic manner in organic matrix than are enamel crystallites. The crystals in dentin are plate-like in shape and 30% smaller in size than those in enamel. Small amounts of other minerals, such as carbonate and fluoride, are also present.

Properties of Dentin

1. Hardness

The hardness of dentin is one-fifth that of enamel. Its hardness at DEJ is 3 times more than near pulp.

Hardness of dentin also increases with advancing age due to continuation of the mineralization.

2. Strength

Tooth strength, rigidity, and integrity rely on an intact substrate. Dentin has a tensile strength of approximately 40MPa and a compressive strength of 266MPa .

3. Elastic modulus

Low modulus of elasticity of dentin (18 GPa) makes it flexible in nature. This flexibility provides support or cushion to the brittle enamel.

4. Permeability

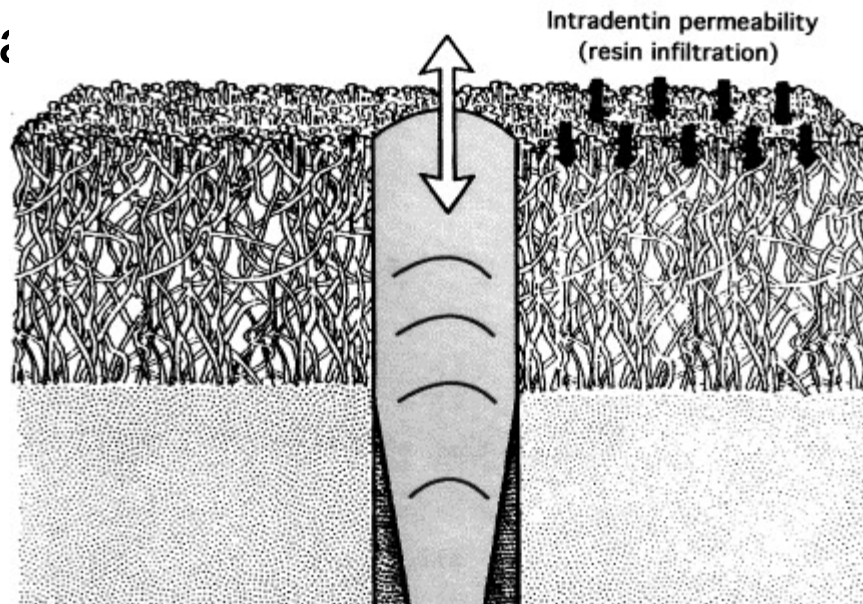
Unlike enamel which is semipermeable (provide an effective layer serving to protect the underlying dentin.), dentin is a permeable structure and its permeability primarily depends on the remaining dentin thickness (i. e., length of the tubules and the diameter of the tubules).

The diffusion gradient is also affected by the intratubular cellular, collagenous, and mineral inclusions

Transdentin Permeability (hydrodynamic fluid flux)

Intradentin permeability (resin infiltration)

tubular channels



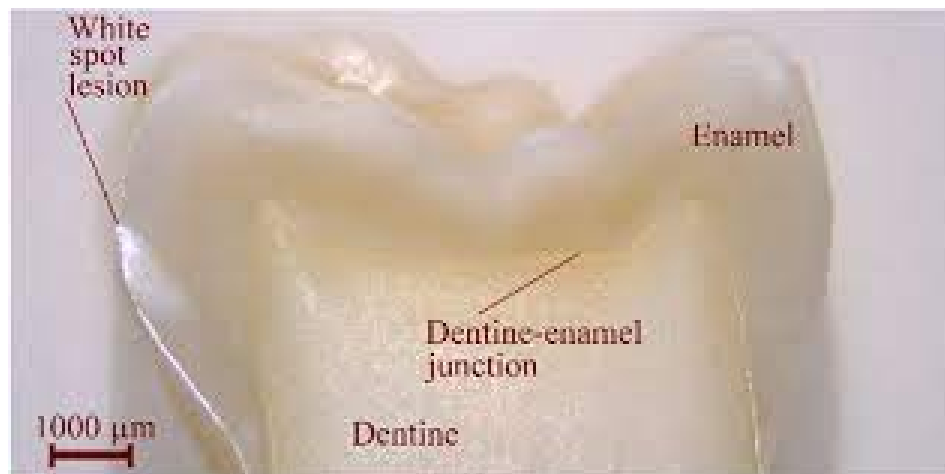
Dentin can be distinguished from enamel (during tooth preparation) by:

1. Color: dentin is normally yellow-white and slightly darker than enamel, in older patients dentin is darker and become brown or black in cases in which it has been exposed to oral fluids, old restorative materials or slowly advancing caries.

2. Reflectance: dentin surfaces are more opaque and dull, being less reflective to light than enamel surfaces, which appear shiny.

3. Hardness: dentin is softer than enamel, sharp explorer tends to catch and hold in dentin.

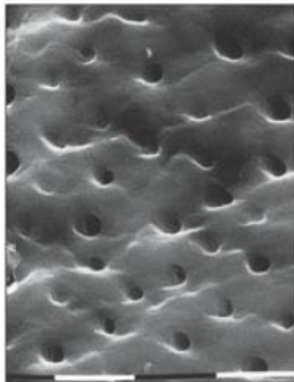
4. Sound: when moving an explorer tip over the tooth, enamel surfaces provide a sharper, higher pitched sound than dentin surfaces.



According to location dentin can be divided into:

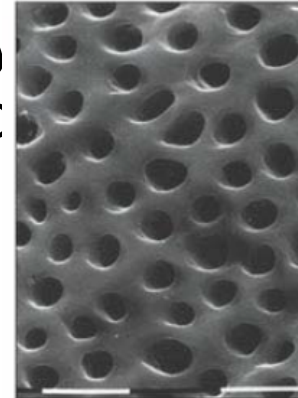
Outer Dentin:

- Close to DEJ , dentinal tubules are small in diameter, with much larger surface area near the DEJ and the Intertubular dentin makes up 96% of the surface



Inner Dentin:

- Close to the pulp, tubules diameters are larger and the distance between tubule centers is half that of tubules at DEJ. Thus, the intertubular matrix area is only a small fraction of the surface

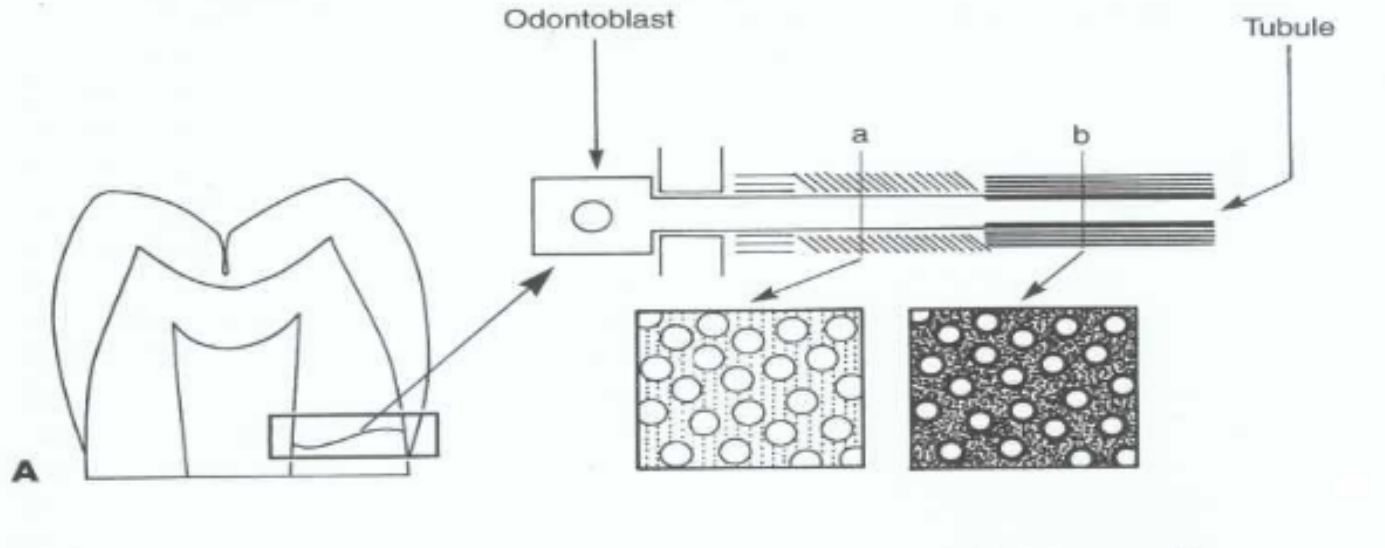


Permeability of Dentin

The permeability of dentin is directly related to its protective function. Whenever the external cap of enamel and cementum is lost from the periphery of the dentinal tubules through dental caries or during preparation with burs or abrasion and erosion, the exposed tubules become conduits or path between the pulp and the external oral environment.

Smear layer formed after cavity preparation.

Histology of dentin:



Normal dentin

(A) has characteristic tubules that follow a wavy path from the external surface of the dentin, and grows inward. The more recently formed dentin near the pulp (a) has both large tubules with little or no peritubular dentin and calcified intertubular dentin filled with collagen fibers. The older dentin, closer to the external surface (b), is characterized by smaller, more widely separated tubules and a greater mineral content in the intertubular dentin.

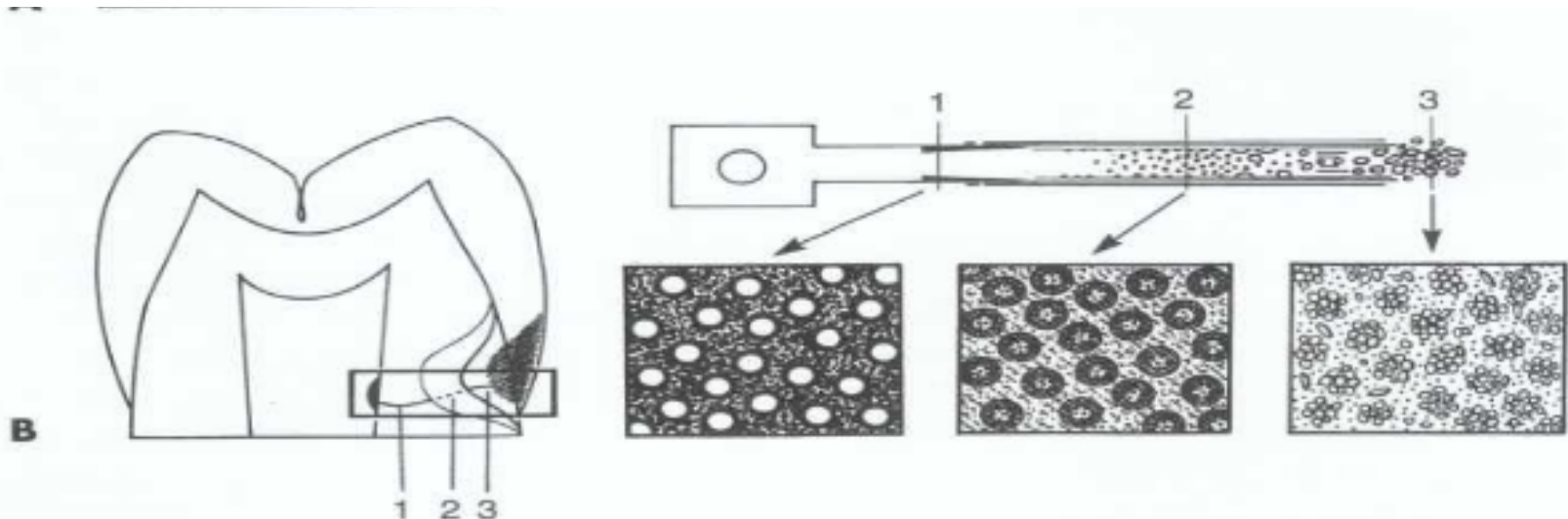
Horizontal lines indicate predentin; diagonal lines indicate increasing density of minerals; darker horizontal lines indicate densely mineralized dentin and increased thickness of peritubular dentin.

Carious dentin (B)

The most superficial infected zone of carious dentin (3) is characterized by bacteria filling the tubules, and granular material in the intertubular space.

Pulpal to (below) the infected dentin is a zone where the dentin appears transparent in mounted whole specimens. This zone (2) is affected (not infected) carious *dentin* and is characterized by loss of mineral in the intertubular and peritubular dentin. Many crystals can be detected in the lumen of the tubules in this zone.

Normal dentin (1) is found pulpal to (below) the transparent dentin.



Characteristic of dentin caries:

Progression of caries in dentin is different from progression of caries enamel.

- Dentin contains much less mineral and possesses microscopic tubules that provide a pathway for the ingress of acids and egress of mineral.
- The dentinoenamel junction (DEJ) has the least resistance to caries attack and allows rapid lateral spreading once caries has penetrated the enamel. Because of these characteristics, dentinal caries is V-shaped in cross-section with a wide base at the DEJ and the apex directed pulpally.
- Caries advances more rapidly in dentin than in enamel because dentin provides much less resistance to acid attack because of less mineralized content.
- Caries produces a variety of responses in dentin, including pain, demineralization, and remineralization.

-Once bacterial invasion of the dentin is close to the pulp, toxins and possibly even a few bacteria enter the pulp, resulting in inflammation of the pulpal tissues.

-The pulp-dentin complex reacts to caries attacks by attempting to initiate remineralization and blocking off the open tubules. These reactions result from odontoblastic activity and the physical process of demineralization and remineralization.

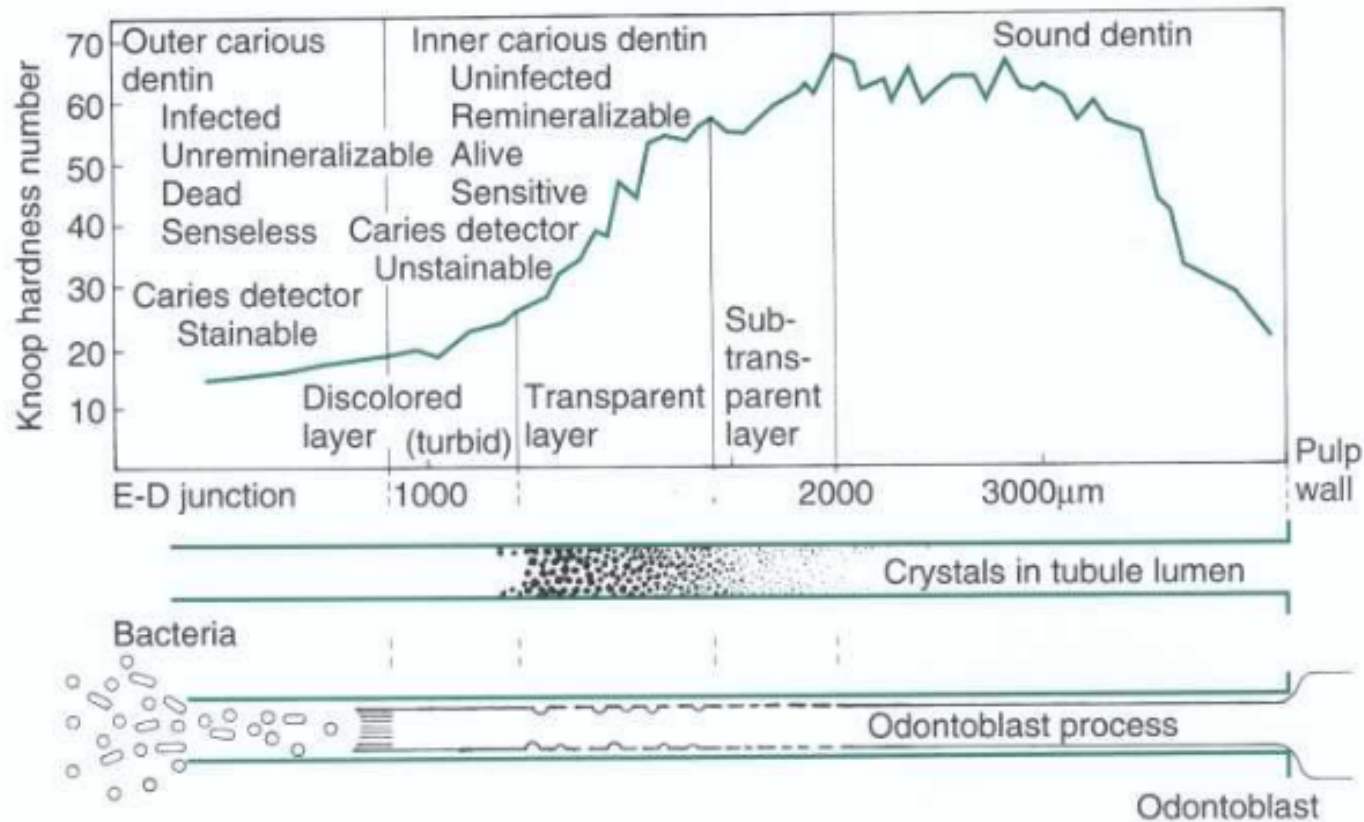
-Dentin responds to the stimulus of its first caries demineralization episode by deposition of crystalline material in both the lumen of the tubules and the intertubular dentin of affected dentin.

Hypermineralized areas may be seen on radiographs as zones of increased radiopacity (often S-shaped following the course of the tubules) ahead of the advancing, infected portion of the lesion.

This repair only occurs if the tooth pulp is vital.

Zones of the dentin lesion:

- Zone 1 normal dentin** (no bacteria)
- Zone 2 sub-transparent dentin** (no bacteria, demineralized zone, and remineralizable)
- Zone 3 transparent dentin** (no bacteria ,crystals in tubule, also remineralizable)
- Zone 4 turbid dentin** (bacterial invasion, not remineralizable)
- Zone 5 infected dentin** (bacterial invasion, decomposed dentin, no mineral or collagen\`)



Advanced carious lesion:

Demineralization of the body of the enamel lesion results in the weakening and finally collapse of the surface covering and resulted cavity will provide retentive region for biofilm thus will accelerating the caries lesion progression.

The DEJ is a weak area with less resistance to the carious process than either the enamel or the dentin individually. The resultant lateral spread of the lesion at the DEJ produces the characteristic second cone of caries activity in the dentin.

Necrotic dentin is recognized clinically as a wet, mushy, easily removable mass.

The leathery dentin below necrotic dentin is easily removed by hand instruments

Removal of the bacterial infection is an essential part of all operative procedures. it is not necessary to remove all the dentin that has been affected by the caries process.

it is not necessary to remove all the dentin that has been affected by the caries process. In operative procedures, it is convenient to term dentin as either infected, and thus requires removal, or affected, which does not require removal.

Affected dentin is softened, demineralized dentin that is not yet invaded by bacteria (zones 2 and 3).

Infected dentin (zones 4 and 5) is both softened and contaminated with bacteria.

The outer layer (infected dentin) can be selectively stained in vivo by caries detection solutions.

This solution stains the irreversibly denatured collagen in the outer carious layer.



In a tooth with a deep carious lesion, no history of spontaneous pain, normal responses to thermal stimuli, and a vital pulp (demonstrated by electric testing), a deliberate, incomplete caries excavation may be indicated.

This procedure is termed **indirect pulp capping** and is characterized by placement of a thin layer of calcium hydroxide on the questionable dentin remaining over the pulp.

A **direct pulp cap** is the placement of calcium hydroxide directly on exposed pulpal tissue (a pulpal exposure) and the surrounding deeply excavated dentinal area.

The techniques of indirect and direct pulp capping may stimulate the formation of reparative dentin.







Dentin and pulp reaction to caries and operative procedure

(CH3 in 4th edition)

By

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DENTIN THICKNESS

- We must remember that no material can provide better protection for the pulp than dentin.
- The remaining dentin thickness, from the depth of cavity preparation to the pulp, is the most important factor in protecting the pulp from insult.

Remaining dentin thickness

- The studies have shown that 0.5 mm thickness of dentin reduces the effect of toxic substances on the pulp by 75%, 1.0mm thickness of dentin reduces the effect of toxins by 90%, little if any pulpal reaction occurs when there is a remaining dentinal thickness of 2mm or more
- ▮ It helps us to make a decision concerning the use of bases and liners.

Three levels of dentinal reaction to caries :

(1) Reaction to low intensity, slowly advancing caries lesion ;a long-term, low-level of acid or caries:

Direct exposure of the pulp tissue to microorganisms , toxins and other metabolic by-products, especially hydrogen ion, can penetrate via the dentinal tubules to the pulp.

Early stages of caries or mild caries attacks produce long-term, low-level acid demineralization of dentin.

In slowly advancing caries, a vital pulp can repair demineralized dentin by remineralization of the intertubular dentin and by apposition of peritubular dentin.

Dentin responds to the stimulus of its first caries demineralization episode by deposition of crystalline material in both the [lumen of the tubules](#) and [the intertubular dentin](#) of affected dentin in front of the advancing infected dentin portion of the lesion.

Hypermineralized areas may be seen on radiographs as zones of increased radiopacity (often S-shaped following the course of the tubules) ahead of the advancing, infected portion of the lesion.

This repair only occurs if the tooth pulp is vital.

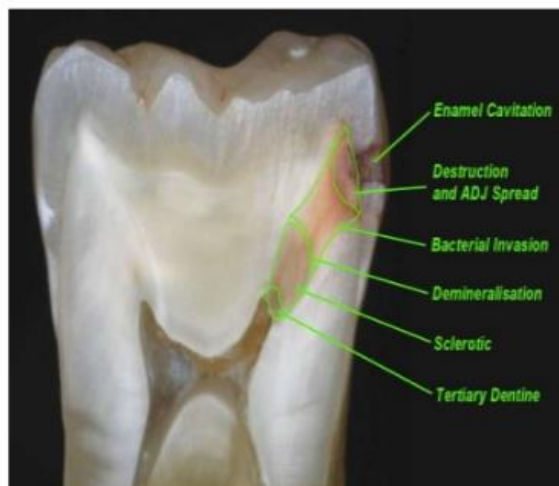
Dentin that has more mineral content than normal dentin is termed *sclerotic dentin*.

Sclerotic dentin formation occurs ahead of the demineralization front of a slowly advancing lesion and may be seen under an [old restoration](#).

Sclerotic dentin is usually shiny and more darkly colored, but feels hard to the explorer tip. By comparison, normal freshly cut dentin lacks a shiny, reflective surface and allows some penetration from a sharp explorer tip. **The apparent function of sclerotic dentin is to wall off a lesion by blocking (sealing) the tubules.**

The permeability of sclerotic dentin is greatly reduced in comparison to normal dentin because of the decrease in the tubule lumen diameter . Therefore it may be more difficult to bond a restorative material to sclerotic dentin.

SCLEROTIC DENTINE



Sclerosis is the result of **occlusion of the dentinal tubules** by a mineral substance with a refractive index similar to that of the rest of the dentine

Increases with age

(2) Reaction to a moderate-intensity caries ;

The second level of dentinal response is to moderate intensity (or intermediate) irritants.

More intense caries activity results in **bacterial invasion** of the dentin.

The infected dentin contains a wide variety of pathogenic materials or irritants, including **high acid levels, hydrolytic enzymes, bacteria, and bacterial cellular debris**. These materials can cause the degeneration and death of the odontoblasts and their tubular extensions below the lesion, as well as a mild inflammation of the pulp.

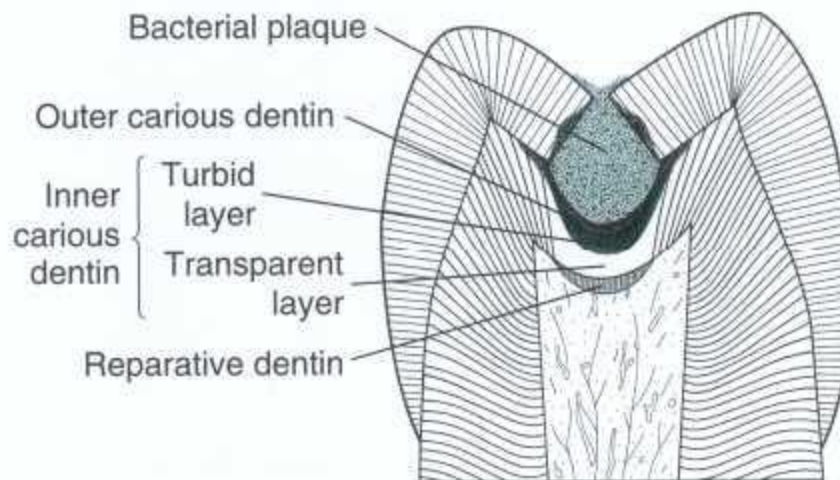
Groups of these dead, empty tubules are termed **dead tracts**.

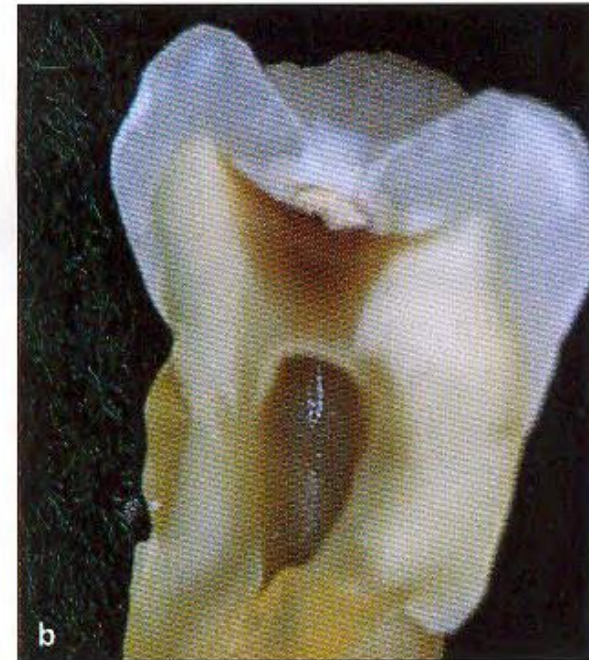
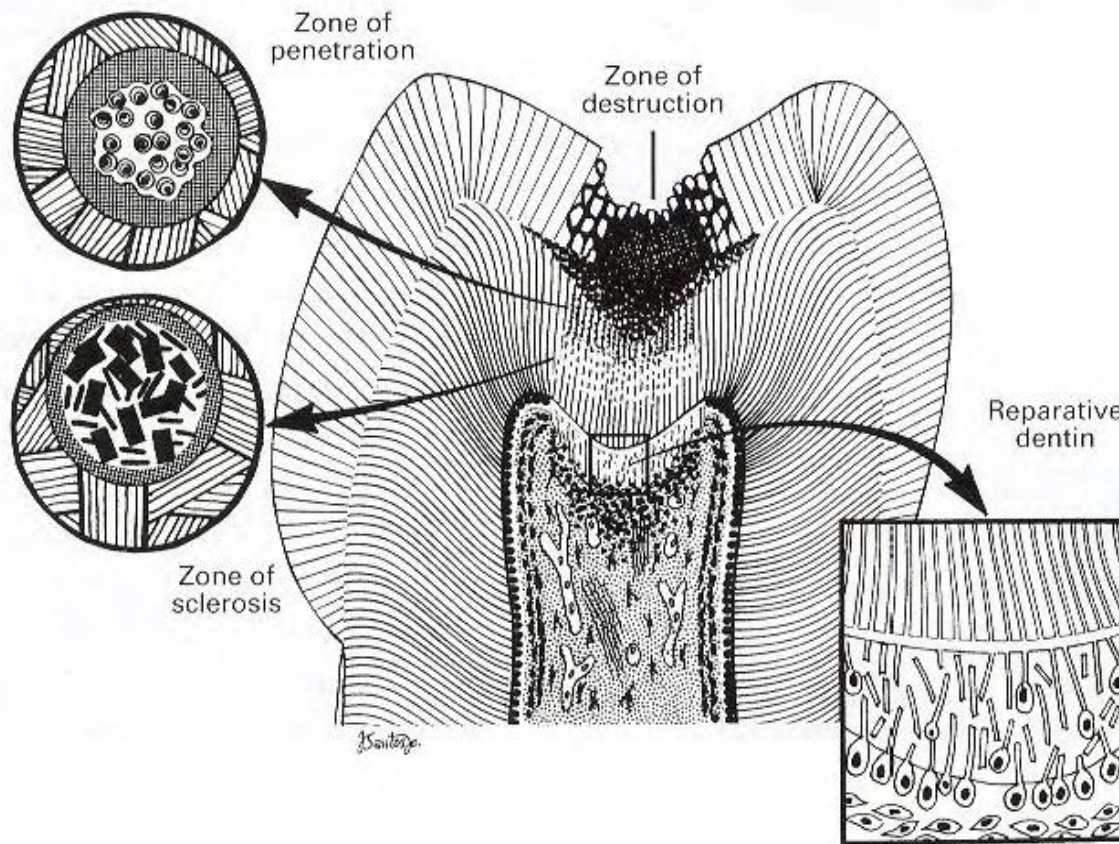
The pulp may be irritated sufficiently from high acid levels or bacterial enzyme production to cause the formation (from undifferentiated mesenchymal cells) of replacement odontoblasts (secondary odontoblasts).

These cells produce reparative dentin (reactionary dentin) on the affected portion of the pulp chamber wall .

This dentin (reparative) is **different** from the normal dentinal apposition that occurs throughout the life of the tooth by primary (original) odontoblasts. The structure of reparative dentin can vary from well-organized tubular dentin (less often) to very irregular a tubular dentin (more often), depending on the severity of the stimulus.

Reparative dentin is a very effective barrier to diffusion of material through the tubules and is an important step in the repair of dentin.





Figs 1-19a and 1-19b Carious response. Acid demineralization and enzymatic destruction of the collagen matrix lead to irreversible cavitation. (a) Bacteria fill and demineralize the lumens of the tubules peripherally, but dissolved minerals reprecipitate deeper to augment sclerosis and hypermineralization of subcarious dentin. Reparative dentin with irregular and noncontinuous tubules forms a final barricade against bacterial metabolites. (b) Note the lateral spread of the caries lesion at the DEJ and a hypermineralized sclerotic zone around the pulp.

(3) Reaction to severe, rapidly advancing caries:

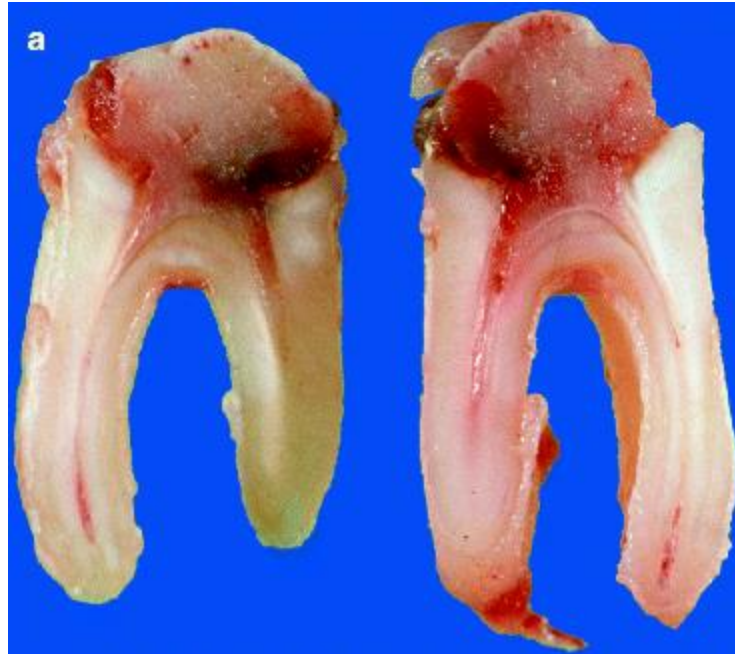
The third level of dentinal response is to severe irritation.

Acute, rapidly advancing caries with very high levels of acid production overpowers dentinal defenses and results in **infection, abscess, and death of the pulp.**

In comparison to other oral tissues, the pulp is poorly tolerant of inflammation. As small, localized infections in the pulp produce an inflammatory response involving capillary dilation, local edema, and stagnation of blood flow.

Because the pulp is contained in a sealed chamber and its blood is supplied through very narrow root canals, any stagnation of blood flow can result in local anoxia and necrosis.

The local necrosis leads to more inflammation, edema, and stagnation of blood flow in the immediately adjacent pulp tissue, which then becomes necrotic that rapidly spreads to involve the entire pulp.



Maintenance of pulp vitality is dependent on the adequacy of pulpal blood supply.

Recently erupted teeth with large pulp chambers and short, wide canals with large apical foramina have a much more favorable prognosis for surviving pulpal inflammation than fully formed teeth with small pulp chambers and small apical foramina.



TABLE 3-9

Pit-and-Fissure Caries Treatment Decision-Making*

POSTERIOR TOOTH	CLINICAL DIAGNOSIS	PREDICTION/OBSERVATION	TREATMENT
Pit and fissure	Noncavitated	Caries unlikely/no progression	No treatment
		Caries likely/progression	Sealant and antimicrobial; fluoride
	Cavitated		Restoration and antimicrobial; fluoride

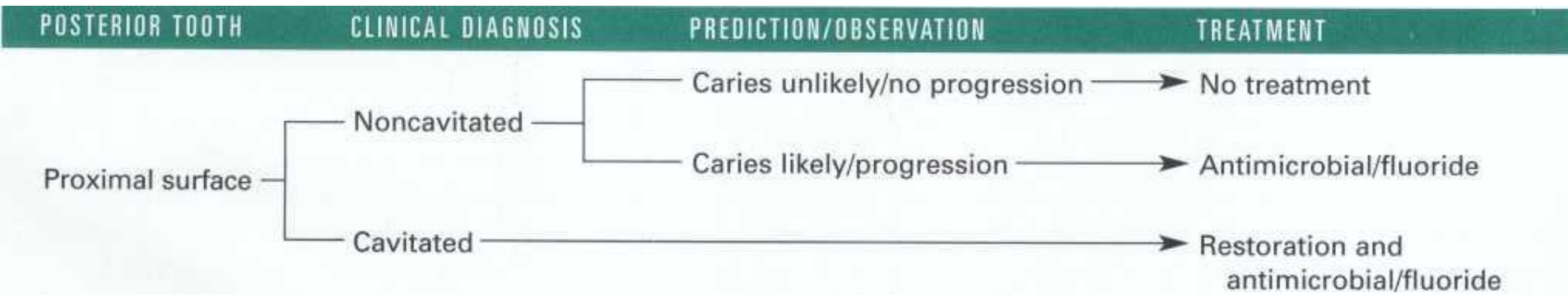
Noncavitated (caries-free):

- No radiolucency below occlusal enamel
- Deep grooves may be present
- Superficial staining may be present in grooves
- Mechanical binding of explorer may occur

Cavitated (diseased): extensive enamel demineralization has lead to destruction of the walls of the pit or fissure and bacterial invasion has occurred

- Chalkiness of enamel on walls and base of pit or fissure
- Softening at the base of a pit or fissure
- Brown-gray discoloration under enamel adjacent to pit or fissure
- Radiolucency below occlusal enamel

Proximal Caries Treatment Decision-Making"

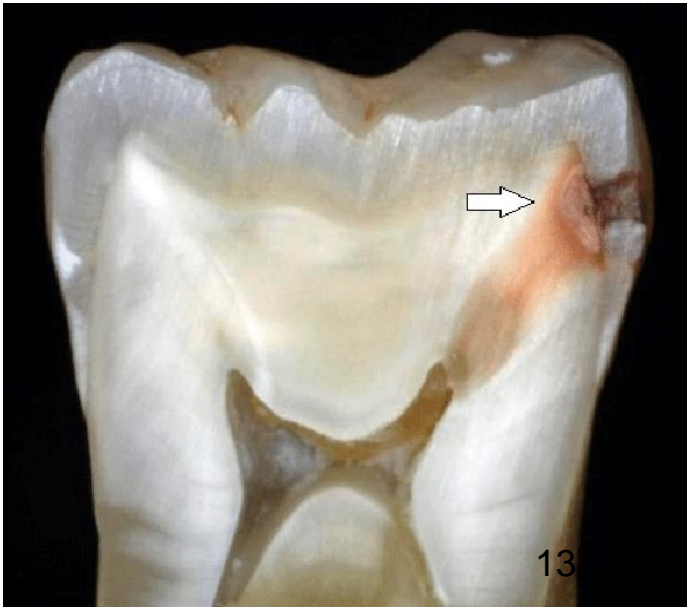


Noncavitated:

- Surface intact; use of an explorer to judge surface must be done with caution because excessive force can cause penetration of intact surface over demineralized enamel
- Opacity of proximal enamel may be present
- Radiolucency may be present
- Marginal ridge is not discolored
- Opaque area may be seen in enamel by translucination

Cavitated:

- Surface broken, detectable visually or tactilely;
- Marginal ridge may be discolored
- Opaque area in dentin on translucination
- Radiolucency is present



CARIES TREATMENT

Early restorative intervention should be avoided if possible, why?

1. As tooth preparation is irreversible and commits the tooth to the restorative cycle.
2. All restorations fail at some time and require either repair or replacement, resulting in yet another insult to the tooth tissues.
3. This repeated insult can ultimately lead to the loss of the tooth.

Caries prevention

Diet

Decreasing the frequency of fermentable carbohydrate consumption and elimination or substitution is essential.

Fluoride

Fluoride has produced the following reductions in caries.

Oral hygiene

A well maintained oral hygiene regime helps to maintain the bacterial balance within the oral cavity.

The different concepts in caries removal

1-Ozone therapy

2-Air abrasion technique

3-Chemomechanical caries removal

4-Smart bur

5-Stepwise excavation

6-Use of laser in tooth preparation

7-Use of ultrasonic in tooth preparation

In high **risk caries** development patient, the treatment should consist of both restorative and preventive measures. If patient has cavitated carious lesions should be restored first then fissure sealant and fluoride should be applied. Patient education is important in treatment success to increase motivation to obtain good oral health.

Principles of operative intervention:

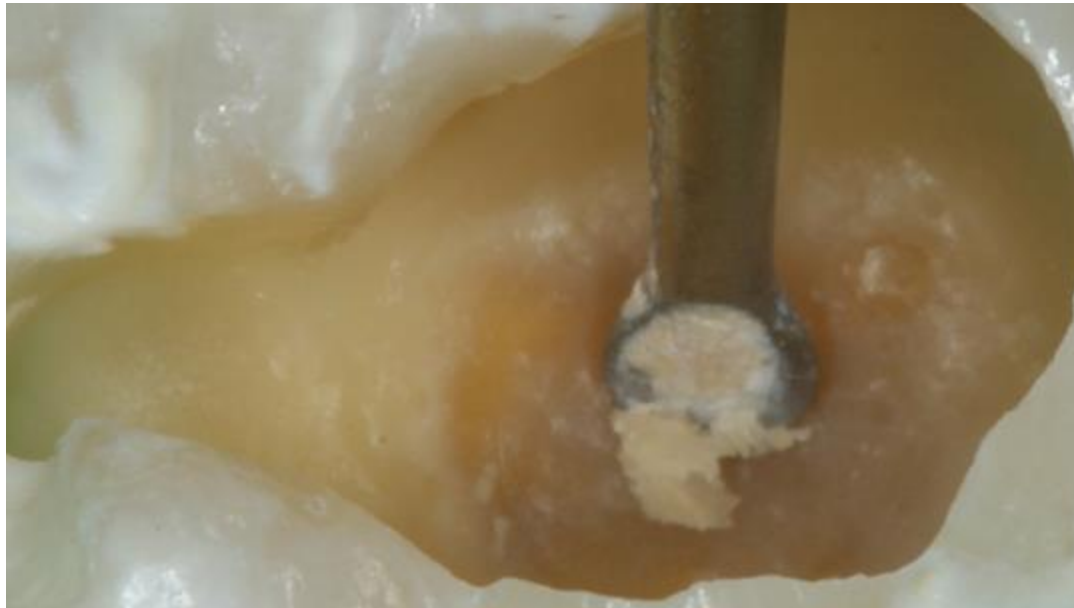
In contrast to Black's principles of cavity preparation, which included the establishment of outline form including extension for prevention, the development of resistance and retention form, creation of convenience form, the treatment of residual caries, the finishing of cavity margins and cavity toilet,

Now the general principles of tooth preparation are determined by:

- The nature and extent of the lesion.
- The quantity and quality of the tooth tissue remaining following preparation.
- Functional load.
- The nature and properties of the restorative system to be used.

Effective caries removal can be accomplished with:

- (1) hand instrumentation using spoon excavators. The use of spoon excavators may result in peeling off amounts of softened dentin larger than intended and therefore result in inadvertent pulp exposure. Thus, hand excavation requires great skill and sharp instruments.



(2) a slow-speed handpiece with a large round bur. A simple technique is to run the handpiece slowly enough that the bur stalls shortly after contacting the dentin. Repeated applications of the bur will remove dentin in small increments and allow the operator to carefully monitor changes in both hardness and color.



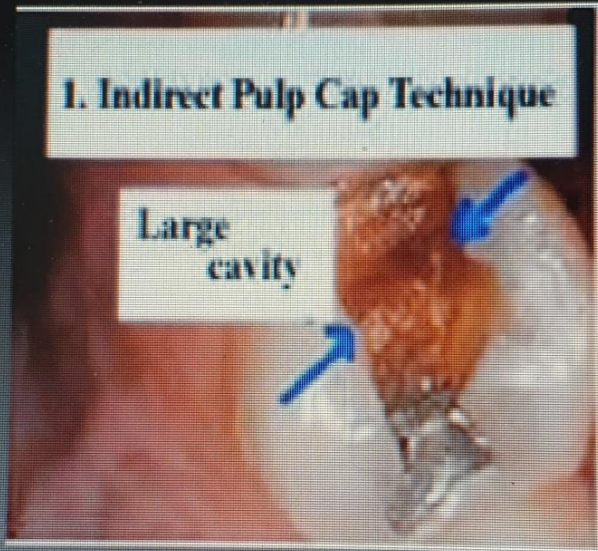
(3) a high-speed handpiece using a round bur operated just above stall-out speed (low speed).

After removal of softened dentin, it is then helpful to carefully evaluate the excavated area with a **sharp explorer** to determine if the remaining dentin is hard and sound.

Extreme care must be used with the explorer to prevent penetration into the pulp.

Penetration of the explorer into the pulp may cause pulpal infection, increasing the possibility of pulpal death.

INDIRECT PULP CAPPING



Usually all soft, infected dentin is removed during caries control procedures. However, in **asymptomatic** teeth that have deep lesions (where complete excavation of softened dentin is anticipated to produce pulpal exposure), the softened dentin nearest the pulp may be **left**.

The deliberate retention of softened dentin near the tooth pulp and medication of the remaining dentin with calcium hydroxide is termed an **indirect pulp cap**.

The goals of the caries control procedure are to prevent pulp exposure and aid pulpal recovery by medication. The portion of the remaining softened dentin is covered with a calcium hydroxide liner and the excavated area is restored with a temporary material.

Calcium hydroxide promotes reparative dentin bridges over any area of frank pulpal exposure. Such repair usually occurs in 6 to 8 weeks and may be evident radiographically in 10 to 12 weeks.



DIRECT PULP CAPPING

A *direct pulp cap* is a technique for treating a pulp exposure with a material that seals over the exposure site and promotes **reparative dentin formation**.

If the exposure site is the consequence of infected dentin extending into the pulp, termed a **carious pulpal exposure**, infection of the pulp already has occurred and removal of the tooth pulp is indicated and proceed with the root canal therapy (Endodontic treatment).

If, however, the pulp exposure occurs in an area of normal dentin (usually as a result of operator error or misjudgment), termed a **mechanical pulpal exposure**, and bacterial contamination from salivary exposure does not occur, the potential success of the direct pulp cap procedure is enhanced.

Pulp instrument traumatization:

If the pulp is penetrated by an instrument during the operative procedure, then a decision must be made whether to proceed with root canal therapy or do a **direct pulp cap**.



Direct Pulp Capping

Technique for treating a pulp exposure with a material that seals over the exposure site & promotes reparative dentin formation..

Requirements of direct pulp capping:

- Asymptomatic vital tooth
- Pin-point exposure (0.5mm or less in diameter)
- Non-hemorrhagic or easily controlled.
- Dry, sterile filed
- Non-carious atraumatic exposure



With either type of exposure (carious or mechanical) , a more favorable prognosis for the pulp following direct pulp capping may be expected if:

1. The tooth has been asymptomatic (no spontaneous pain, normal response to thermal testing, and is vital) before the operative procedure.
2. The exposure is small, less than 0.5 mm in diameter.
3. The hemorrhage from the exposure site is easily controlled.
4. The exposure occurred in a clean, uncontaminated field (such as provided by rubber dam isolation).
5. The exposure was relatively a traumatic and little desiccation of the tooth occurred, with no evidence of aspiration of blood into the dentin (dentin blushing).

A deep caries excavation close to the pulp, which may result in either an undetected pulpal exposure or a visible pulpal exposure, should be covered with a calcium hydroxide liner that can stimulate formation of dentin bridges (reparative dentin) over the exposure.

Pulp capping Material

- 1- Calcium Hydroxide Ca(OH)_2 .
- 2- Mineral Trioxide Aggregate MTA.
- 3- Tri-calcium phosphate.
- 4- Bioaggregate.
- 5- Biodentin
- 6- Bonding systems.

THANK YOU



Tooth Structure Odontogenic pain, Dentin sensitivity, & Theories

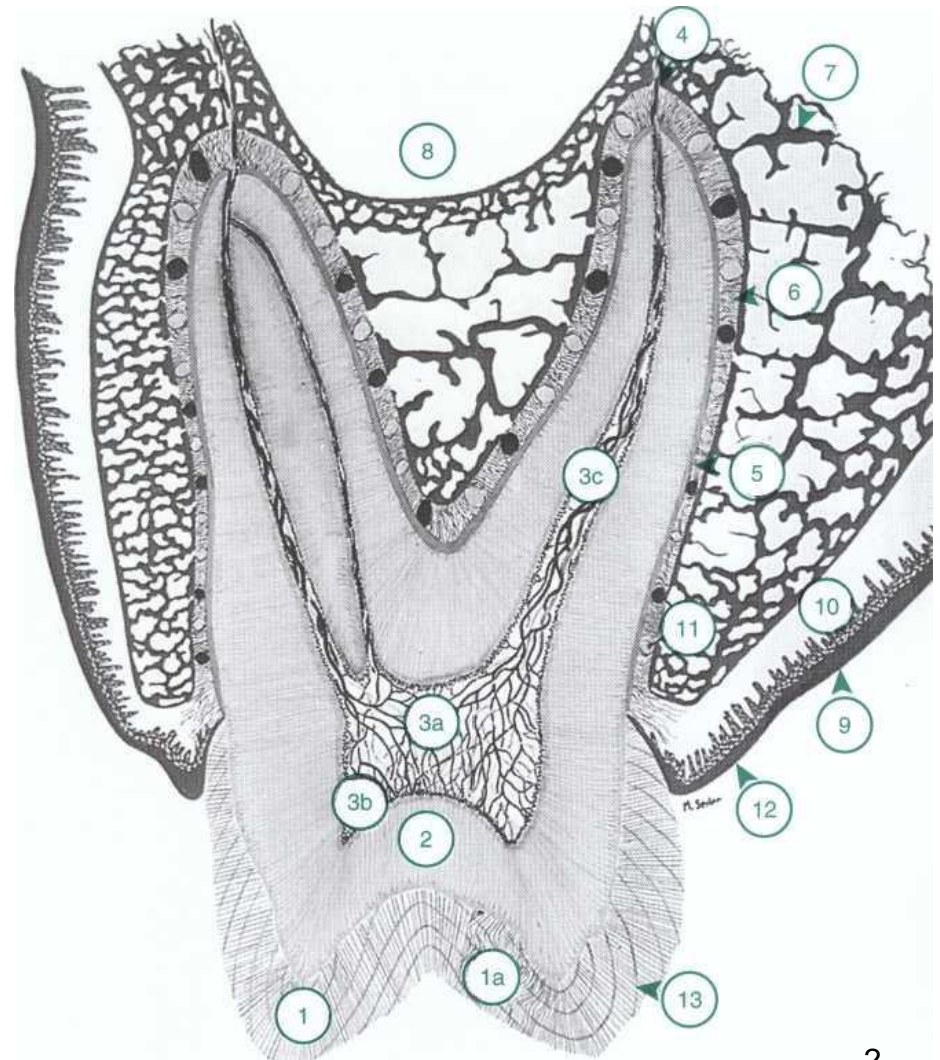
By

Assistant Prof. Dr. Maan M. Nayif

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The tooth is composed of enamel, pulp-dentin complex, and cementum

1, enamel; 2, dentin; 3a, pulp chamber; 3b, pulp horn; 3c, pulp canal; 4, apical foramen; 5, cementum; 6, periodontal fibers in periodontal ligament; 7, 13, Lines of Retzius.



Enamel:

Enamel is formed by cells called *ameloblasts*, which originate from the embryonic germ layer known as *ectoderm*. Enamel covers the anatomic crown of the tooth and varies in thickness in different areas. Enamel is a very brittle structure with a high elastic modulus and low tensile strength, which indicates a rigid structure.

Enamel is soluble when exposed to an acid medium, fluorides are present during enamel formation or are topically applied to the enamel surface, the solubility of surface enamel is decreased.

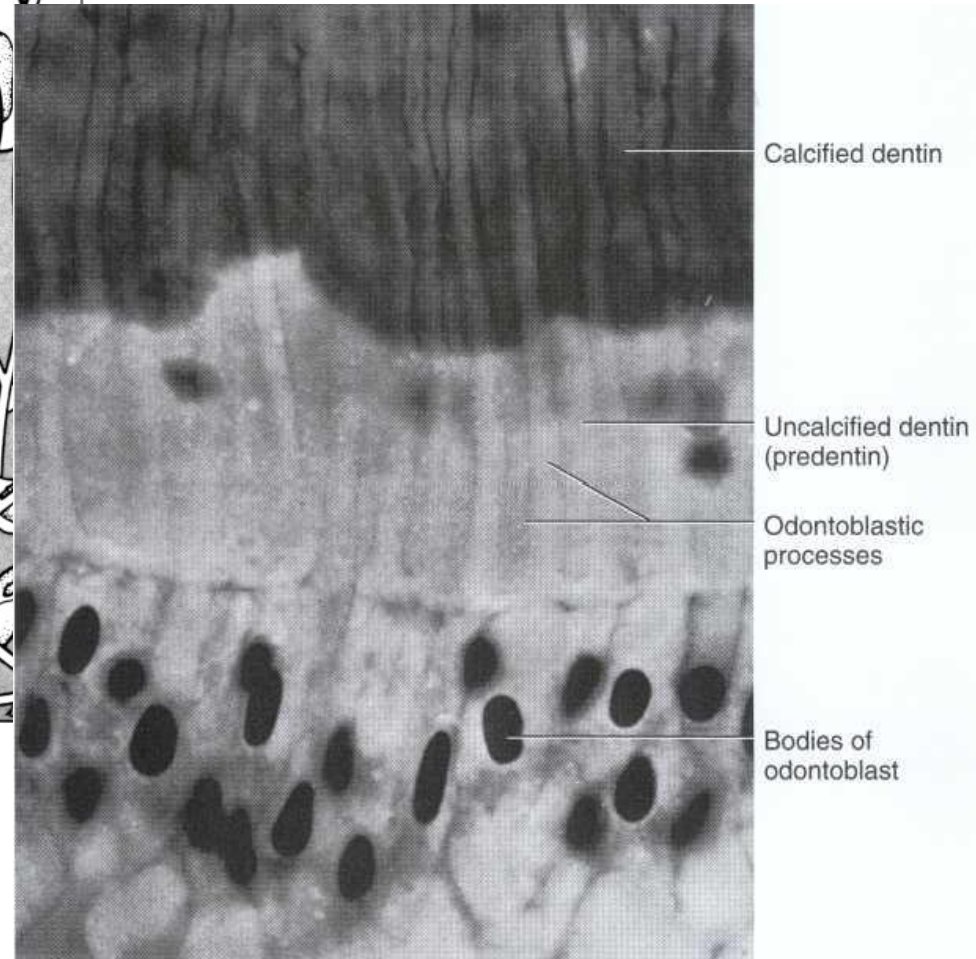
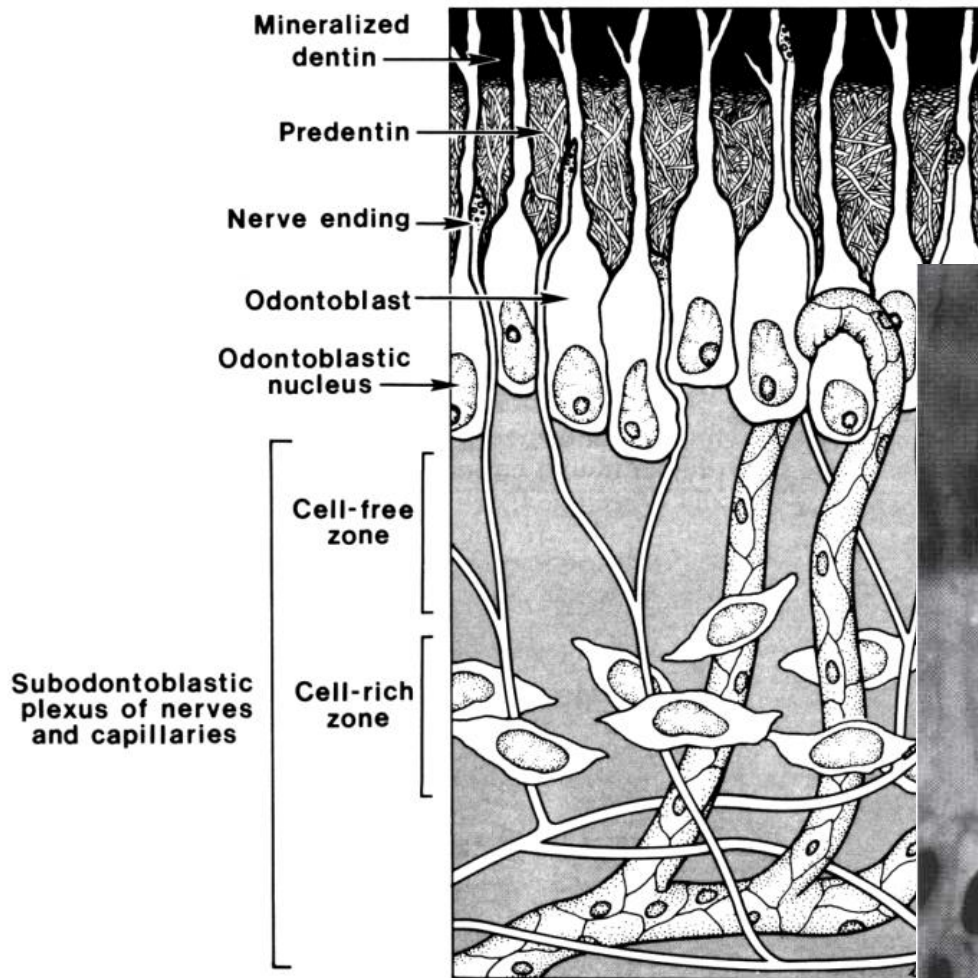
Pulp-Dentin Complex.

Dentin and pulp tissues are specialized connective tissues of mesodermal origin formed from the dental papilla of the tooth bud.

These two tissues are considered by many investigators **as a single tissue**, which thus form the pulp-dentin complex.

Dentin is formed by cells called **odontoblasts** *that* considered **part of both dentin and pulp tissues** because their cell bodies are in the pulp cavity but their long slender cytoplasmic cell processes (*Tomes fibers*) extend well into the tubules in the mineralized dentin.

Because of the odontoblastic processes the dentin is considered a living tissue with the capability to react to physiologic and pathologic stimuli.



Unlike enamel, dentin formation continues after tooth eruption and throughout the life of the pulp. The most recently formed layer of dentin is always on the pulpal surface. The most recently formed layer of dentin is always on the pulpal surface.

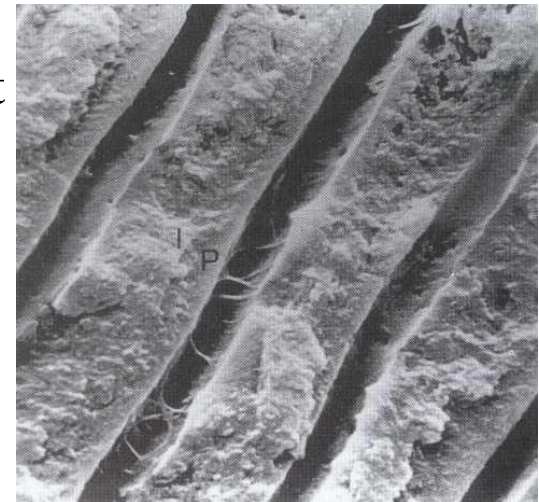
Predentin : most recently formed layer of dentin is always on the pulpal surface it represent unmineralized zone of dentin is immediately next to the cell bodies of the odontoblasts .

Primary dentin: Dentin forming the initial shape of the tooth and is usually completed 3 years after tooth eruption (for permanent teeth).

Secondary dentin: After the primary dentin is formed, dentin deposition continues at a reduced rate even without obvious external stimuli, Therefore the continuous deposition result reduction in the size of the dental pulp.

Peritubular dentin: Highly mineralized layer that line the dentinal tubule.

Intertubular dentin: less mineralized than peritubular dentin located in between dentinal tubules .



Sclerotic or transparent dentin:

Results from aging (physiologic) or mild irritation (such as slowly advancing caries) (Reactive) and causes a change in the composition of the primary dentin. Tubules replaced by calcified materials that might obliterate tubules and provide protection to the pulp from irritation.

Reparative dentin (tertiary dentin) :

is formed by replacement odontoblasts (termed *secondary odontoblasts*) in response to moderate-level irritants, such as attrition, abrasion, erosion, trauma, moderate-rate dentinal caries, and some operative procedures.

It usually appears as a localized dentin deposit on the wall of the pulp cavity immediately sub adjacent to the area on the tooth that has received the injury (a dentin deposit underneath the affected tubules).

Reparative or tertiary dentin is a defense reaction to an area of moderate-intensity injury.

Dental Pulp

The *dental pulp* occupies the pulp cavity in the tooth. Each pulp organ is circumscribed by the dentin and is lined peripherally by a cellular layer of odontoblasts adjacent to the dentin.

The dental pulp is composed :

(myelinated and unmyelinated nerves, arteries, veins, lymph channels, connective tissue cells, intercellular substance, odontoblasts, fibroblasts, macrophages, collagen, and fine fibers).

Anatomically the pulp organ is divided into:

- (1) coronal pulp located in the pulp chamber in the crown portion of the tooth, including the pulp horns that are directed toward the incisal ridges and cusp tips.
- (2) radicular pulp located in the pulp canal(s) in the root portion of the tooth. The radicular pulp is continuous with the peri-apical tissues by connecting through the apical foramen or foramina of the root.

Accessory canals may extend from the pulp canal(s) laterally through the root dentin to the periodontal tissues. The shape of each pulp conforms generally to the shape of each tooth .

Function of the Pulp:

- (1) formative or developmental function , The formative function is the production of primary and secondary dentin by the odontoblasts.
- (2) nutritive, supplies nutriment and moisture to the dentin through the blood vascular supply to the odontoblasts and their processes.
- (3) sensory or protective, provides sensory nerve fibers within the pulp to mediate the sensation of pain. The pulp usually does not differentiate between heat, touch, pressure, or chemicals.
- (4) defensive or reparative. is related primarily to its response to irritation by **mechanical, thermal, chemical, or bacterial stimuli.**

Such irritants can cause the degeneration and death of the affected odontoblastic processes and corresponding odontoblasts and the formation of replacement odontoblasts (from undifferentiated pulpal mesenchymal cells) that lay down irregular or reparative dentin.

- However, inflammation may become irreversible and can result in the death of the pulp because the confined, rigid structure of the dentin limits the inflammatory response and the ability of the pulp to recover.

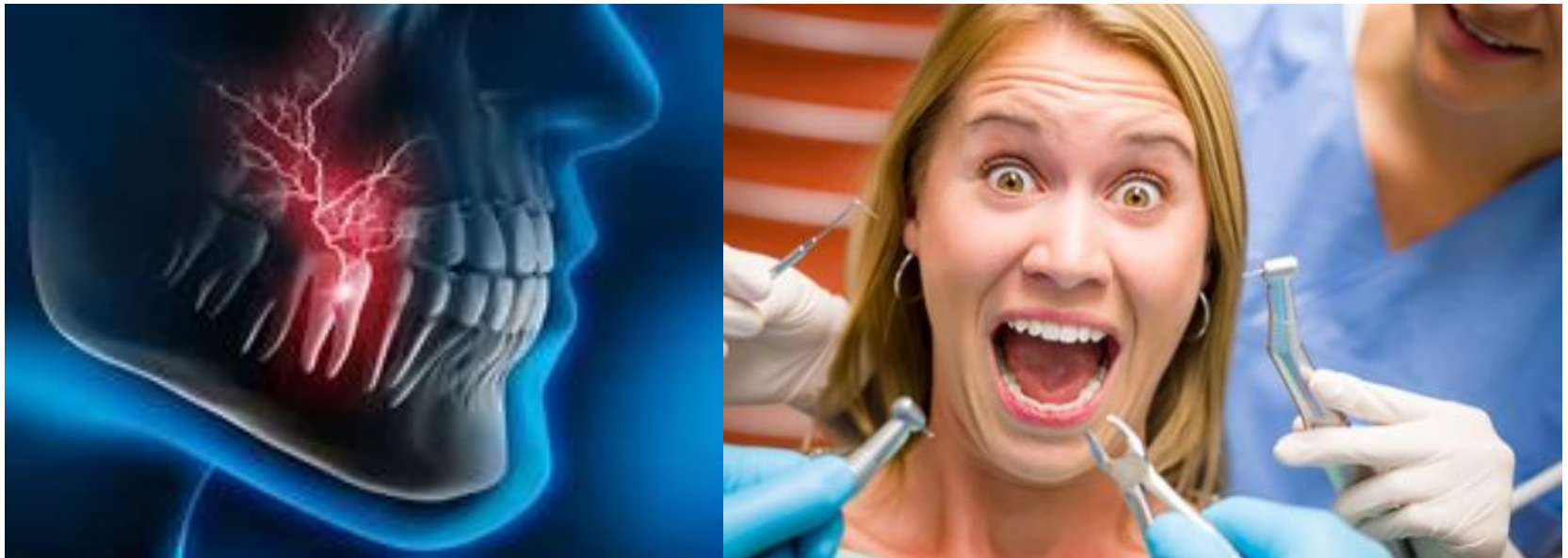
Odontogenic pain can be originated from the following conditions:

- Dentin hypersensitivity
- Pulpitis (Reversible & irreversible)
- Apical pain
- Cracked tooth
- Periapical periodontitis
- Acute periapical abscess
- Periapical granulomas/cysts
- Traumatic periodontitis
- Periodontal abscess
- Perio-endo/endo-perio lesions
- Acute alveolar osteitis
- Acute peri-coronitis

Odontogenic pain (Dental pain) refers to pain initiating from the teeth or their supporting structures(the mucosa, gingivae, maxilla, mandible or periodontal membrane).

Pain is unpleasant sensation associated with actual or potential tissue damage. It could be acute or chronic.

For an **effective diagnosis and treatment**, the clinician should have a thorough knowledge of the various pain complaints related to the orofacial region and the different options available for their optimal management.



One of the primary services provided by the dentist is diagnosis and relief of pain of **pulpal origin**.

PULPITIS•

Inflammation of the pulp (and periapical tissues) is commonly associated with pain constituting ~90% of emergency visits with tooth pain as the chief complaint.

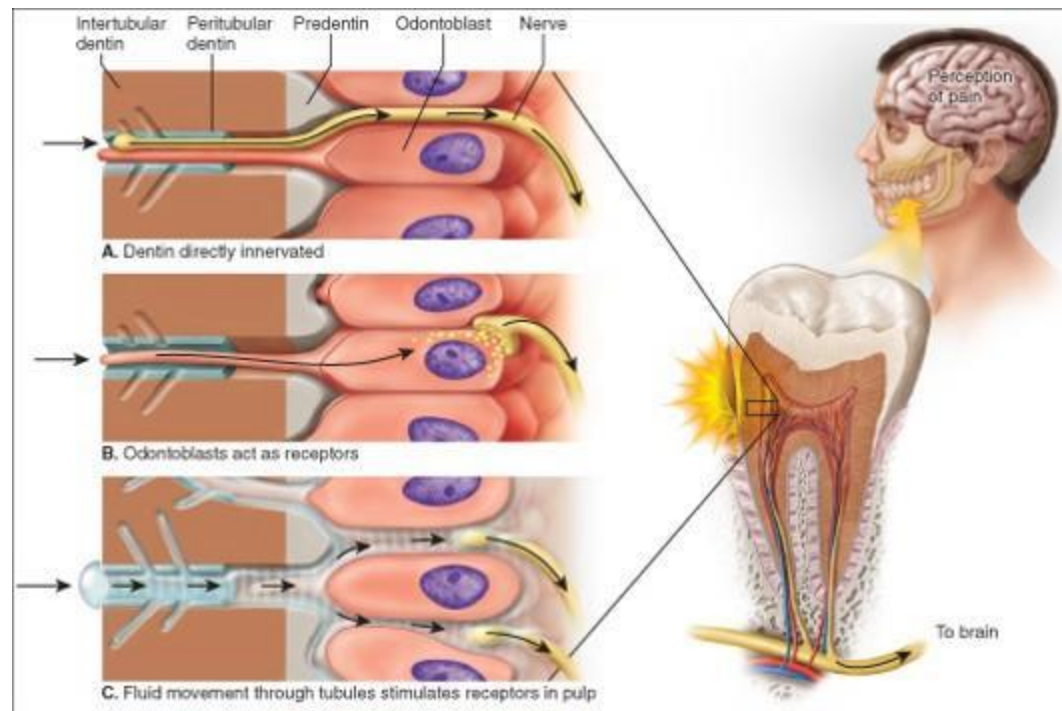
- This pain is attributed to activation of pulpal or periapical nociceptors.
- Tooth caries is the most predominant causes of pulpitis while other causes contribute to a less cause.
- Different types of pain among them is short sharp 'neuralgic' pain with extreme sensitivity to cold and sweet flavours, or some time could be severe pain interrupt patient sleep.

For managing odontogenic pain the “3-D’s” principle—**diagnosis**, **dental treatment**, and **drugs**— should be used.

- The first and foremost step is to determine the condition causing the pain and then to discover that what caused that condition.
- Removal of the cause usually leads to rapid recovery and should be done by an appropriate dental treatment.
- Medications should only be used to complement the dental treatment .

Caries is one of the most factor could initiate the pain. Often, pain is not reported even when caries invades dentin, except when deep lesions bring the bacterial infection close to the pulp.

These pains are due to stimulation of pulp tissue by movement of fluid through dentinal tubules that have been opened to the oral environment by cavitation.



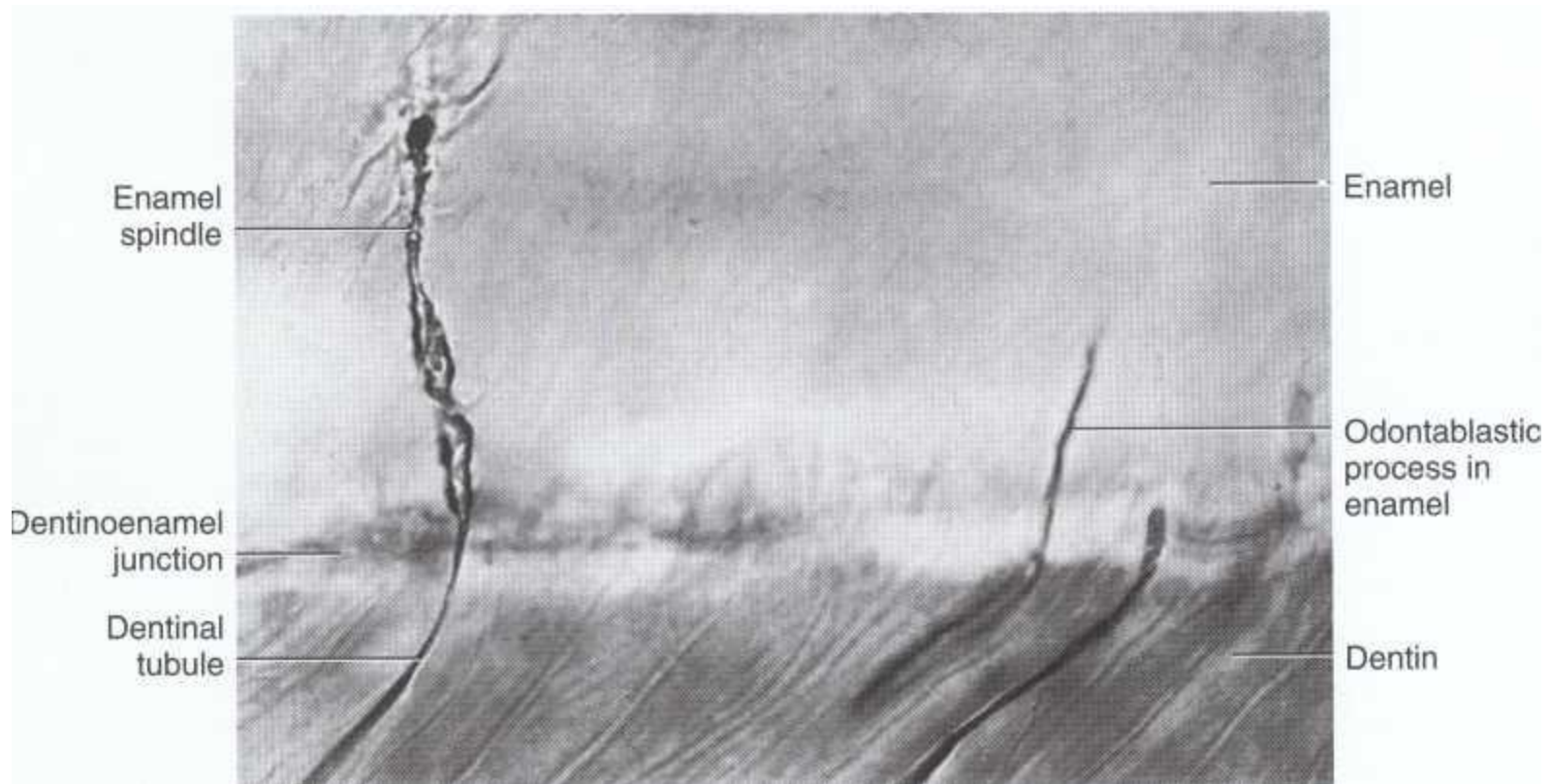
Dentine sensitivity: is a common form of odontogenic pain and is described as a short, sharp pain from exposed dentin in response to (thermal , tactile , osmotic , evaporative or chemical stimuli) and that cannot be ascribed to any other pathosis or any form of dental defect.

Sensitivity is encountered whenever odontoblasts and their processes are stimulated during operative procedures, even though the **pain receptor** mechanism appears to be within **the dentinal tubules near the pulp**.

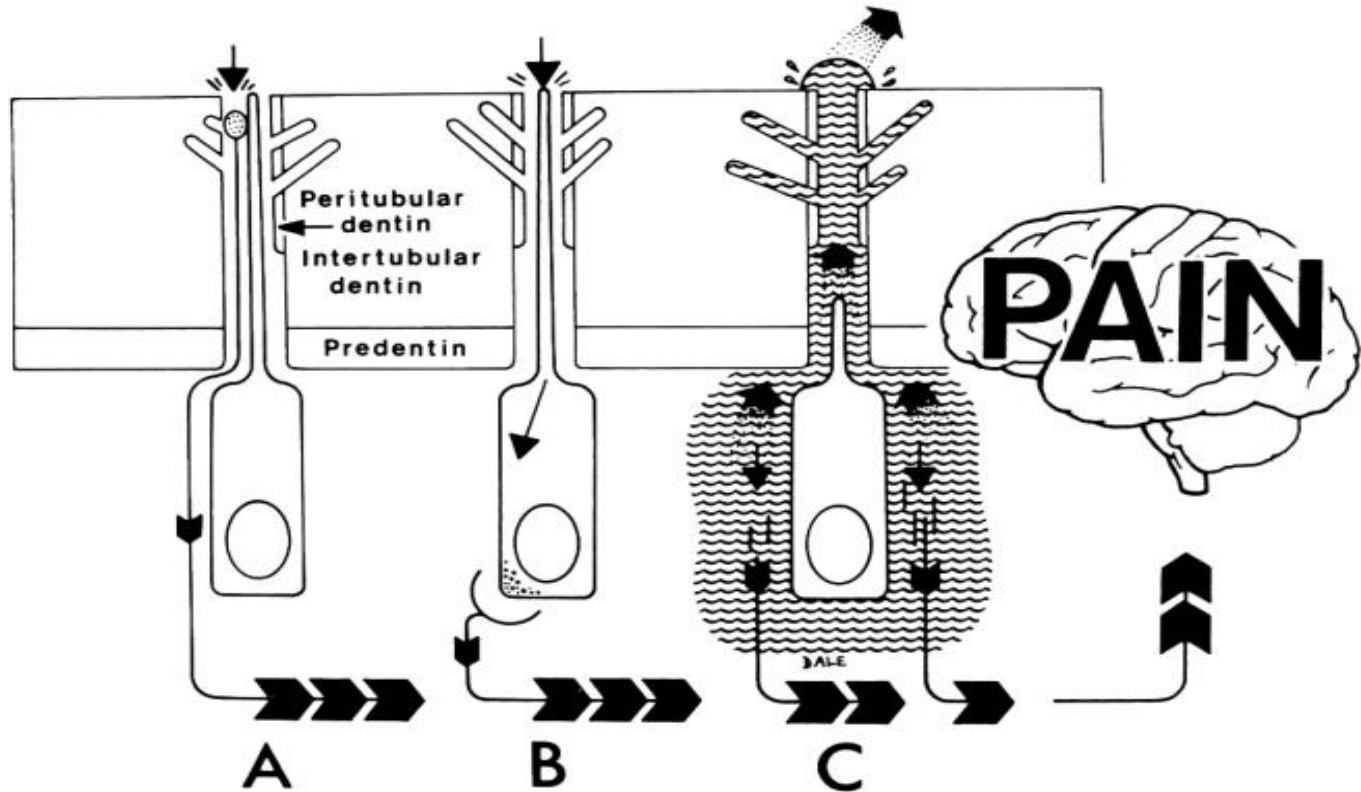
Because many tubules contain mechanoreceptor nerve endings near the pulp, small fluid movements in the tubules arising from cutting, drying, pressure changes, osmotic shifts, or changes in temperature account for the majority of pain transmission (Fig. 2-23).

Odontoblastic processes sometimes cross the DEJ into the enamel; these are termed *enamel spindles*.

They may serve as pain receptors, thereby explaining the **enamel sensitivity** experienced by some patients during tooth preparation.



Sensitivity Theories:

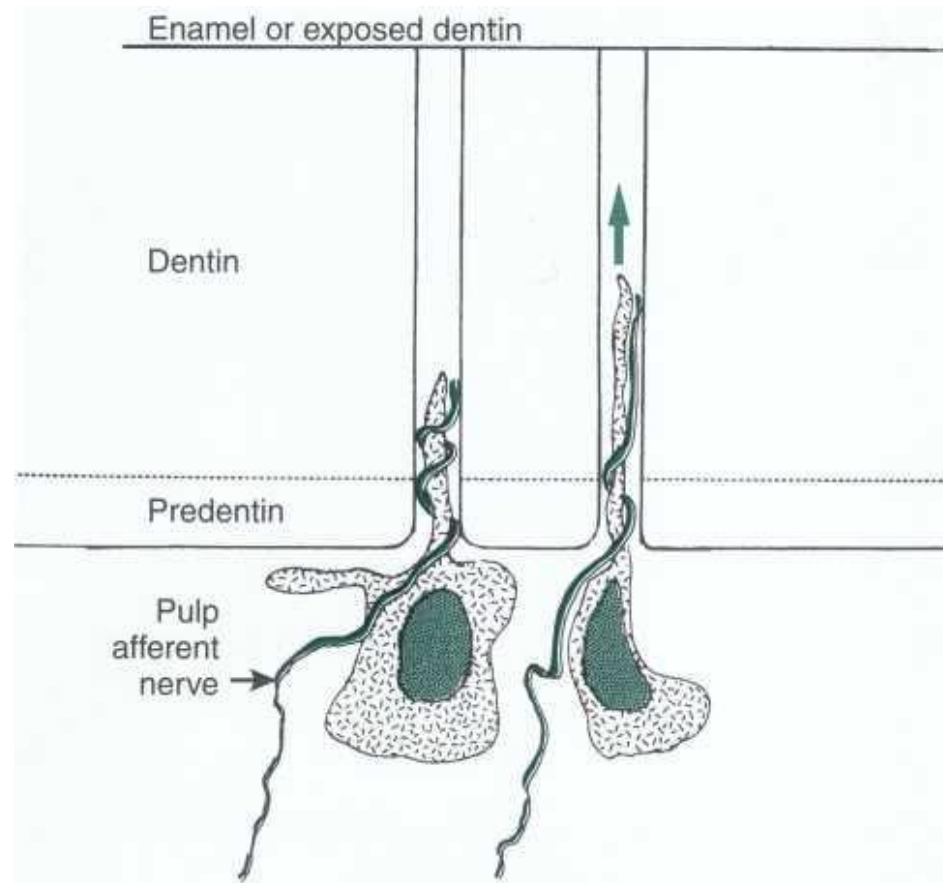


A, Classical theory proposed that stimuli applied to dentin caused direct stimulation of nerves in dentin.

B, Modified theory proposed that stimuli applied to the odontoblastic process would be transmitted along the odontoblast and passed to the sensory nerves via some sort of synapse.

C, Hydrodynamic theory proposed that fluid movement within tubules transmits peripheral stimuli to highly sensitive pulpal nerves.

The most accepted theory of pain transmission is the *hydrodynamic theory*, which accounts for pain transmission through small, rapid movements of fluid that occur within the dentinal tubules .



Stimuli that induce fluid movements in dentinal tubules distort odontoblasts and afferent nerves (*arrow*), leading to a sensation of pain. Many operative procedures such as cutting or air-drying induce such fluid movement

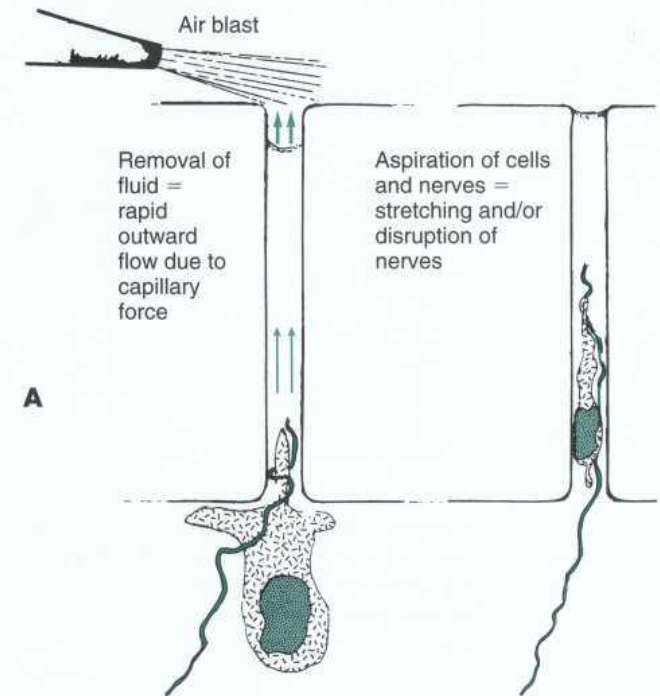
Dentinal tubules are normally filled with odontoblastic processes and *dentinal fluid*, a transudate of plasma.

When enamel or cementum is removed during tooth preparation, the external seal of dentin is lost and the tubules become fluid-filled channels from the cut surface directly to the pulp.

Fortunately, pulpal fluid has a slight positive pressure that forces fluid outward toward any breach in the external seal.

The tubules become more numerous, and increase in diameter closer to the pulp, deep dentin is a less-effective pulpal barrier than is superficial dentin near the dentino-enamel or dentino-cemental junctions.

The dentin should not be dehydrated by compressed air blasts; it should always maintain its normal fluid content.







Pulpal Pathosis Part I

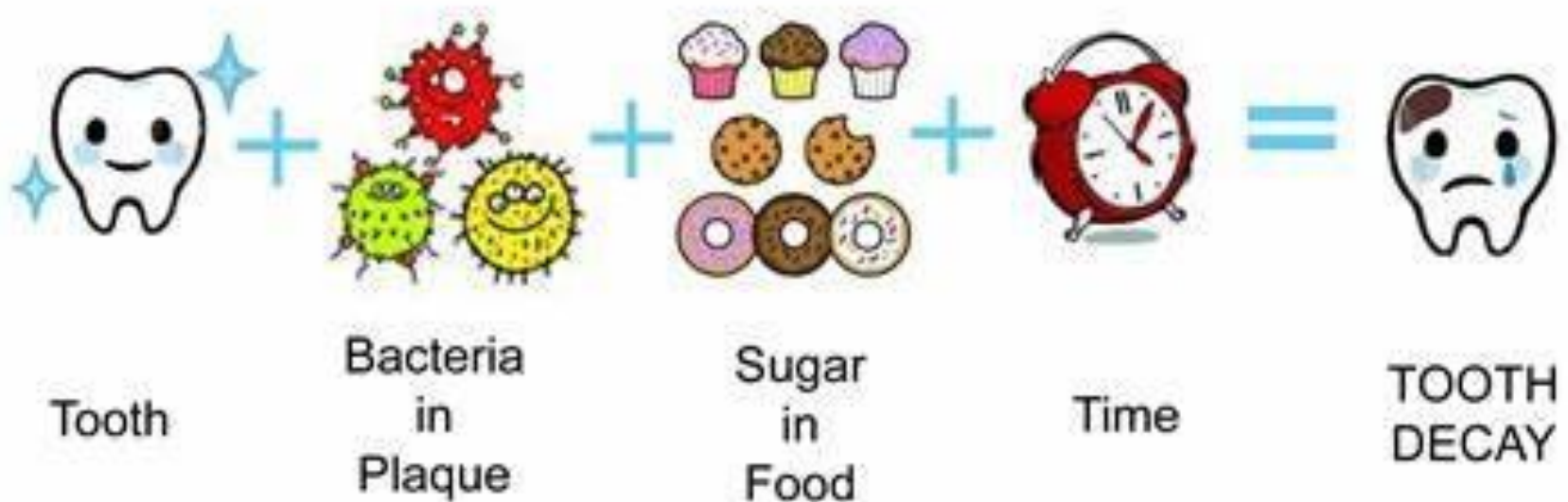
(CH2&9 in 4th edition)

By

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The response of the pulp to dental caries may vary depending on whether the caries process is progressing **rapidly or slowly** or is **completely inactive** (i.e., arrested caries).



Carious attack rate may be influenced by following factors:

- 1• Age of the host
- 2• Composition of the tooth
- 3• Nature of the bacterial flora of the lesion
- 4• Salivary flow
- 5• Buffering capacity of the saliva
- 6• Antibacterial substances in the saliva
- 7• Oral hygiene
- 8• Cariogenicity of the diet and frequency
- 9• Caries-inhibiting factors in the diet

Effect of tooth Preparation on dental Pulp

Pulpal inflammation resulting from the operative procedures is often termed as **dentistogenic pulpitis**. During tooth preparation, pulpal reaction depends on following factors:

1. Pressure

Pressure of instrumentation causes aspiration of odontoblasts or nerve endings from pulp tissues into the dentinal tubules. This disturbs the metabolism of odontoblasts leading to their complete degeneration and disintegration.

2. Heat Production

If pulp temperature is elevated destructive reaction occurs. “Heat” is a function of:

- ◆ **RPM**, more the RPM greater is the heat production
- ◆ **Pressure**: It is directly proportional to heat generation
- ◆ **Surface area of contact**: The more the contact between tooth structure and revolving tool, greater is the heat generation
- ◆ **Desiccation**: It causes aspiration of odontoblasts into tubules. Subsequent disturbances in their metabolism may lead to the complete degeneration of odontoblasts.

3. Vibrations

Vibrations are an indication of eccentricity in instruments. Higher the amplitude, more destructive is the pulp response.

4. speed of rotation

Ultrahigh speed should be used for removal of enamel and superficial dentin. A speed of 3,000–30,000 rpm without coolant can cause pulpal damage.

5. nature of cutting Instrument

Use of worn off and dull instruments can cause vibration and reduced cutting efficiency. This further encourages the clinician to apply excessive operating pressure, which results in increased temperature leading to thermal injury to pulp.

6. use of coolants

Water spray is considered as the ideal coolant. In deep cavities, cotton pellet instead of air blast should be used to dry the prepared cavity because air blast can cause desiccation of dentin which can damage the odontoblasts

Keys to minimize the adverse pulpal reaction from rotary instrument are:

1. Adequate air –water coolant spray.
2. Light pressure of instrument on tooth structure.
3. Sharp rotary cutting instrument.
4. Preservation of tooth structure.

General guidelines

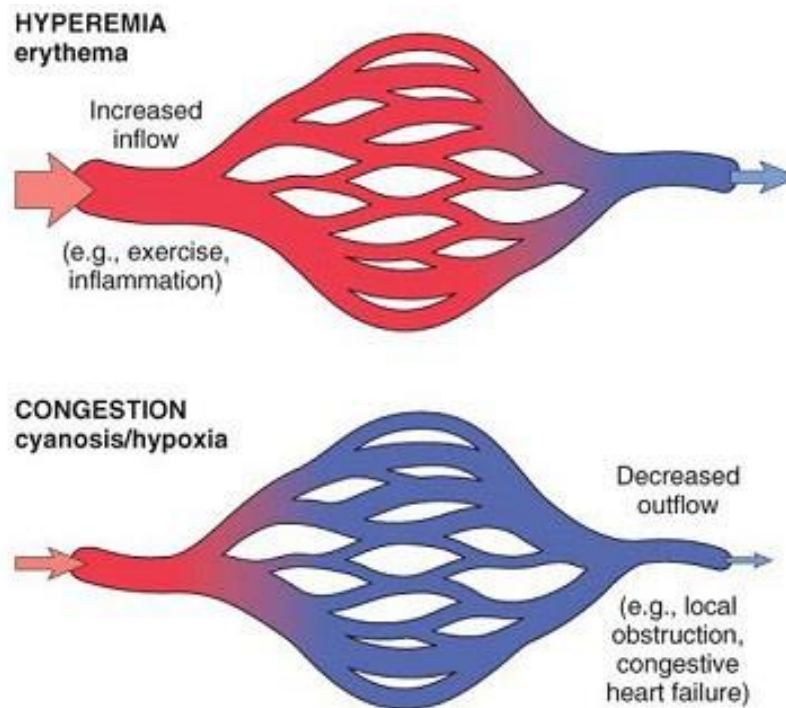
One of the primary services by the dentist is diagnosis and relief of pain of pulpal origin which is important to the successful practice of operative dentistry.

Clinical interpretation of pain from pulpal inflammation is somewhat **empiric**, thus it need experience, precise examination and test.

When an irritant (e.g., sugar, cold, acid from caries) first contacts dentin, the patient may be alerted by a **twinge** of pain.

This pain is usually only momentary, ceasing if the irritant is removed.

If irritation (e.g., sugar, cold, acid from caries) **continues** or the irritant is applied **repeatedly**, **hyperemia** (increased blood flow and volume) and inflammation of the pulp can result, which will cause the pain elicited from the irritation to linger than a few seconds.



Healthy Pulp

The healthy pulp is vital and free of inflammation with following characteristics:

1. A symptomatic and produces a mild-to-moderate transient response to thermal and electrical stimuli.

When the stimulus is removed, the response subsides almost immediately.



2. No pain response when percussed or palpated.



3. Radiographs reveal a clear canal that tapers smoothly toward the apex. There is no evidence of root resorption, and the lamina dura is intact.



The protection reactions of healthy dental pulp against caries :

- (1) Decrease in the permeability of the dentin; In this reaction the dentinal tubules become partially or completely filled with mineral deposits consisting of apatite and whitlockite crystals.
- (2) The ability of the pulp to produce reparative dentin beneath a carious lesion is another mechanism for limiting the diffusion of toxic substances to the pulp and
- (3) Inflammatory and immune reactions; When caries has invaded the dentin, some changes are occurring in the pulp as a result to the diffusion of soluble irritants and inflammatory stimuli into the pulp (bacterial toxins and enzymes, antigens, organic acids, and products of tissue destruction).

According to Grossman

Diseases of the pulp has been classified as:

I Pulpitis (inflammation)

Reversible

1. Acute (symptomatic).
2. Chronic (asymptomatic)

Irreversible pulpitis

1. Acute
 - Abnormally responsive to cold.
 - Abnormally responsive to heat.
2. Chronic
 - Asymptomatic with pulp exposure
 - Hyperplastic pulpitis.
 - Internal Resorption.

II Pulp Degeneration

- Calcific (radiographic diagnosis).
- Others (histopathologic diagnosis).

III Pulp Necrosis

- **Reversible pulpitis:**

Reversible pulpitis is *not* a disease; it is a symptom, the pulp is inflamed and affected with (causative factors):

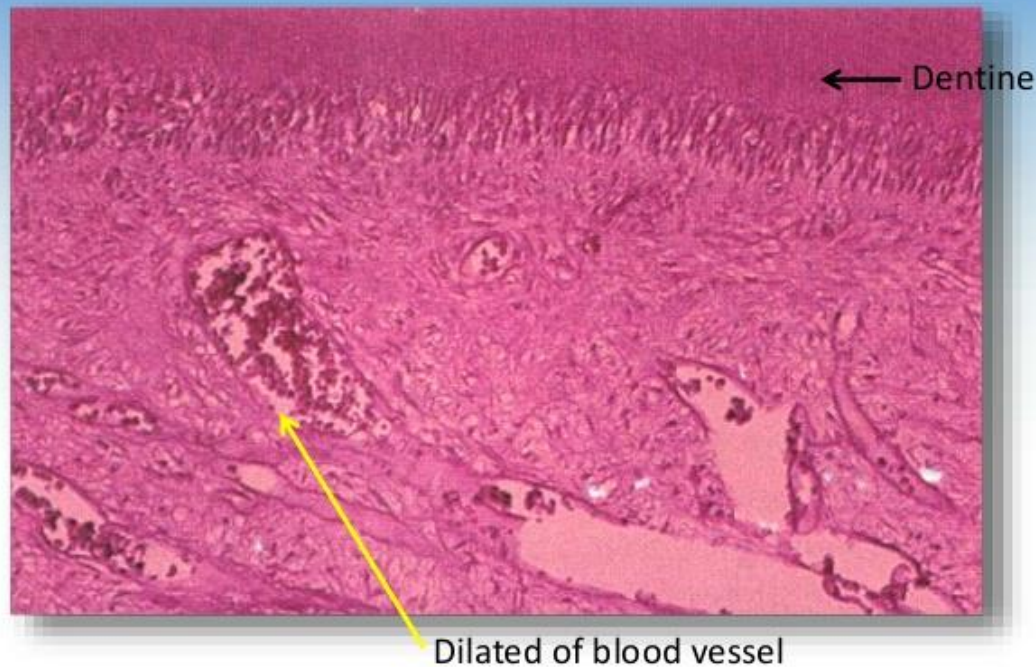
Thermal stimuli : usually cold-cause a quick, sharp, hypersensitive response and subsides as soon as the stimulus removed.

Other irritant (e.g., sugar, acid from early caries, periodontal scaling, microleakage, and unbased or defective restorations.)

This pain is usually only momentary, ceasing if the irritant is removed.

This is a clinical diagnosis based on subjective and objective findings indicating that the inflammation should resolve and the pulp return to normal. Conservative removal of the irritant will resolve the symptoms.

Reversible pulpitis



The reaction is because the pulp is contained by unyielding dentinal walls; thus drainage of the increased blood is limited by the constricted apical foramen.

Restorative treatment is possible as long as irritant, such as touching ice to the tooth, causes pain lingers no more than 10 to 15 seconds after removal of the irritant.

Summary of Reversible pulpitis / Pulpal hyperemia

- Reversible pulpitis is a mild to moderate inflammatory condition of the pulp caused by the noxious stimuli in which the pulp is capable of returning to the uninfamed state following removal of the stimuli.
- Symptomatic reversible pulpitis is characterized by sharp pain lasting for a moment, and generally disappears when the stimulus is removed like Cold , sweet, or sour usually causes it.

- The pulp is sensitive to temperature changes, particularly cold, application of cold is an excellent method of locating and diagnosing the involved tooth.
- Thermal test are useful in locating the affected tooth if unknown.
- A tooth with reversible pulpitis reacts normally to percussion, palpation and mobility.
- The electric pulp test, using less current than on a control tooth, is an excellent confirmation test.

The background is an abstract painting with visible brushstrokes. It features a mix of cool and warm tones: various shades of blue (teal, light blue, and dark blue) are layered with vibrant reds and yellows. The strokes are thick and expressive, creating a textured, painterly effect.

**Thank
you!**



Pulpal Pathosis Part II

(CH2&9 in 4th edition)

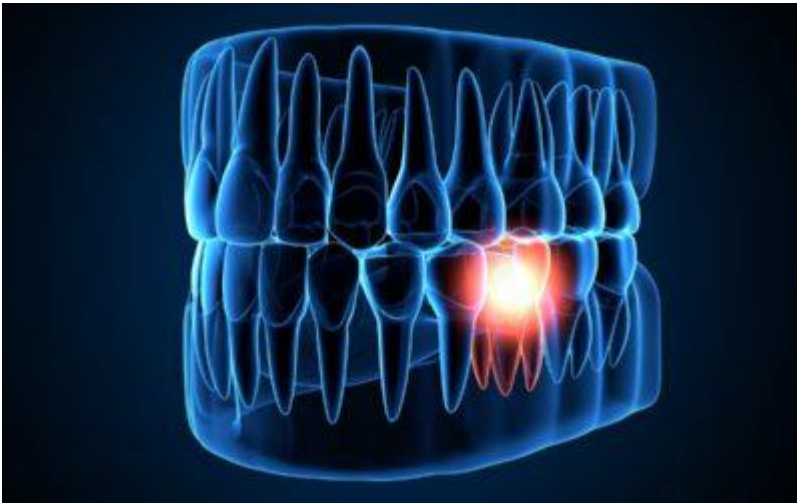
By

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•Irreversible pulpitis:

•Irreversible pulpitis is a persistent inflammatory condition of the pulp, symptomatic or asymptomatic caused by a noxious stimulus.



Irreversible pulpitis may be **acute, subacute, or chronic**; it may be **partial or total, infected or sterile**.

Clinically, the acutely inflamed pulp is symptomatic, whereas, the chronically inflamed pulp is asymptomatic in most cases.

Although uncommon, asymptomatic irreversible pulpitis may be the conversion of symptomatic irreversible pulpitis to a silent state.

Symptomatic Irreversible Pulpitis:

is characterized by spontaneous, intermittent, or continuous paroxysms of pain.

Sudden temperature changes (usually cold) elicit prolonged episodes of pain (i.e., pain that lingers after the thermal stimulus is removed).

This pain may be relieved in some patients by the application of heat. Occasionally, patients may report that a postural change (lying down or bending over) induces pain.

Generally, pain from symptomatic irreversible pulpitis is moderate to severe; it can be sharp or dull, localized or referred.

In most cases, radiographs are not useful in diagnosing symptomatic irreversible pulpitis because the inflammation remains confined to the pulp.

- In the early stages the thermal test may elicit pain that persists after removal of the thermal stimulus. However in the late stages when the pulp is exposed ,it may respond normally to the thermal stimulus.

- Result of examination for mobility and percussion and palpation tests are negative.

When pulpal pain, either spontaneous or elicited by an irritant, lingers more than 15 seconds, infection of the pulp often has occurred and resolution by operative dentistry treatment is usually doubtful; root canal therapy is advised for this pulpal condition.

Development of irreversible pulpitis:

Once bacterial invasion of the dentin (mostly via caries) is close to the pulp, toxins and possibly even a few bacteria enter the pulp, resulting in contamination and inflammation of the pulpal tissues.

Initial pulpal inflammation is thought to be evident clinically by production of sharp pains, with each pain lingering only a few seconds (10 or less) in response to a thermal stimulus.

When the pulp becomes more severely inflamed, a thermal stimulus will produce pain that continues after termination of the stimulus, typically longer than 10 seconds.

This clinical pattern suggests *irreversible pulpitis*, when the pulp is unlikely to recover after removing the caries.

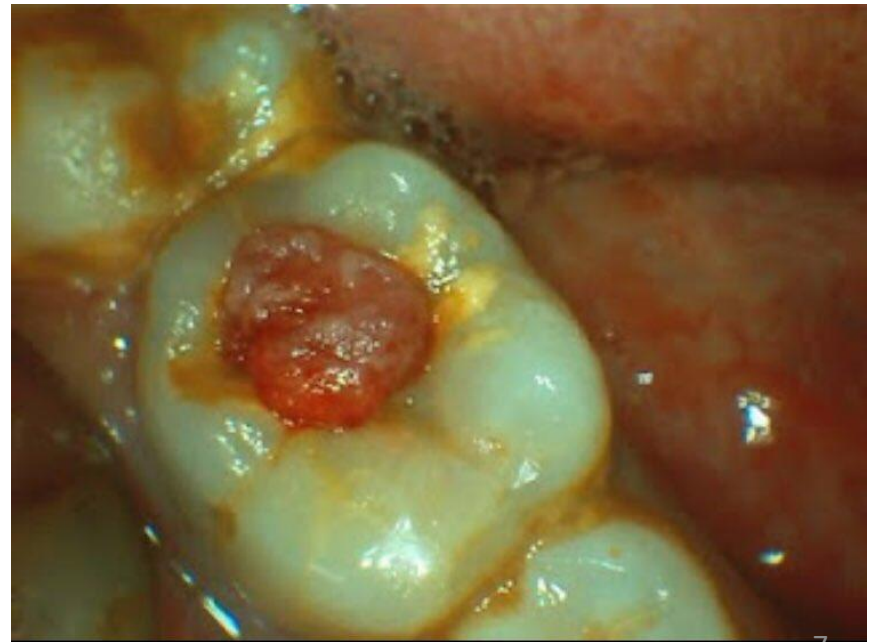
Throbbing, continuous pain suggests partial or total pulp necrosis that is treated only by root canal therapy or extraction.

Although these clinical characteristics are useful as guidelines for pulp treatment, it is emphasized that pulp symptoms can vary widely and are not always predictive of the histologic status of the tooth pulp.

Chronic Hyperplastic Pulpitis

- Chronic hyperplastic pulpitis or (pulp polyp) is a productive pulpal inflammation due to an extensive carious of a young pulp.
- Slow , progressive carious exposure of the pulp is the cause.
- Mechanical irritation from chewing and bacterial infection often provide the stimulus.
- The tooth may respond feebly or not at all to the thermal test, unless one use extreme cold, as from an methyl chloride spray.
- More current than normal may be required to elicit a response by means of the electric pulp tester.

Hyperplastic pulpitis is an example of irreversible pulpitis occurs almost exclusively in primary and immature permanent teeth with open apices. It develops in response to carious exposure of the pulp when the exposure enlarges to form a cavity in the roof of the pulp chamber "pulp polyp". A reddish growth of pulp tissue through and around a carious exposure.



Pulp necrosis:

When this condition (*irreversible pulpitis*) is untreated, suppuration and pulpal necrosis is follows. Necrosis is death of the pulp.

It can be caused by any noxious insult to the pulp , such as bacteria (caries), trauma ,and chemical irritation.



- A normal tooth with a necrotic pulp cause no painful symptoms. Thus presence of necrotic pulp may be discovered only by chance because such tooth is asymptomatic ,and the radiograph is non diagnostic. Tooth with necrotic pulp does not respond to cold ,the electric pulp test ,or the test cavity.

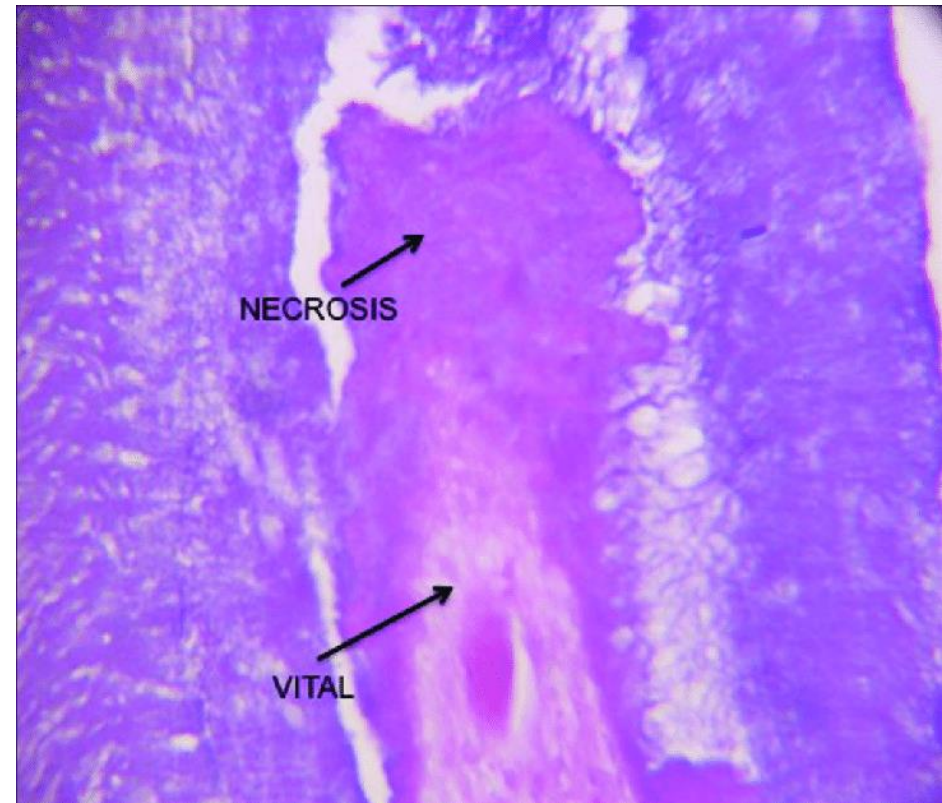
Early pulpal necrosis characterized by spontaneous, continuous throbbing pain or pain elicited by heat that can be relieved by cold and later characterized by no response to any stimulus.

Pulp necrosis may be partial or total.

The teeth with **partial necrosis** can respond to thermal changes ,owing to the presence of vital nerve fibers passing through the adjacent inflamed tissue.

Partial necrosis may be difficult to diagnose, because it can produce some of the symptoms associated with irreversible pulpitis.

Pulpal necrosis is treated by root canal therapy or tooth extraction.



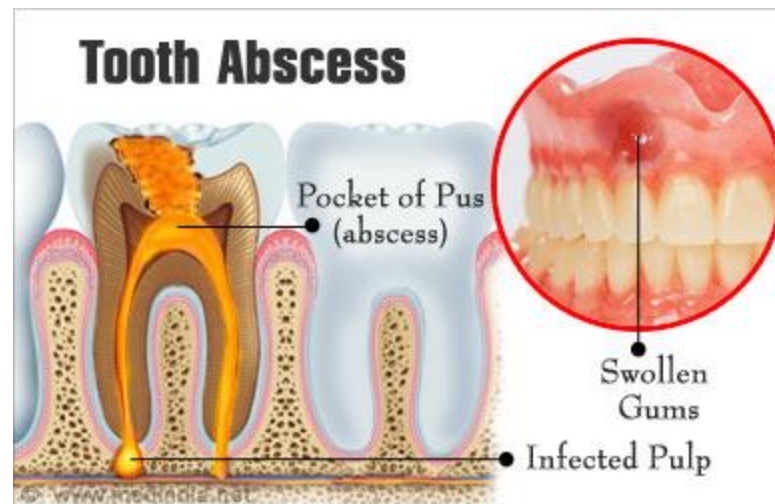
Pulpal Abscesses

When all the pulp's defense systems are failed often the result will be pulpal abscess.

Carious exposure of the pulp results in progressive mobilization of neutrophils and finally to suppuration, in the form of an abscess.

Other chemical like acid or resinous materials when applied on the exposed pulp tissue results in severe inflammation and eventual formation of pulpal abscesses.

In severe injury, destruction extends beyond the cut tubules, often resulting in pulpal abscess and death of the pulp.



Periapical diseases are “an inflammatory conditions occurring around the apex of a tooth caused by a necrotic pulp”

Lesions produced by periapical infection are in close proximity to the apices of the teeth.

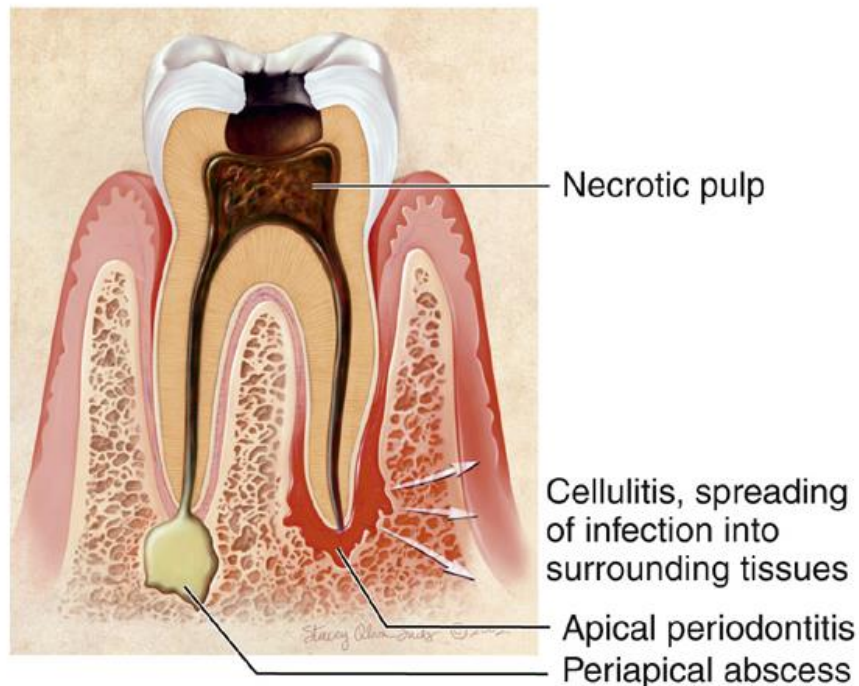
Periapical lesions are recognized by their radiographic appearance and the symptoms they produce.

Most periapical diseases are caused by dead pulps.

The signs and symptoms of periapical disease are related to inflammation.

Acute apical periodontitis:

The bacterial toxins (and sometimes bacteria) that produced the necrosis in the pulp follow the pulp tissue through the apical foramen to the periodontal ligament, resulting in an inflammatory reaction in the periodontium. This inflammation will lead to thickening of the periodontal ligament and manifest itself as tenderness to percussion and chewing.



Pain is the presenting symptom of apical periodontitis it may be provoked by percussion.

The patient may also sense that the affected tooth is higher than nearby ones.

Often the pain is so diffuse that the patient cannot localize it.

Acute apical periodontitis arises so quickly, probably within an hour or two, that bone destruction and its resulting radiographic changes may not be evident.

Chronic Apical Periodontitis:

Generally, chronic apical periodontitis is an asymptomatic periapical lesion that is manifested radiographically.

Bacteria and their endotoxins cascading out into the periapical region from a necrotic pulp cause an inflammatory reaction that produces extensive demineralization of cancellous and cortical bone.

The general absence of symptoms, the presence of a periapical radiolucency, and the confirmation of pulp necrosis confirm the diagnosis of chronic apical periodontitis.



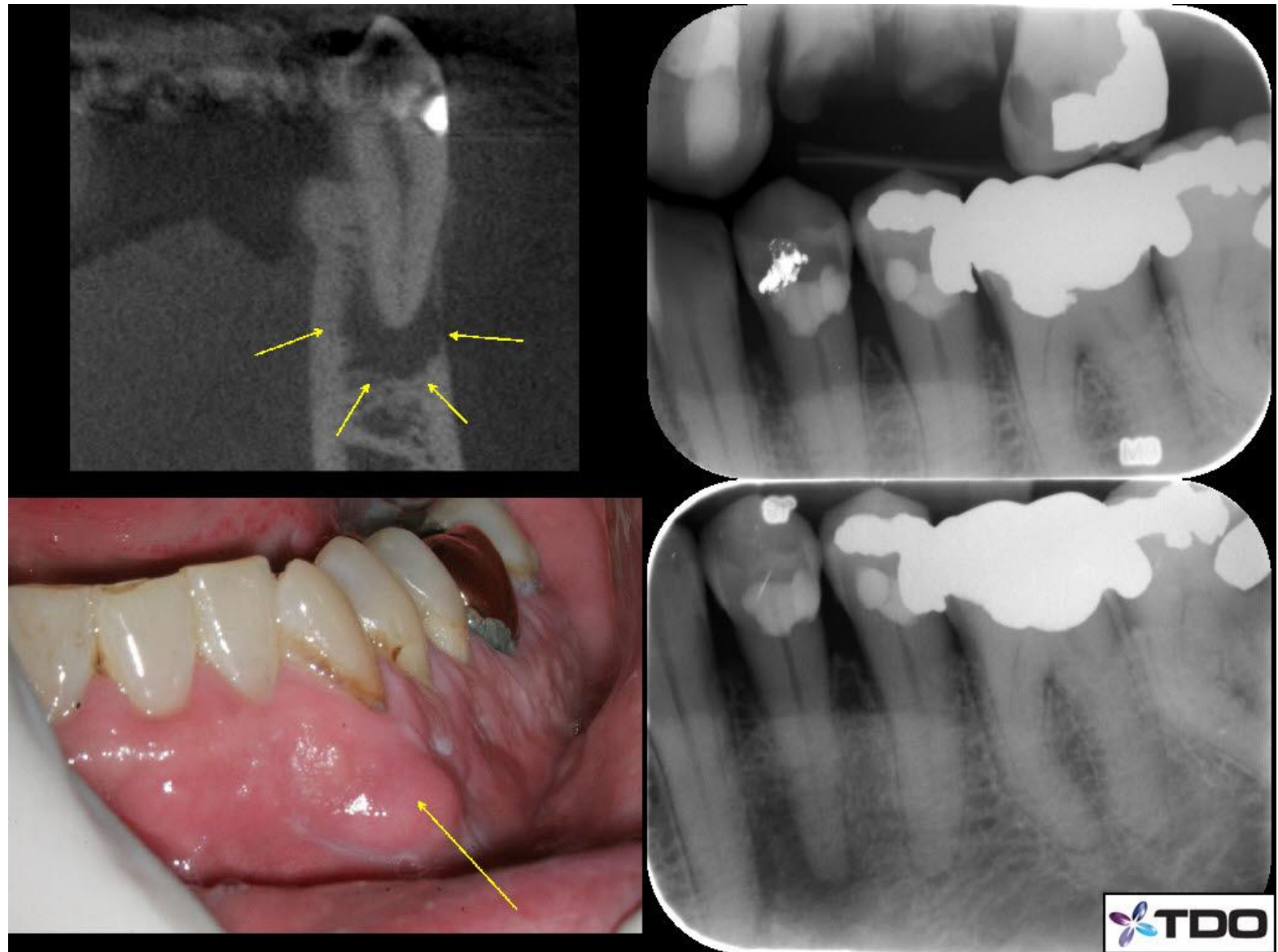
Acute Periradicular Abscess:

An acute periradicular abscess consists of a painful purulent exudate (abscess) around the apex. This abscess is the result of the exacerbation of acute apical periodontitis from an infected, necrotic pulp.

The signs and symptoms of acute periradicular abscess include rapid onset of slight-to-severe swelling, moderate-to severe pain, pain from percussion and palpation, and the possibility of a slight increase in tooth mobility. In more advanced cases, the patient is febrile.

Such type of abscess may known as:

- Periradicular abscess (along the root)
- Periapical abscess(around the root tip)
- Apical Abscess (at the root tip)
- DentoAlveolar abscess



CLINICAL SIGNS & SYMPTOMS

- Acutely painful to biting pressure, percussion & Palpation
- No Response to Electric Pulp Testing .
- Varying degrees of Mobility.
- Tooth may be elevated in alveolar socket
- Patient may exhibit raised temperature and malaise.
- The cervical & submandibular lymph nodes tender to palpation.



INTRODUCTION TO ENDODONTICS

Lecture No. 1 Endodontics



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NSc. 1994

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Conference and symposiums 22

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Endodontology: is the branch of dental sciences concerned with the form, function, health, injuries to and the diseases of the dental pulp and periradicular region, and their relationship with systemic well-being and health.

Endodontic treatment: can be defined as the prevention and or treatment of pulp, apical periodontitis, and principal disease.

THE MAIN AIM OF THE ENDODONTIC THERAPY INVOLVES TO:

- i. Maintain vitality of the pulp.
- ii. Preserve and restore the tooth with damaged and necrotic pulp.
- iii. Preserve and restore the teeth which have failed to the previous endodontic therapy, to allow the tooth to remain functional in the dental arch.

OBJECTIVE OF ENDODONTIC TREATMENT

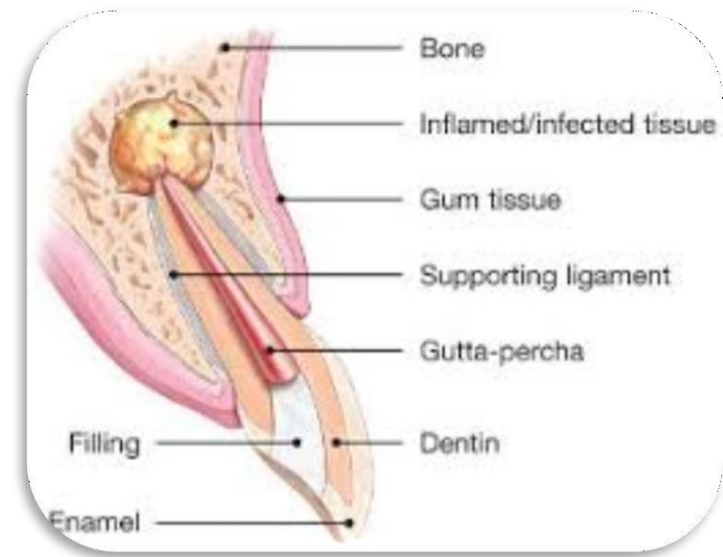
The primary objective of endodontic therapy is to create a biologically acceptable environment within the root canal system which allows the healing and continued maintenance of the health of the peri-radicular tissue.

This objective can be achieved by:

- 1 Eliminating the bacteria (source of infection) from within the root canal system
- 2 Sealing the root canal and tooth to prevent re-infection

SCOPE OF ENDODONTICS

1. The differential diagnosis and treatment of orofacial pain of pulpal and peri radicular origin
2. Prevention of pulpal disease (DPC & IDPC) and vital pulp therapy (pulpotomy)
3. Root canal treatment
4. Retreatment of endodontically treated teeth
5. Management of post-treatment endodontic disease
6. Surgical endodontics
7. Bleaching of endodontically treated teeth
8. Treatment of traumatized teeth.



INDICATIONS OF ENDODONTIC THERAPY

1. Irreversible pulpitis
2. Necrosis or gangrene of the pulp tissue
3. Prosthetic reasons: severely tilted teeth
4. Intentional pulp extirpation (badly broken vital teeth for post placement to get retention)
5. Injury (trauma)

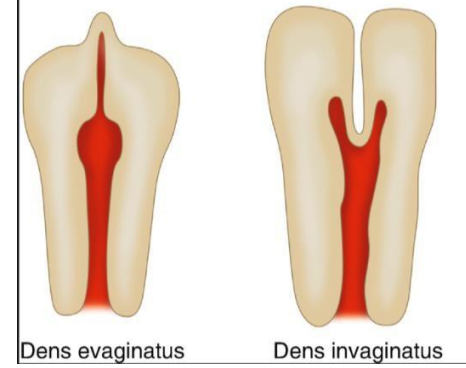
CONTRAINDICATIONS OF ENDODONTIC THERAPY

Factors related to the patient:

1. Lack of time (patient, doctor)
2. Economical reason
3. Restless patient (Down syndrome, Parkinsonism)
4. Lack of interest from the patient
5. Poor hygiene Patient prefers other solution (denture, fixed bridge)



Technical factors that prevent RCT:



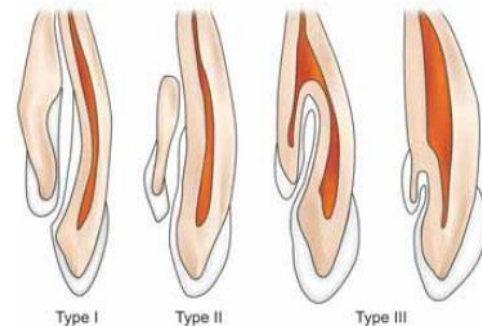
Dens evaginatus

Dens invaginatus



be careful about clinical Abnormality of cingulum

1. Anatomy (e.g. dens in dente, dens **invaginatus** & dens **indente**)
2. Un-retrievable fractured instrument in the canal
3. Non removable root canal filling or cast post
4. Unsatisfactory armamentarium of the dentist



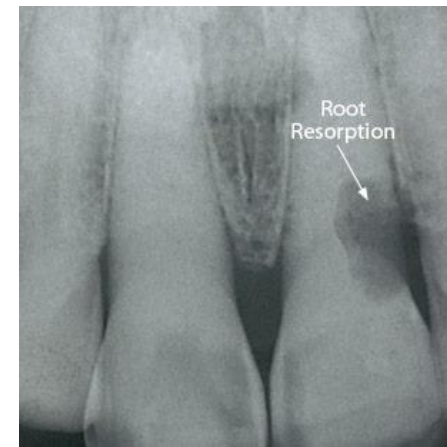
Type I

Type II

Type III

Conservative:

1. Heavy marginal periodontitis
2. Vertical fracture of the root
3. Deep root caries
4. Huge resorption of the root
5. Tooth with no importance (mostly wisdom tooth)



BASIC PHASES OF ROOT CANAL TREATMENT

1The **Diagnostic phase** in which the disease to be treated is "determined" and the treatment plan developed.

2The **Preparatory phase** in which the contents of the root canal are removed, and the canal is prepared to receive a filling material.

3The **obliteration Phase** in which the canal is filled or obliterated with an inert material to obtain an adequate seal as close as possible to C.D.J. (cementodentinal junction).

If there is a defect in any phase, the endodontic treatment will not be succeeded

Instruments used in Endodontics

Dr. Nawfal Zakarya

BDS., MSc., PhD. Conservative dentistry

BASIC INSTRUMENT PACK

1- Mouth mirror



2- Endodontic explorer
DG16



3- Long spoon excavator

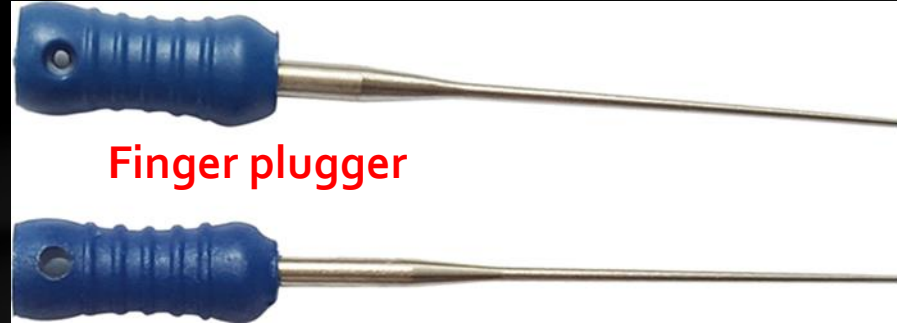
4- Locking tweezers



5- Periodontal probes.



6- Endodontic plugger.



7- Millimeter ruler.



8- Spreader



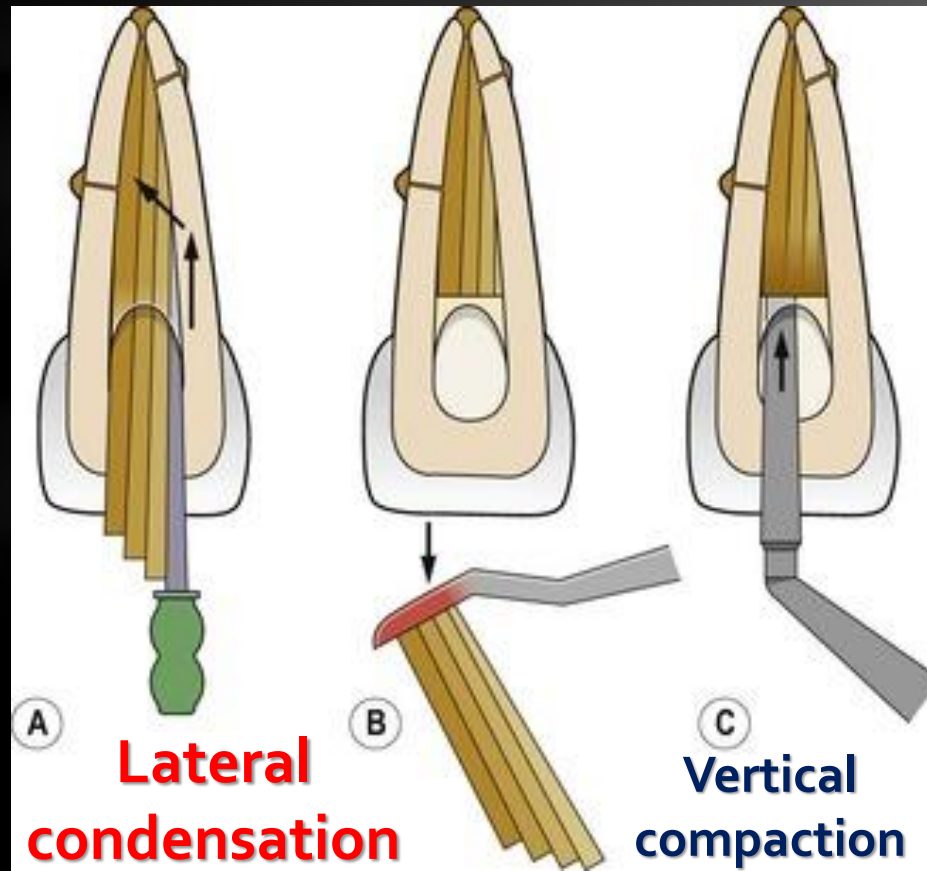
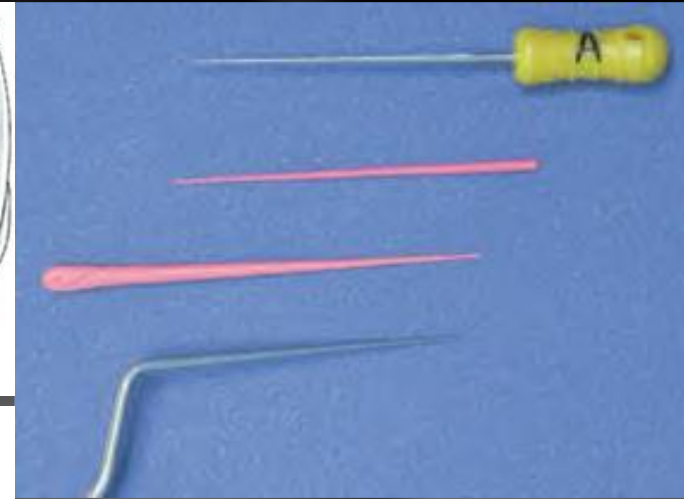
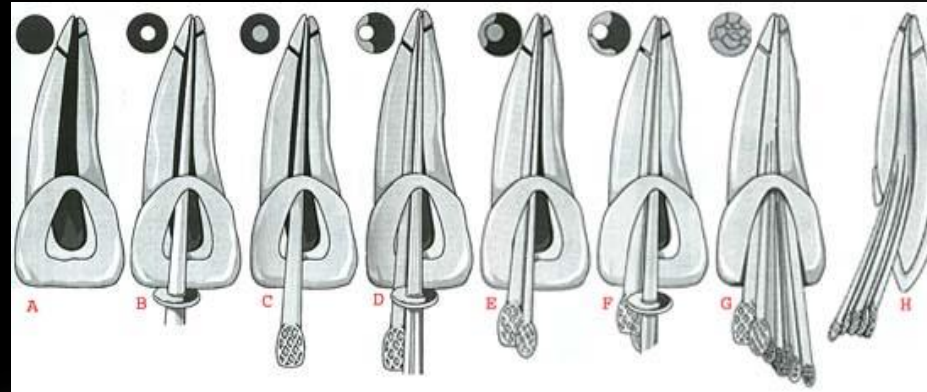
INSTRUMENTS FOR FILLING ROOT CANALS

Spreaders

Lateral condensation

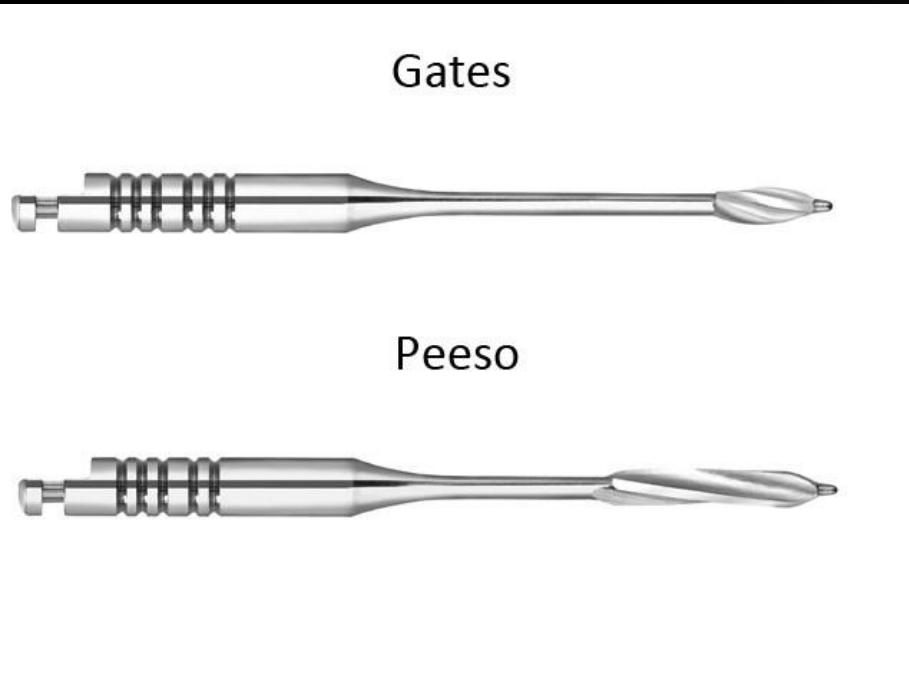
Pluggers

Vertical compaction



1- **Gates-Glidden bur**: is operated at low-speed. It may be used for **coronal root canal enlargement** and to remove gutta-percha from the coronal part of the root canal in retreatment cases.

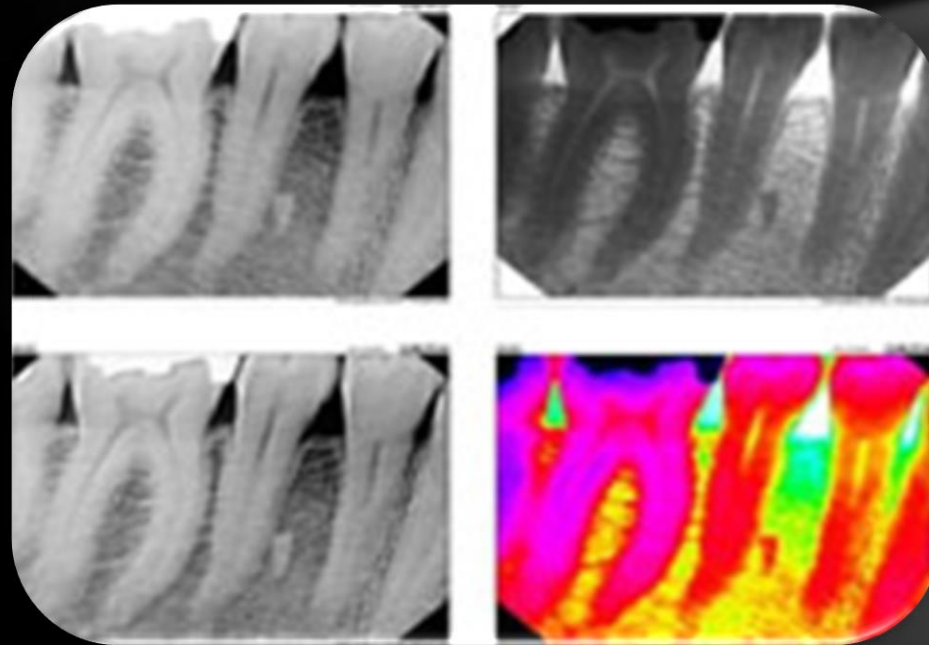
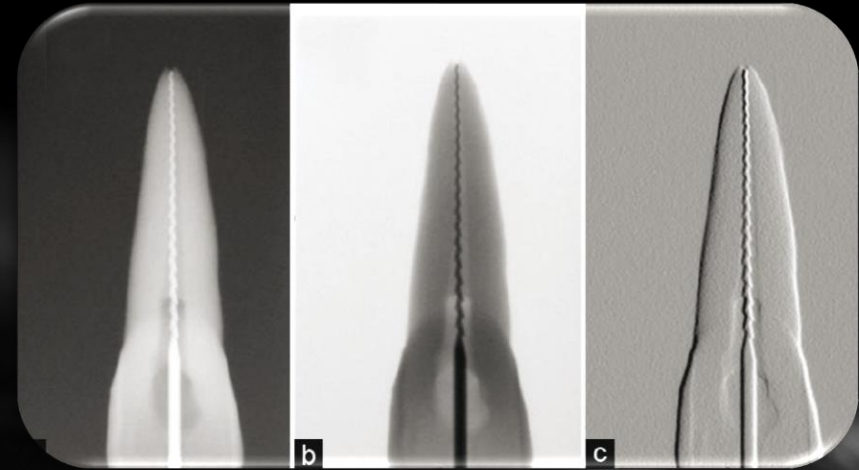
2- **Peeso bur**



DEVICES TO DETERMINE WORKING LENGTH

Radiographs:

An instrument is placed into the root canal and then a conventional or digital radiograph taken. The length of the instrument is measured from the silicon stopper to the tip of instrument.



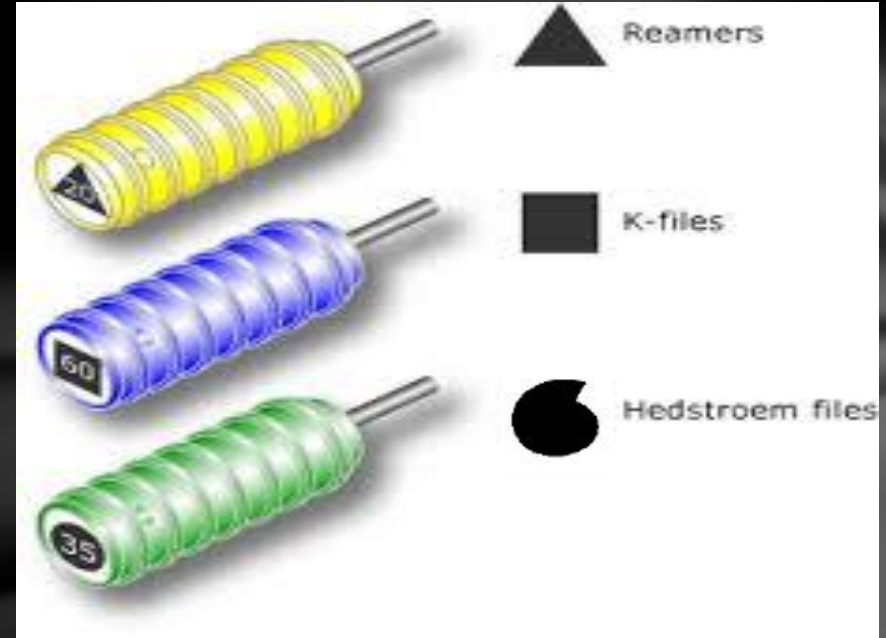
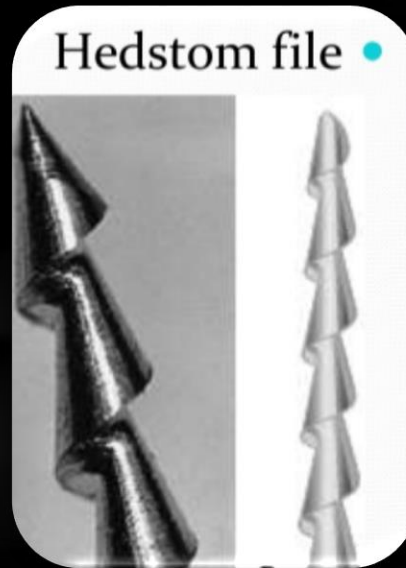
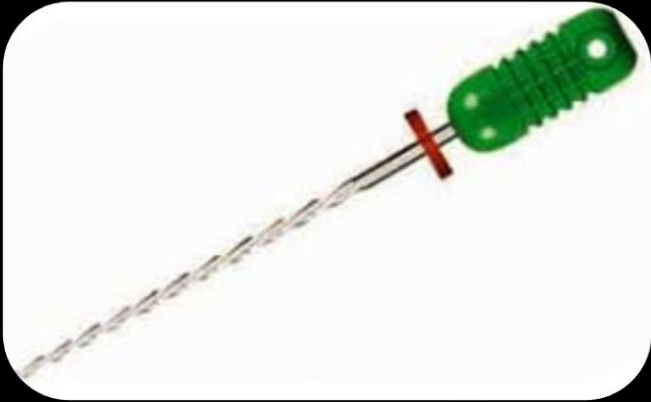
Electronic Apex Locators

A typical EAL has a meter or digital display and two electrodes. One electrode is fashioned into a hook and placed into the oral cavity. The other electrode, in the form of a spring-loaded clip or probe is attached to the endodontic file, which is inserted into the root canal.



INSTRUMENTS FOR ROOT CANAL PREPARATION

Hand instruments



Power assisted instruments



ALLOYS USED FOR MANUFACTURING ENDODONTIC INSTRUMENTS

1. Carbon Steel Alloy
2. Stainless Steel Alloy
3. Nickel-Titanium Alloy

Carbon steel alloy

These alloys contain less than 2.1 % of carbon.

Advantage: they have higher hardness than stainless steel instruments

Disadvantages:

1. Prone to corrosion
2. Can not be autoclaved Prone to rust. eg. Barbed broaches

Stainless steel alloys

These are corrosion resistant alloys, they contains **18% chromium**, **10% nickel** and **0.12% carbon**.

Advantages: corrosion resistant.

Disadvantages:

1. Stiff in nature
2. Prone to distortion
3. Prone to fracture

Eg. K-file, H-file, reamer.

Nickel-titanium alloys

These alloys contain 50% nickel and 50% titanium

The NiTi alloys have many interesting properties:

1. A shape memory effect (ability to return perfectly to its original shape).
2. Super elasticity (low modulus of elasticity).
3. Good biocompatibility.
4. High corrosion resistance.

5. Have greater flexibility in bending.
6. Better resistance to torsional fracture.
7. Cannot be easily pre-curved. **Except heat treated (bendable)**
8. Their cutting efficiency is dependent on cross-sectional shape but is less aggressive compared with stainless steel instruments.
9. NiTi instruments tend to straighten curved root canals **less than** stainless steel instruments.

▪
















HAND INSTRUMENTS

Endodontic hand instruments are standardized in relation to **size, color coding** and physical properties according to the **International Standardization Organization (ISO)**.

d₁ is an assessment of the diameter of the working part at the tip end.

d₂ represents a point at **16** mm from **d₁** where the cutting part of the instrument ends.

Table 6.1 Nominal sizes, diameters, and colour coding of standardized root canal files and reamers

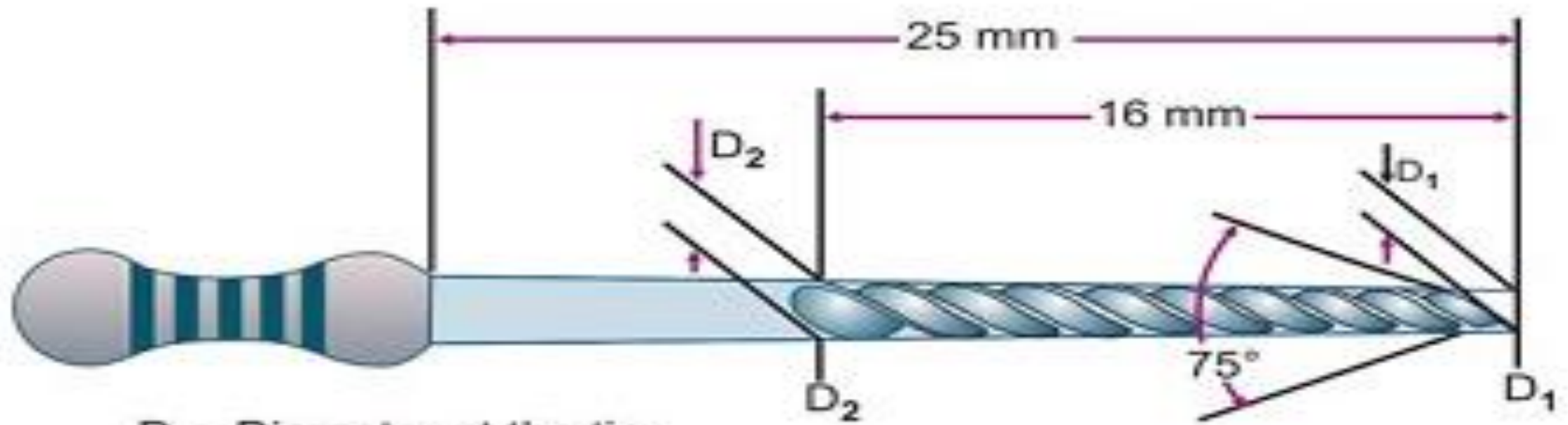
SIZE	d ₁ (mm)	COLOUR
006	0.06	Orange 
008	0.08	Grey 
010	0.10	Purple 
015	0.15	White 
020	0.20	Yellow 
025	0.25	Red 
030	0.30	Blue 
035	0.35	Green 
040	0.40	Black 
045	0.45	White 
050	0.50	Yellow 
055	0.55	Red 
060	0.60	Blue 
070	0.70	Green 
080	0.80	Black 
090	0.90	White 
100	1.00	Yellow 
110	1.10	Red 
120	1.20	Blue 
130	1.30	Green 
140	1.40	Black 

#08	Gray only available in #08	
#10	Purple only available in #10	
#15	#45	#90
#20	#50	#100
#25	#55	#110
#30	#60	#120
#35	#70	#130
#40	#80	#140



ISO taper of 2% that equals to 0.02mm increase in diameter per mm of file.

The lengths of instruments available are normally 21, 25 and 31 mm.



D_1 - Diameter at the tip

D_2 - Diameter at end of cutting blade (16 mm from D_1)

Tip angle is $75^\circ \pm 15^\circ$

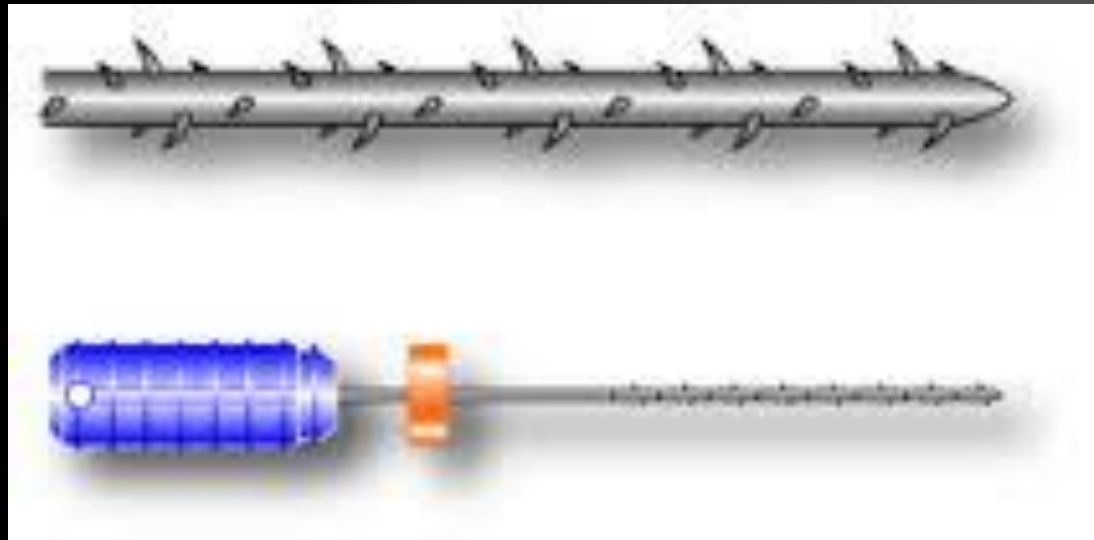
Up to this slide

TYPES OF HAND INSTRUMENTS

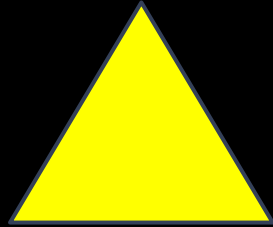
Barbed broaches

Barbed broaches are used mainly for the removal of pulp tissue from wide root canals, and cotton wool dressings from the pulp chamber.

Barbed broaches are made from soft steel wire, the barbs are formed by cutting into the metal and distending the cut portion away from the shaft.



Reamers

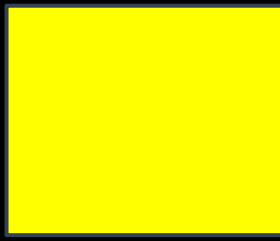


Reamers are manufactured by twisting a tapered stainless-steel blank to form an instrument with sharp cutting edges along the spiral.

They are used with a **half turn twist and pull** action “**reaming action**” which shaves the canal wall, removing dentine chips from the root canal.



FILES



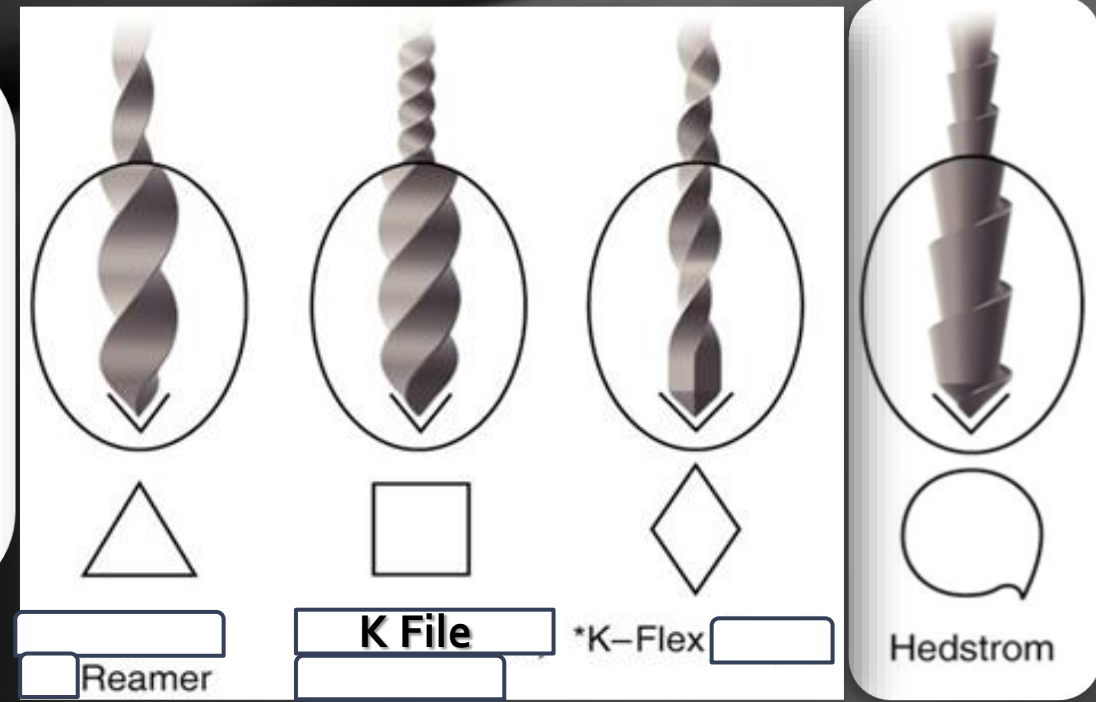
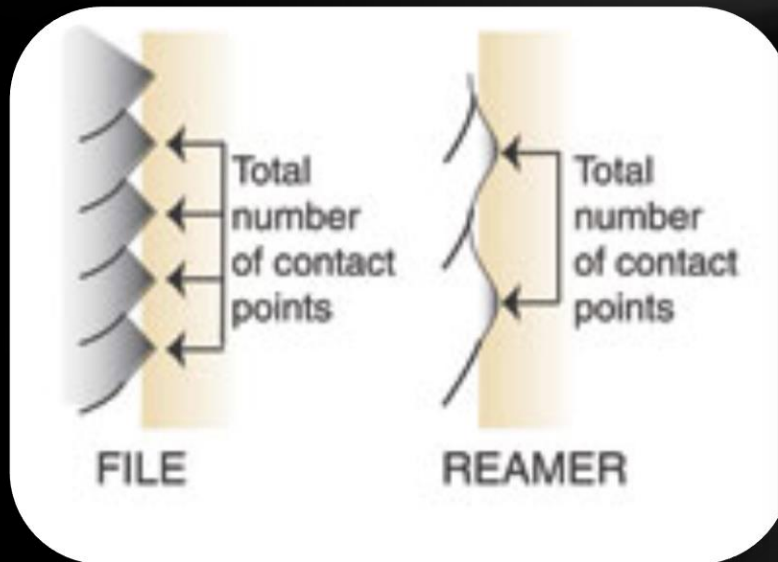
Files are mostly made from stainless steel. they are predominantly used with a filing or rasping action, in which there is little or no rotation of the instrument in the root canal. They are square in cross section.

Files include:

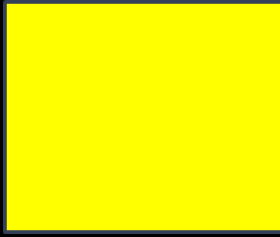
K-file

K-flex file

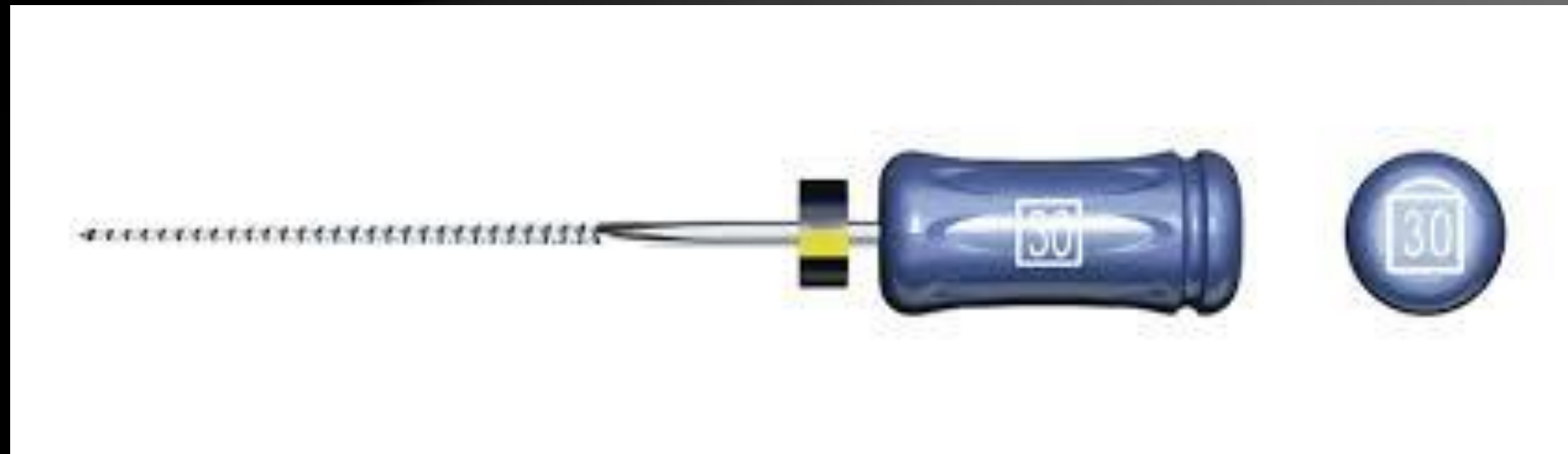
Hedstrom file.



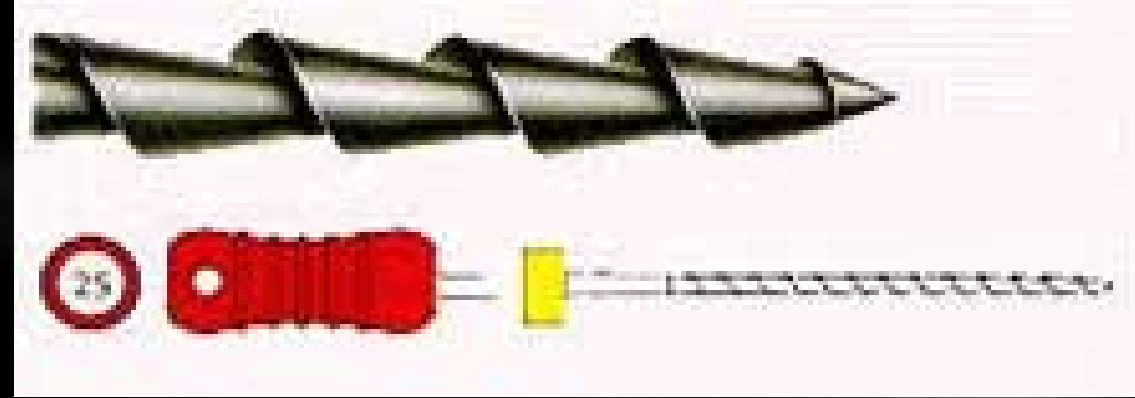
K-file



These files are made, like reamers, by twisting a square blank. They will work either in a reaming or a push-and-pull filing motion.



Hedstrom file



The Hedstrom file is made by a milling process from a steel blank of round cross-section to produce elevated cutting edges.

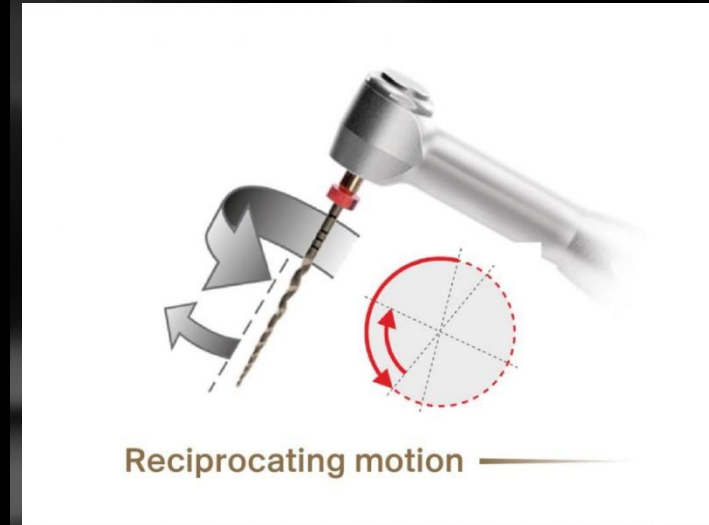
Although the design leads to a sharp and flexible instrument, the file is inherently weaker due to the reduced shaft diameter and is therefore **more prone to breakage in small size**.

It is most effective when used in a pull motion.

With sharp cutting flutes, it is also used to engage and remove retained instruments, gutta-percha and silver points.

Power-assisted root canal instruments

Reciprocating handpieces
eg. Wave One System



Rotary NiTi instruments
Eg. ProTaper Universal System



Armamentarium

- Rubber dam sheet.
- Rubber dam clamps.
- Rubber dam forceps clamp holder.
- Rubber dam frame.
- Rubber dam punch.

Accessories

- Lubricant/Petroleum jelly.
- Dental floss.
- Rubber dam Napkin.



Rubber Dam Sheet



It is made of latex or non-latex.

Available in 2 sizes- ① 5"*5"

② 6"*6"

New material should be used.

Available in varying thickness heavy-medium & thin.

Light and dark sheets are available for colour contrast.

Has a shiny & dull side, dull side will be facing the occlusal side.

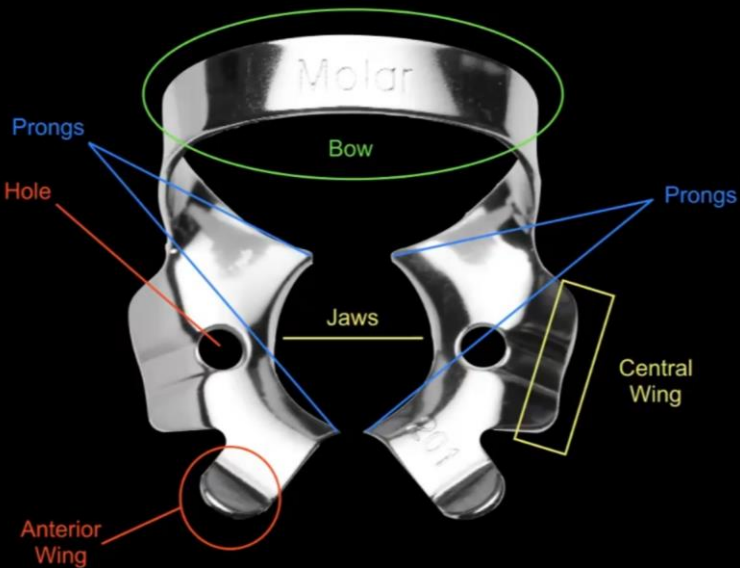
Applying the napkin



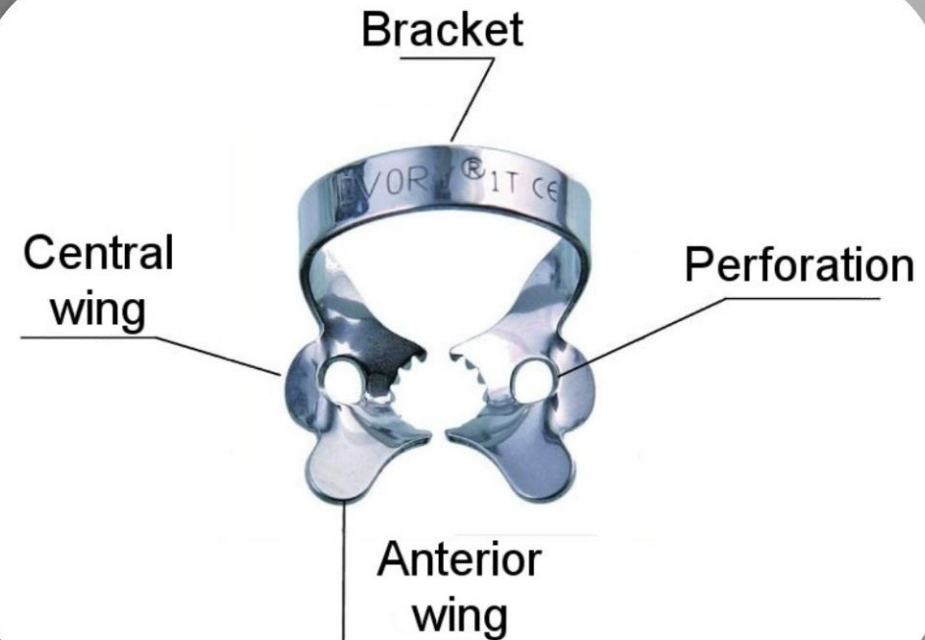
Rubber Dam Clamps

- ✓ These are used to fix the dam to the teeth, that are to be isolated.
- ✓ These also minimally retract the gingiva
- ✓ Subdivided into >Winged

>Wingless

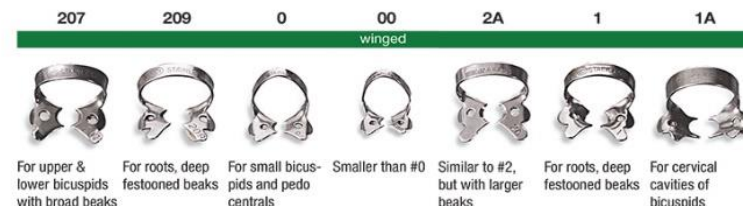
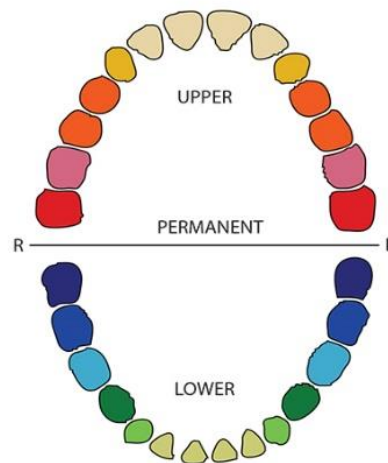
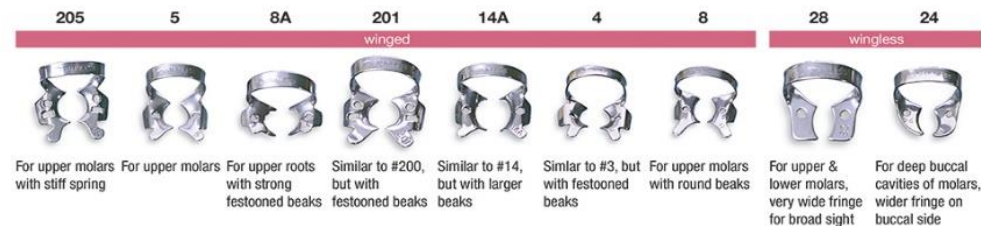
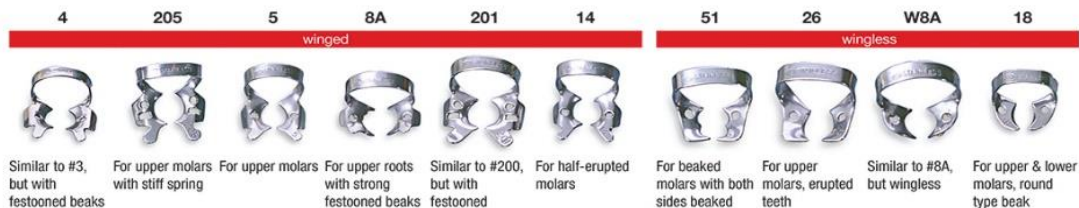


wingless clamp



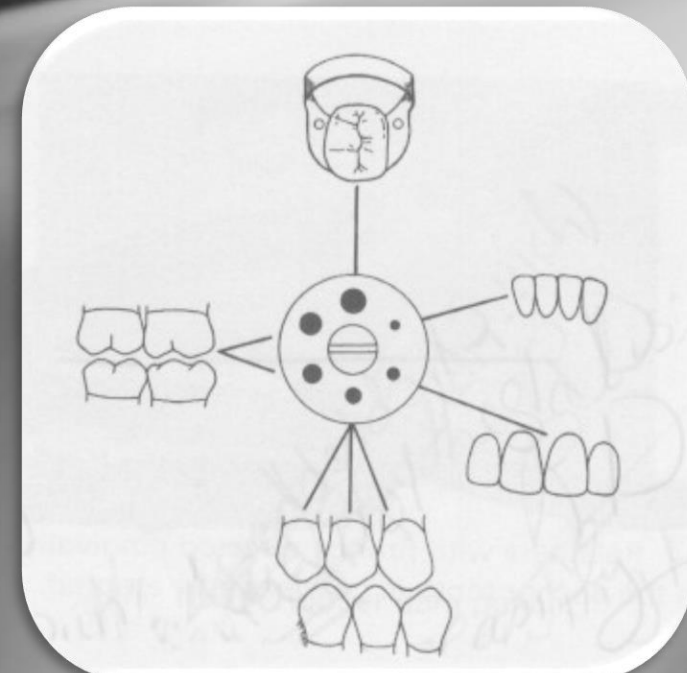
Clamps with wings

For permanent teeth

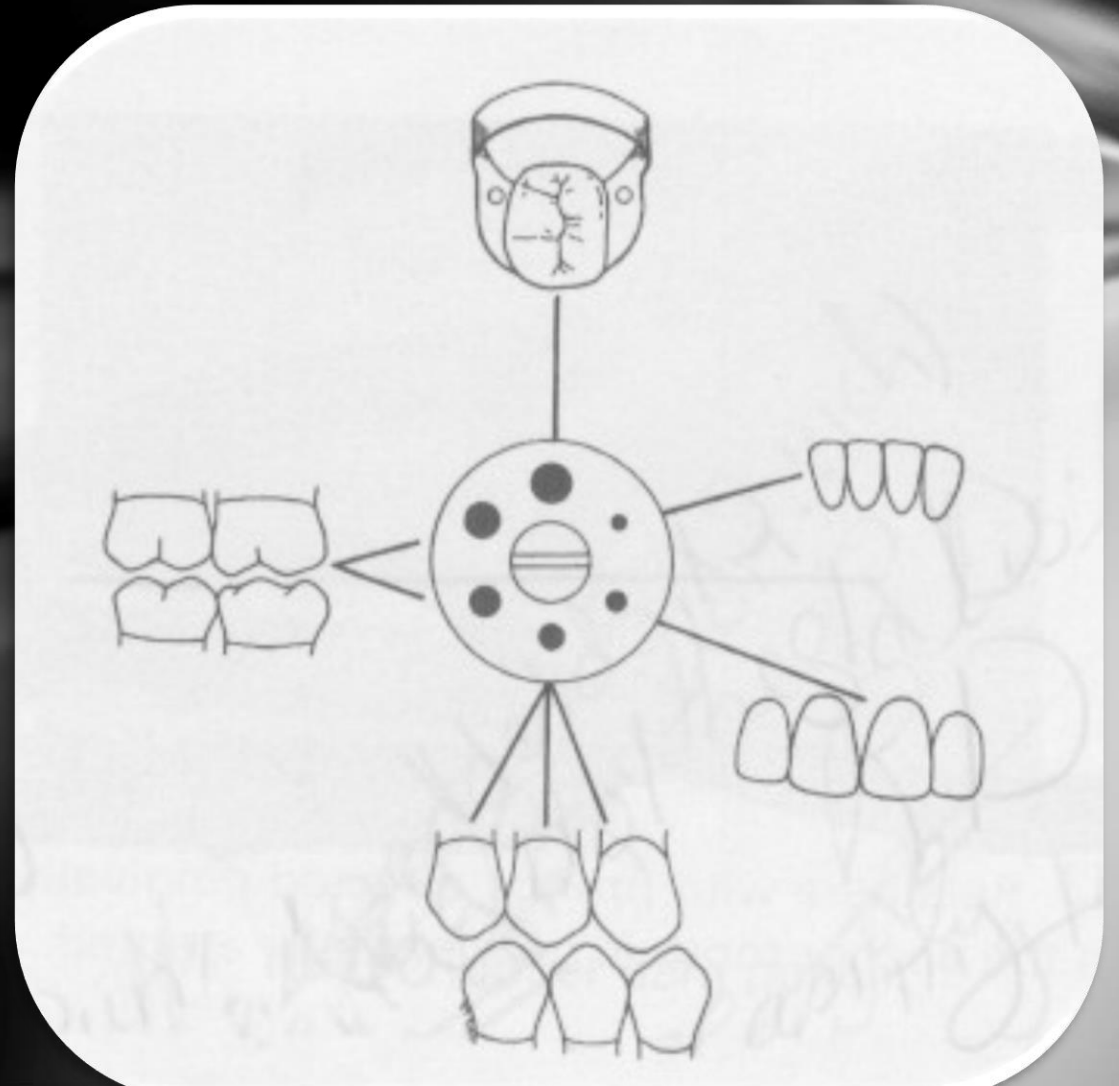


Rubber Dam Punch

- Rubber dam punch is used to make the holes in the sheet through which the teeth can be isolated.
- The working end is designed with a plunger on one side and a wheel on other side.
- This wheel has holes of different sizes on the flat surface facing the plunger.
- The punch must produce a clean cut every time.

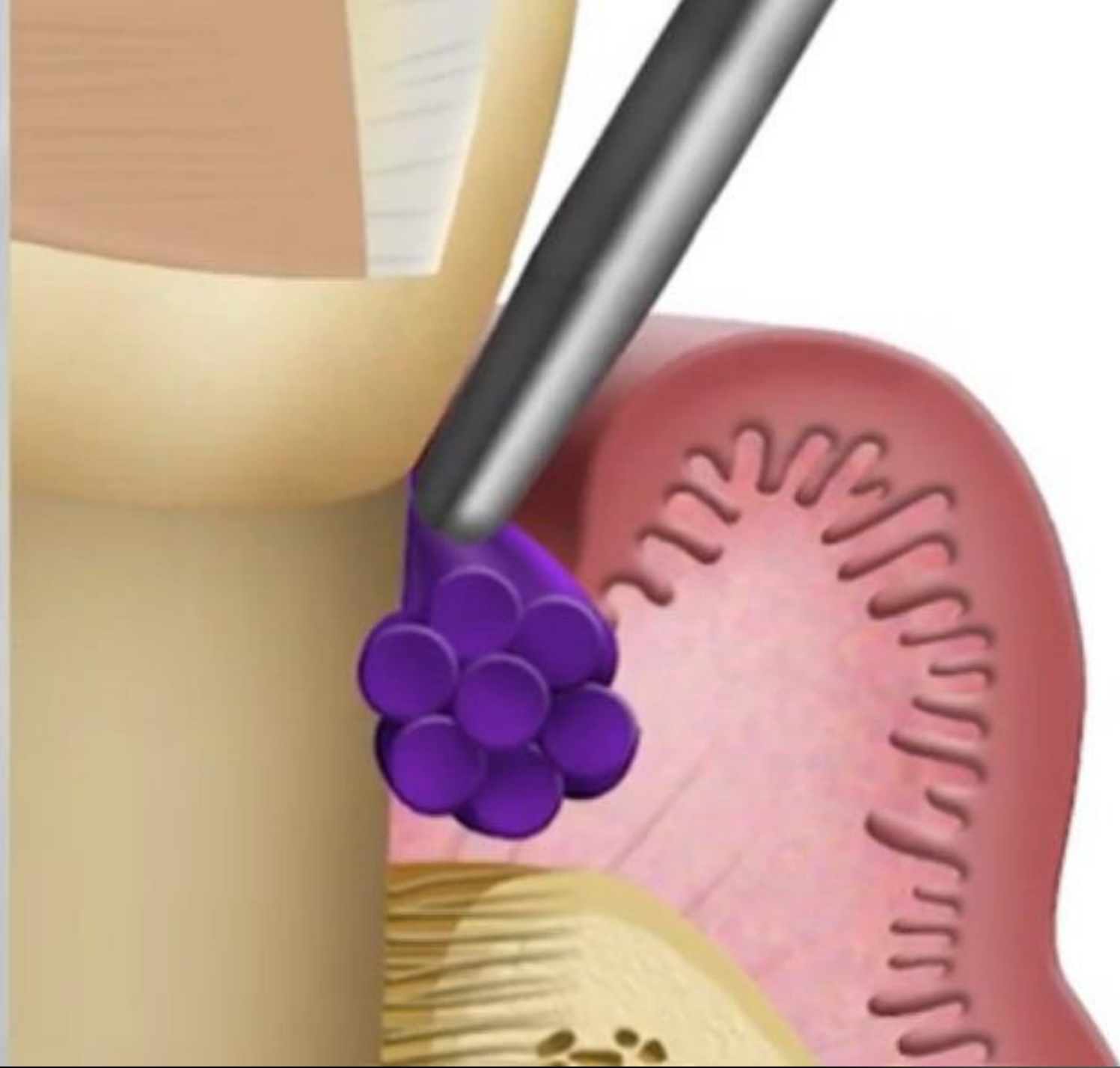


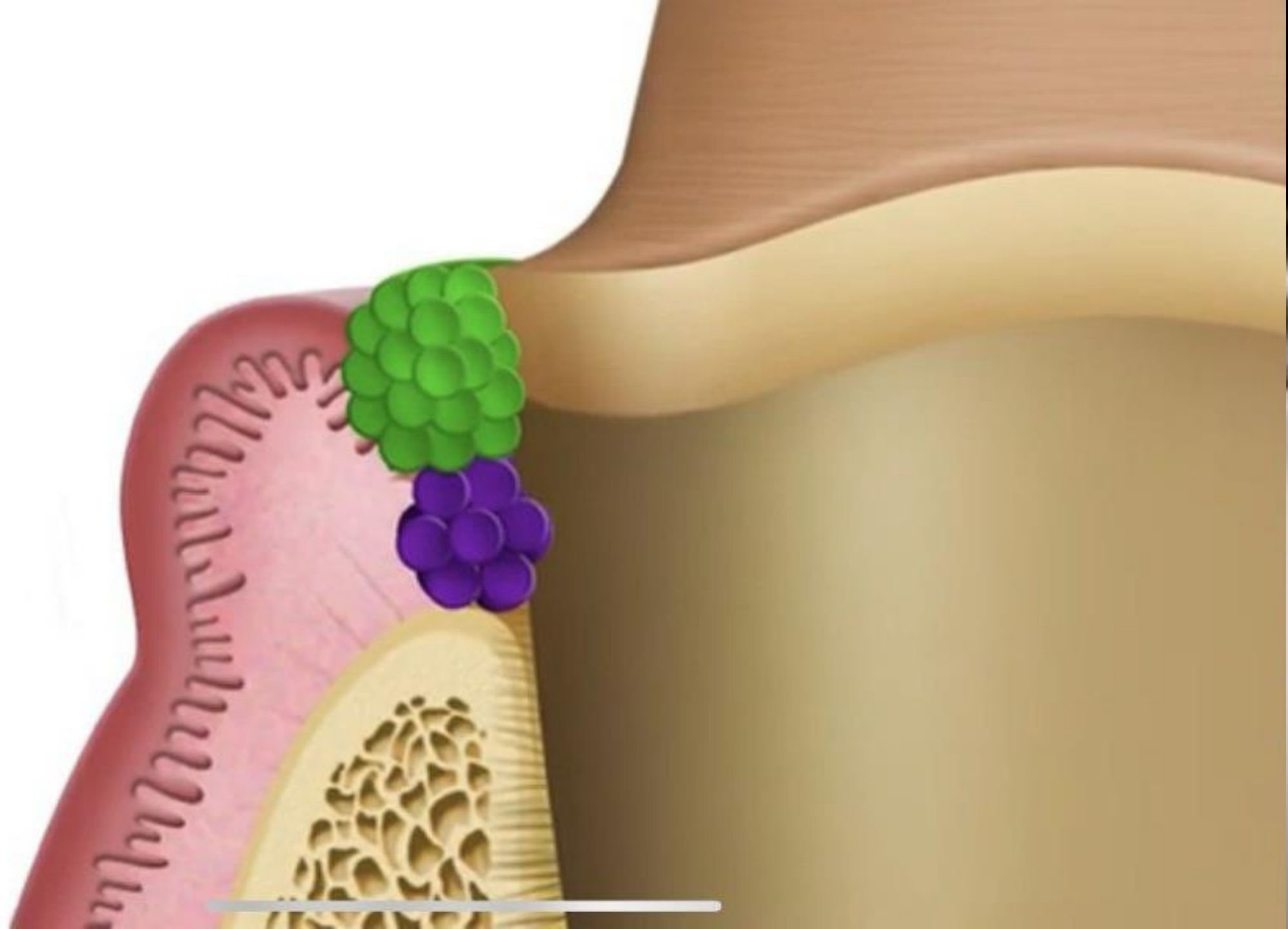
2. Punching the holes



Clamp forceps







Endodontic Access Cavity Preparation

Dr. Nawfal Zakarea

BDS., MSc., PhD., Cons. Dentistry

The main function of an access cavity is

1- To create an unimpeded pathway to the pulp space and the apical foramen of the tooth.

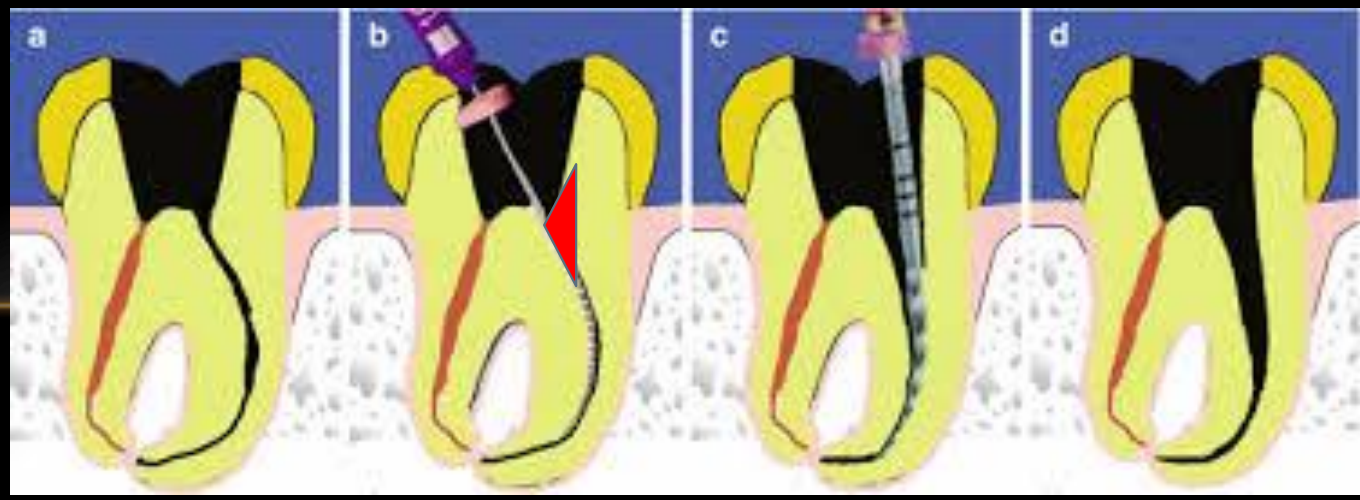
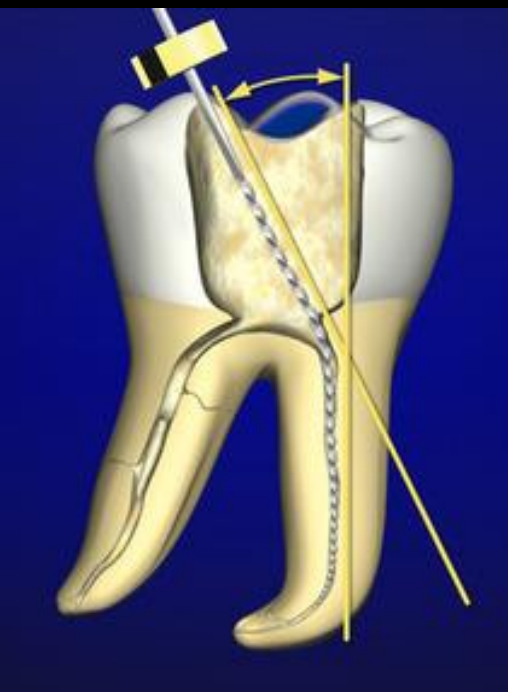
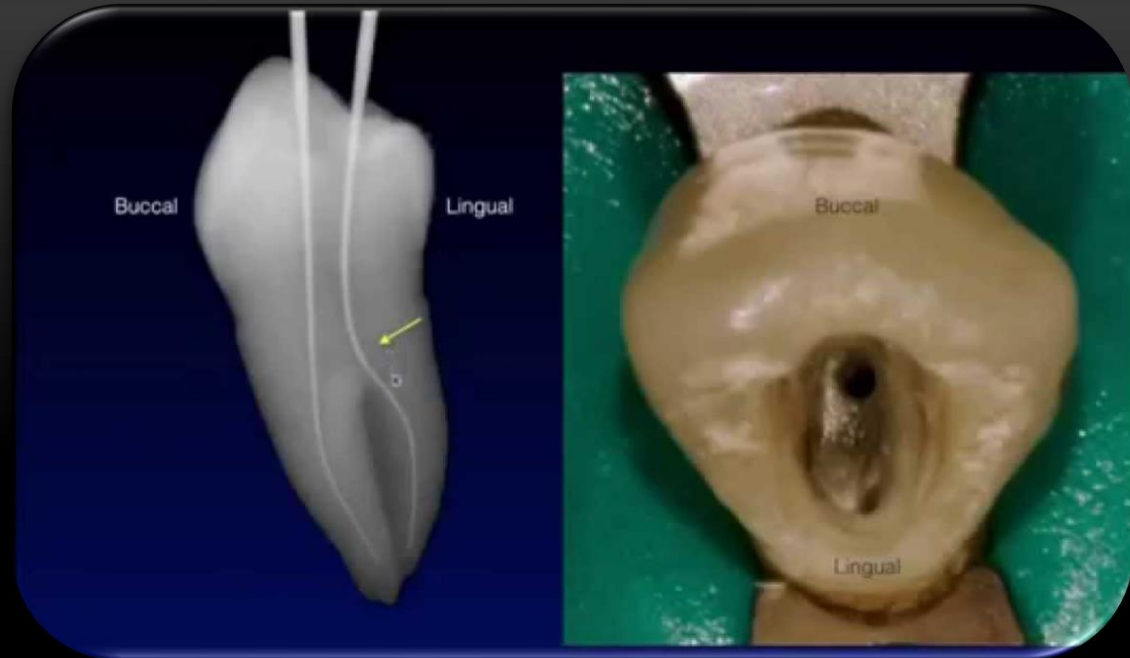
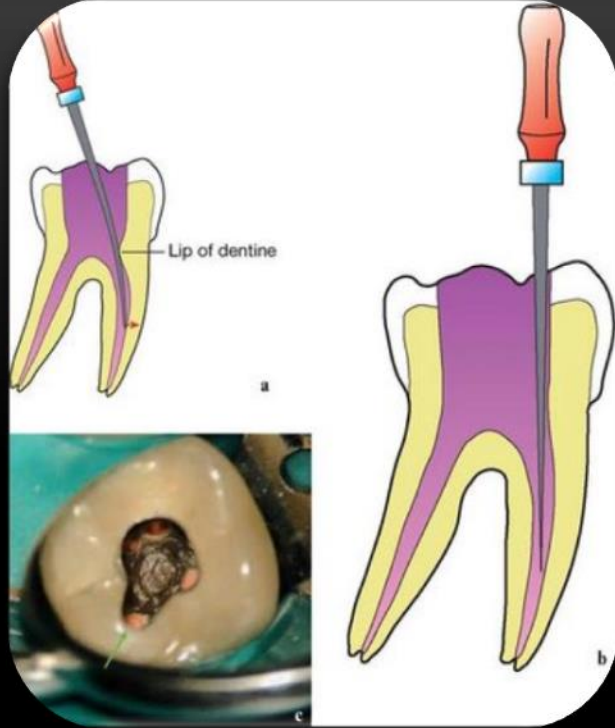
2- Straight-Line Access

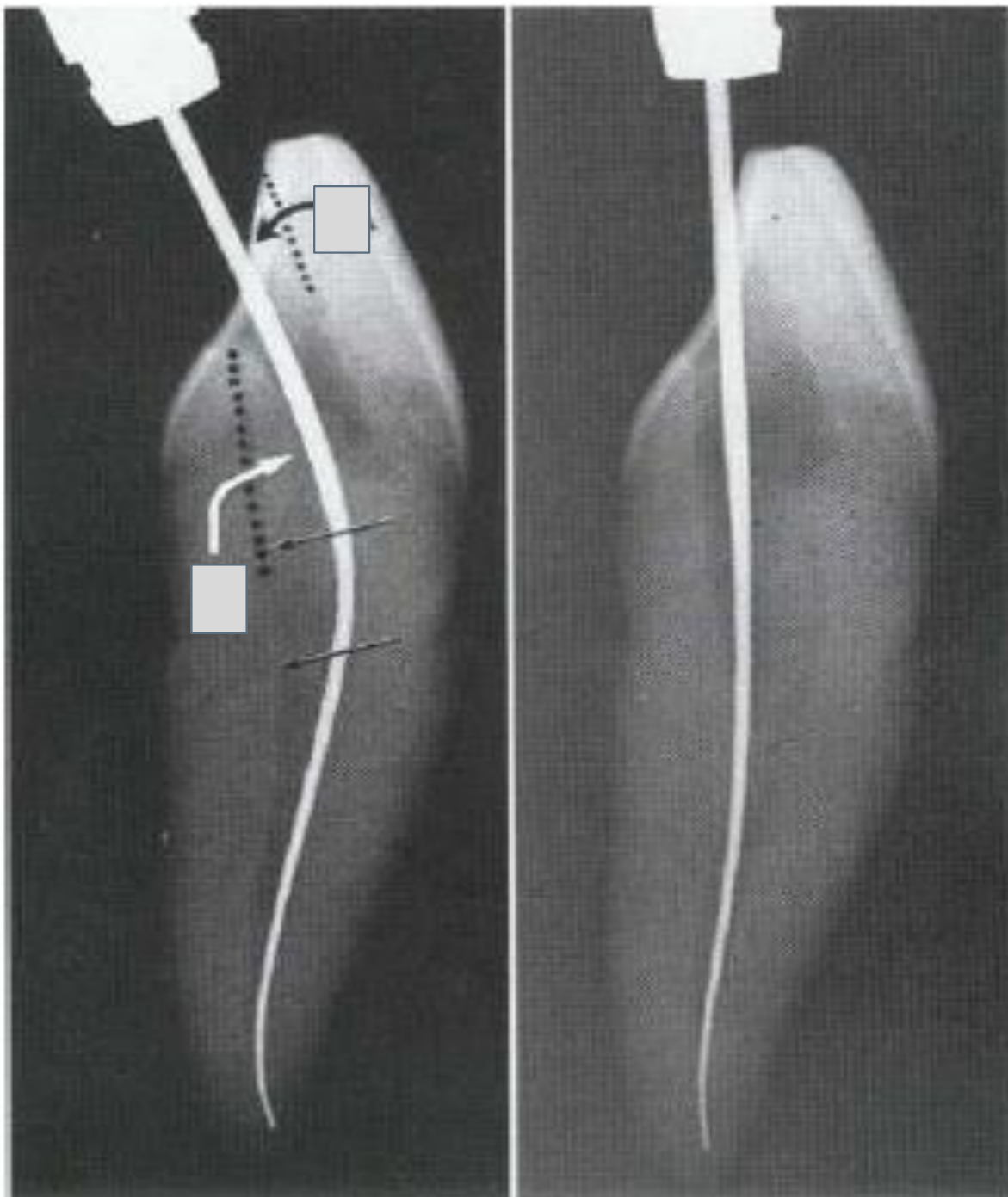
3- prevention of iatrogenic problems and avoidance of technical failure of root canal treatment.

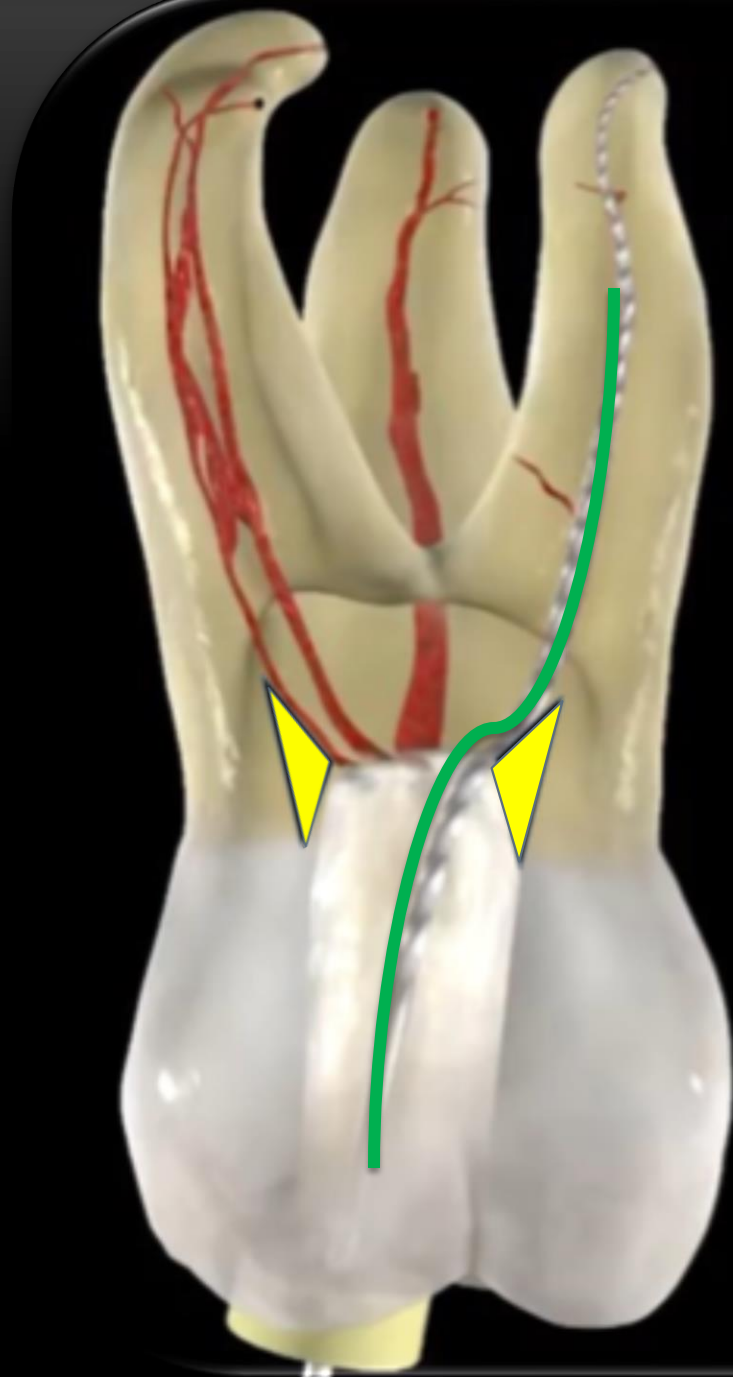
4- Give good vision

5- To locate all root canal orifices

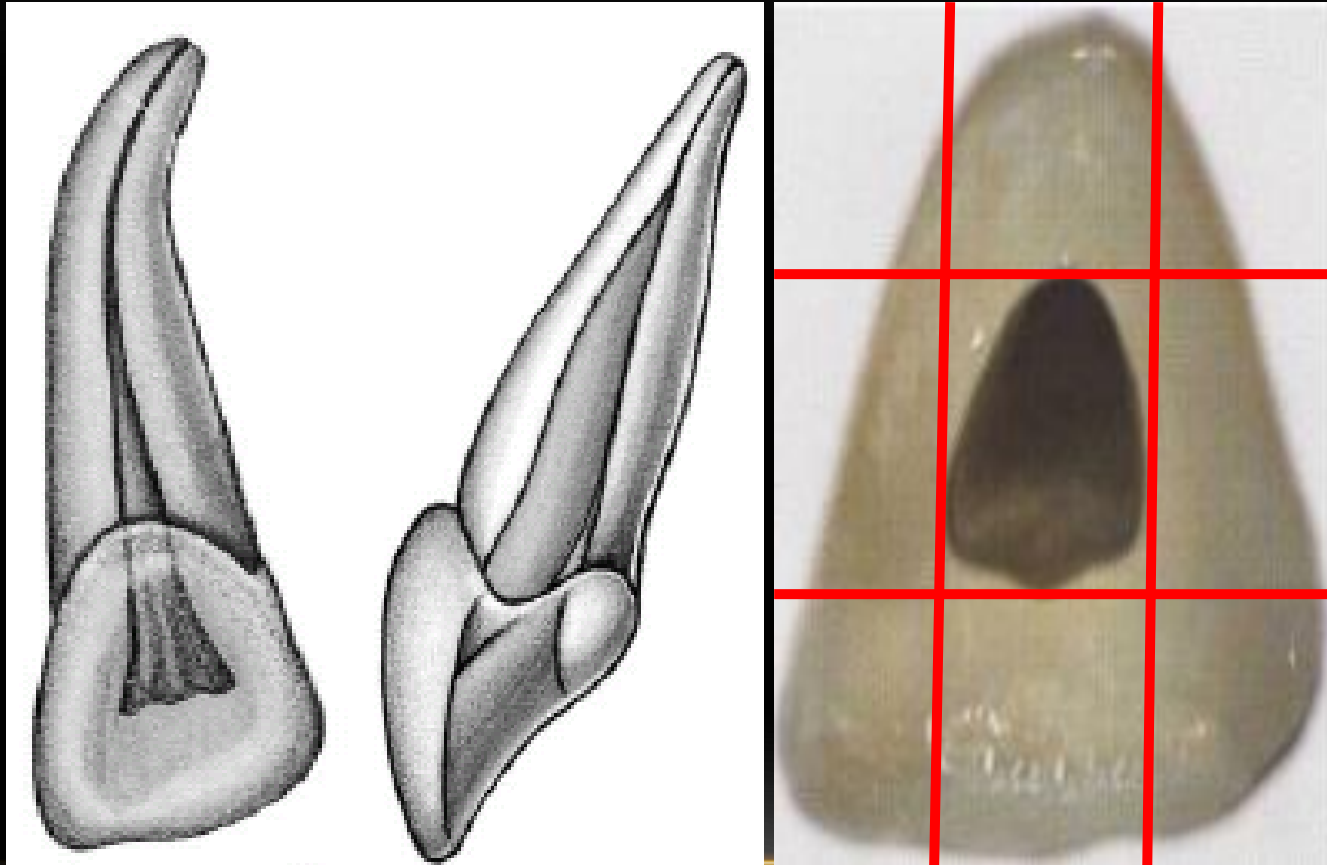
6- To conserve sound tooth structure

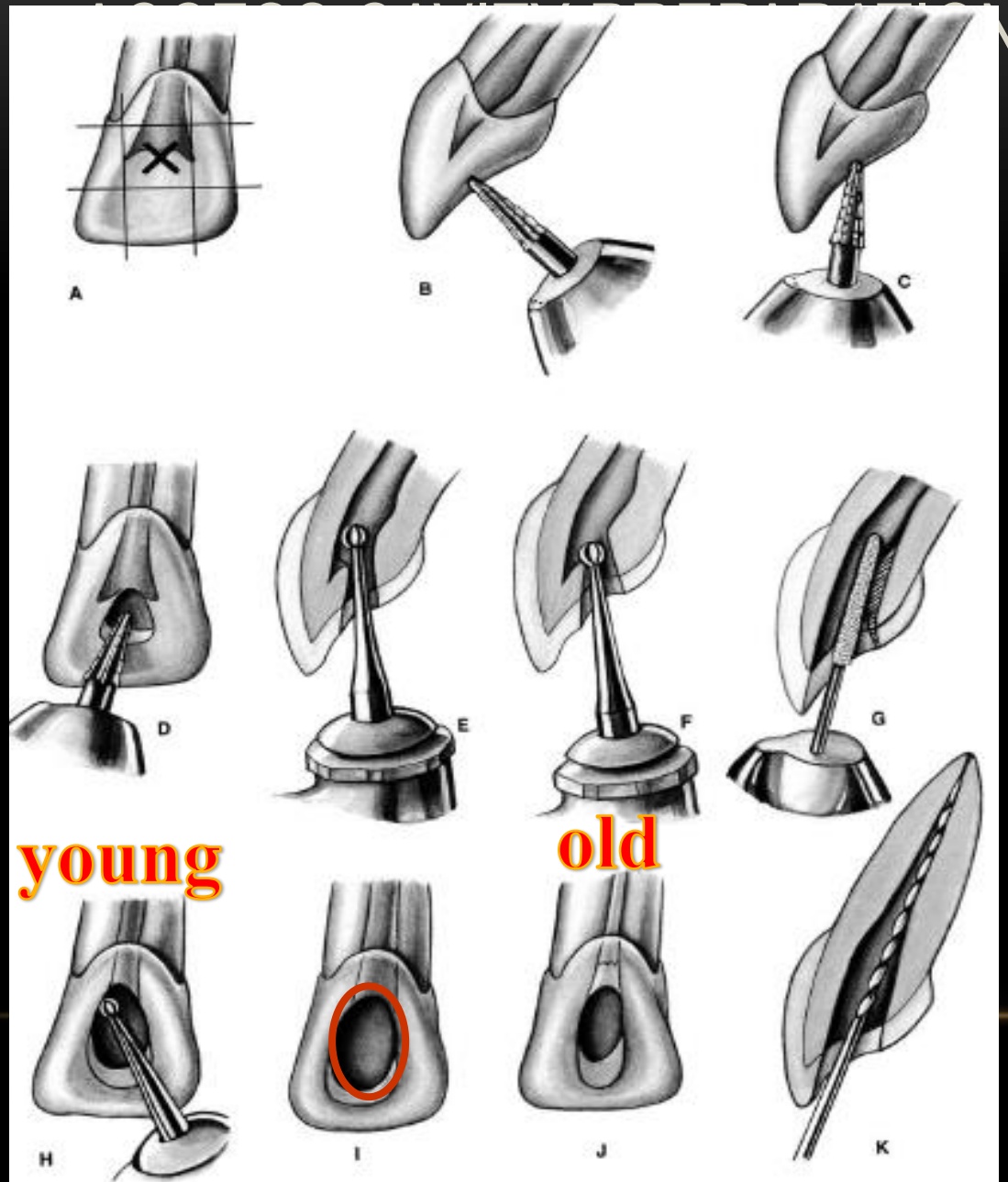


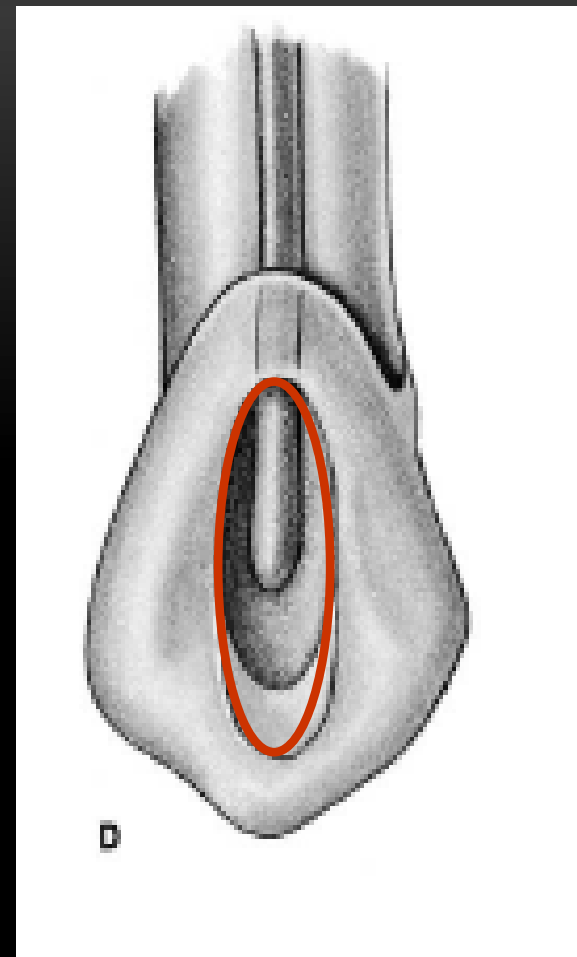
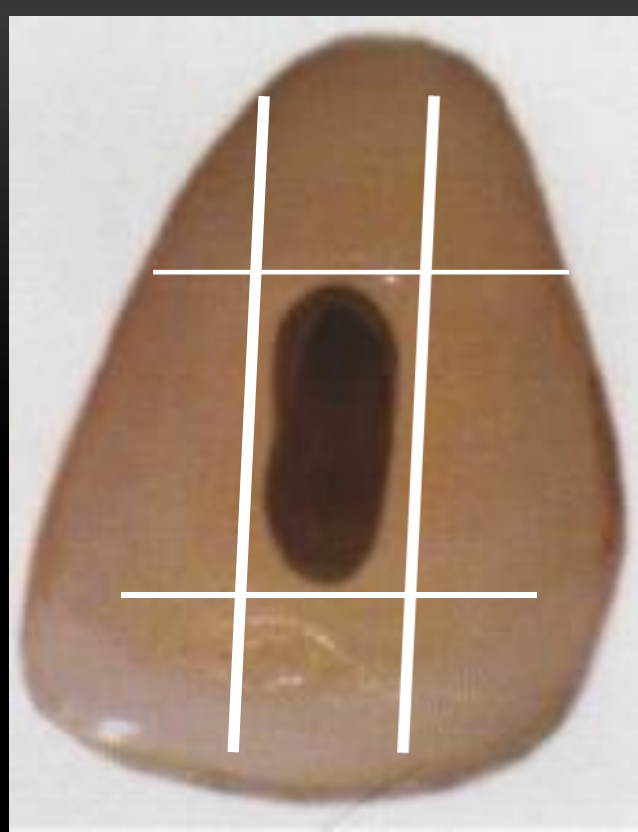
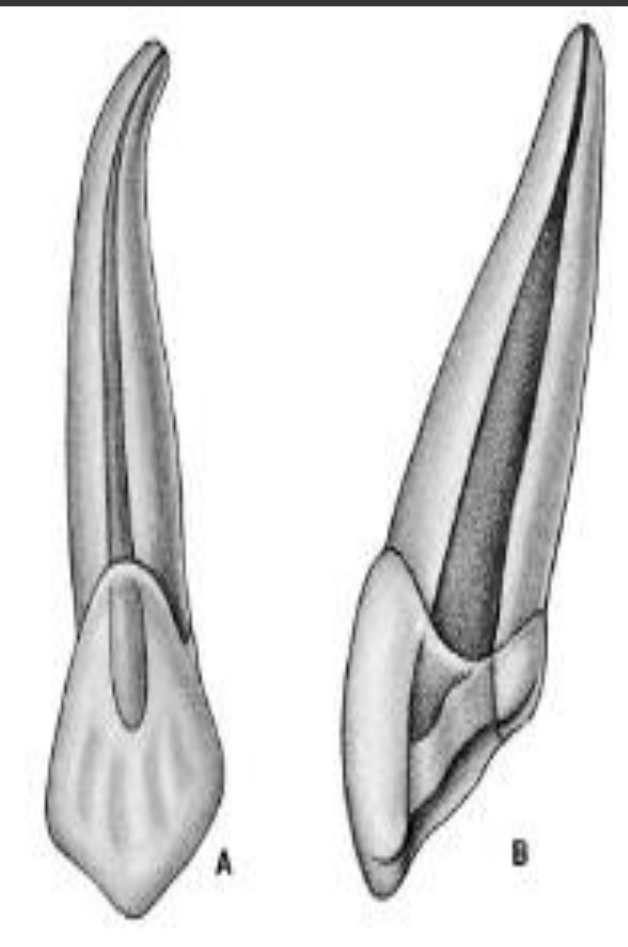




ENDODONTIC ACCESS OF MAXILLARY ANTERIOR TEETH







Access Cavity of Canine

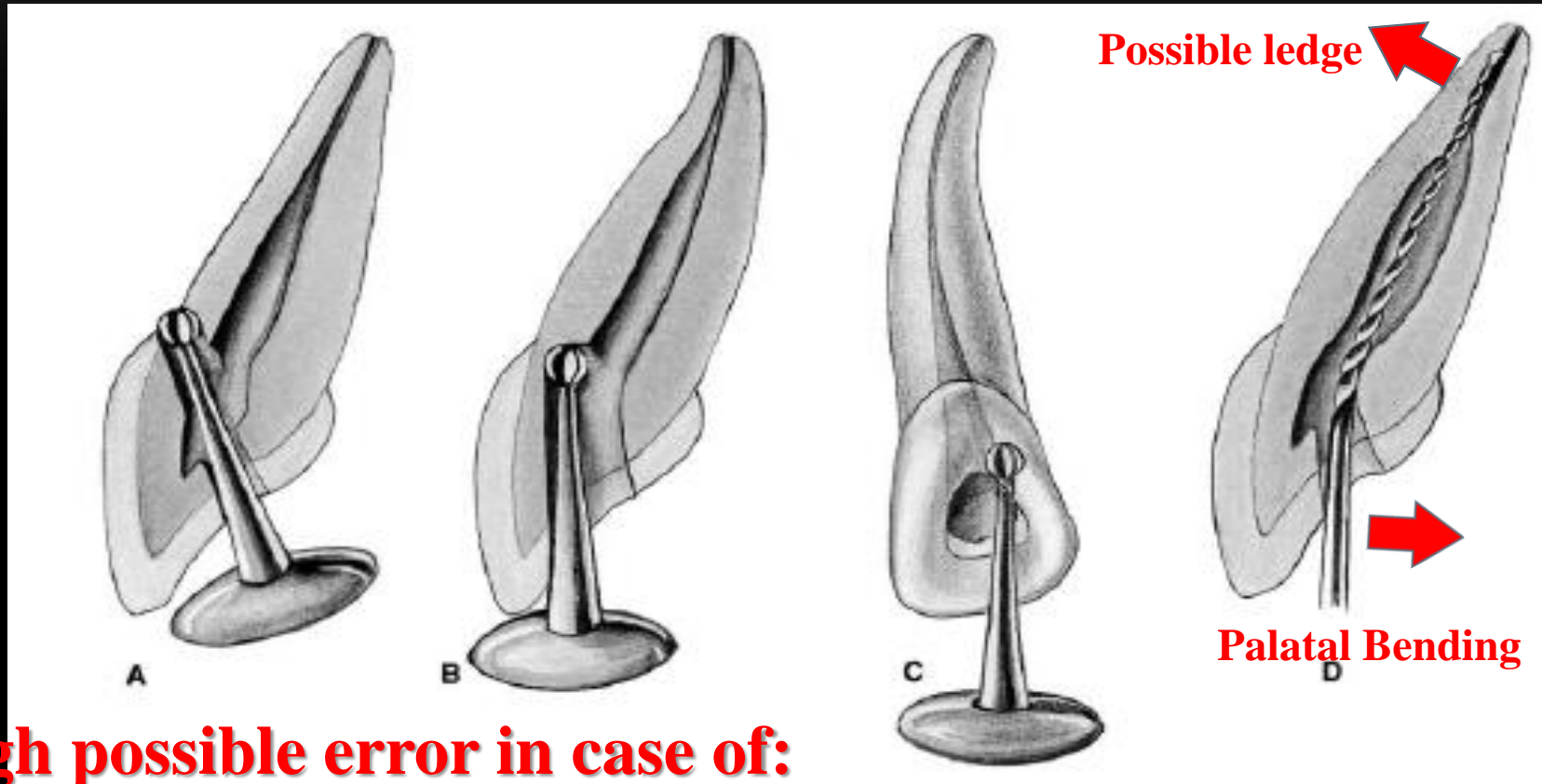
Extensive ovoid shape inciso-cervical, funnel shaped coronal preparation

The canal is usually straight but may show a distal apical curvature.

The pulp chamber is quite narrow mesiodistally, and there is one pulp horn pointed to the incisal angle.

The pulp space is much wider labiopalatally and the pulp space follows the outline.

MAXILLARY ANTERIOR TEETH ERRORS IN CAVITY PREPARATION



High possible error in case of:

1-Crowned tooth

2-Miss understanding of the tooth anatomy

3-Tiled teeth.

In crown

- 1-Missed canal
- 2-Labial perforation
- 3-Lateral perforation

In root error in access cavity may lead to:

- 4-Ledge formation
- 5-Zipping
- 6-Apical transportation
- 7-Apical perforation

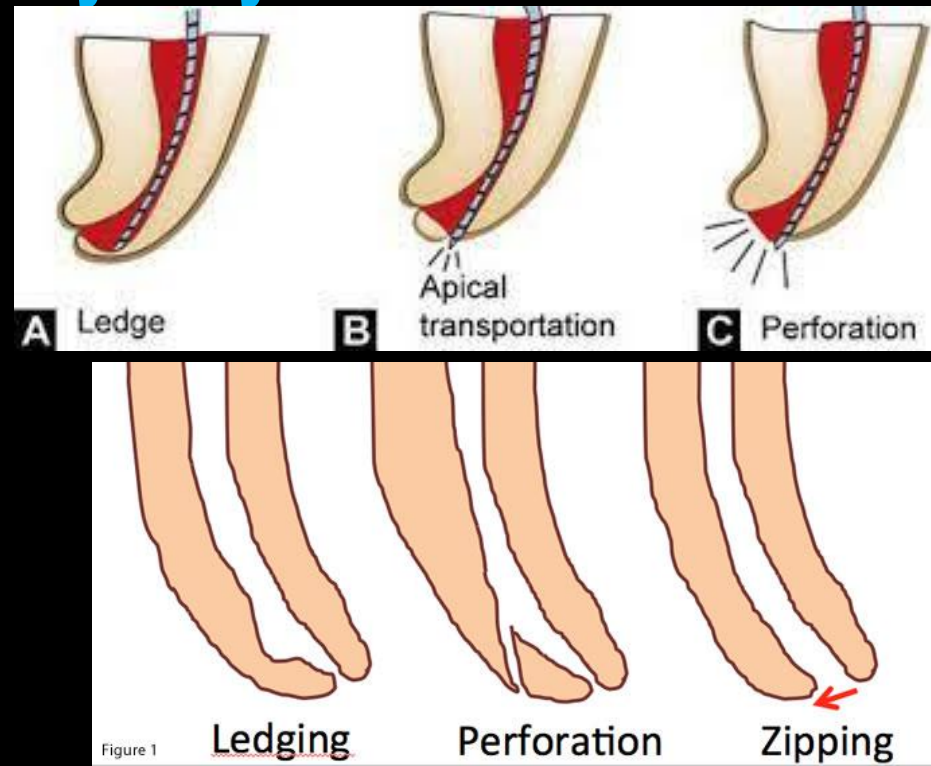
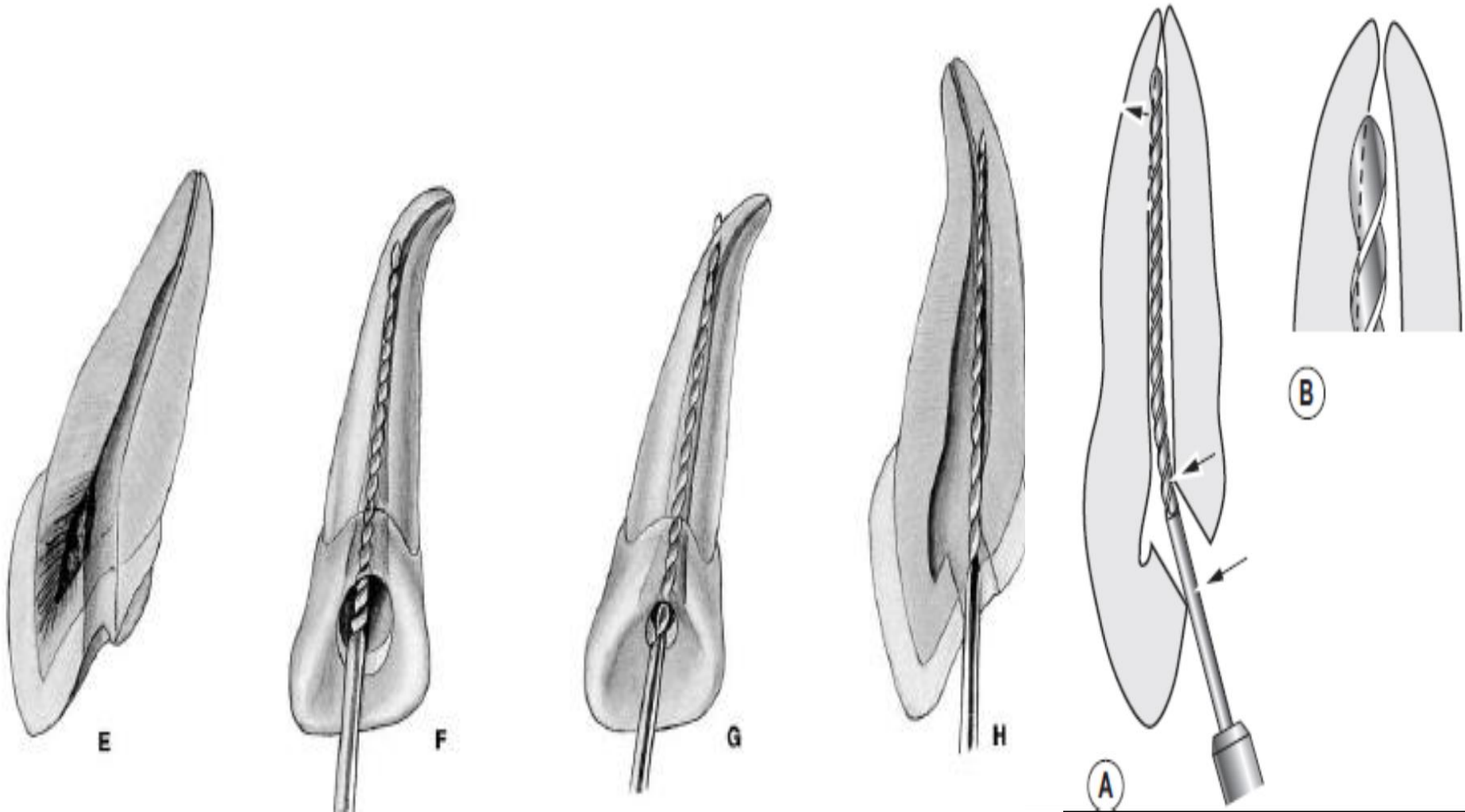


Figure 1

MAXILLARY ANTERIOR TEETH ERRORS IN CAVITY PREPARATION



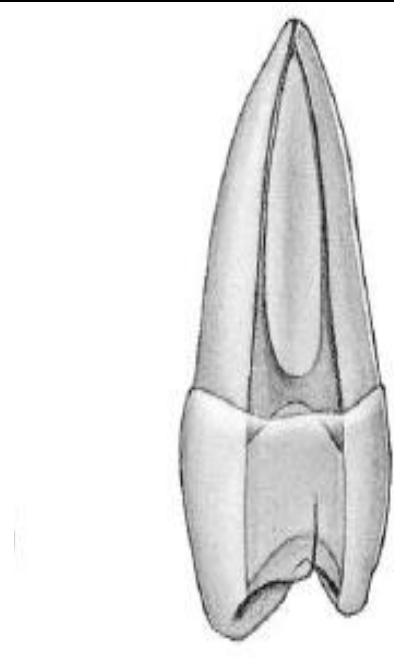
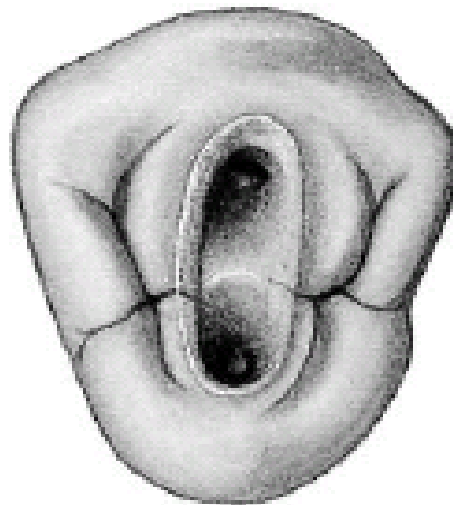
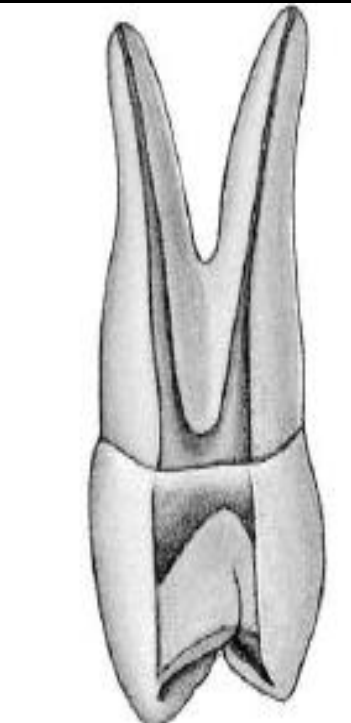
ENDODONTIC PREPARATION OF MANDIBULAR ANTERIOR TEETH



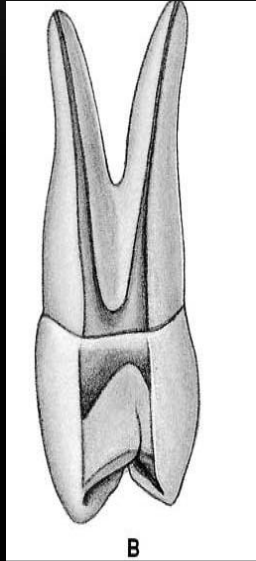


ENDODONTIC ACCESS OF MAXILLARY FIRST PREMOLAR TEETH

Young tooth

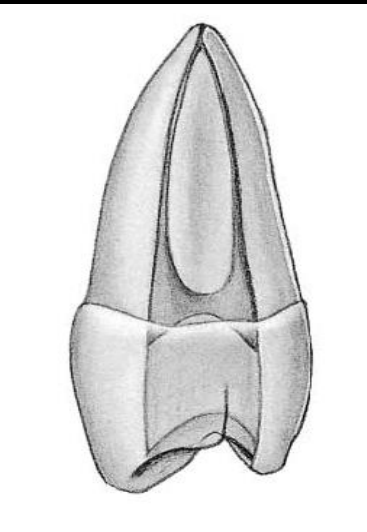


MAXILLARY FIRST PREMOLAR



Generally has 2 root with 2 canals, but in the case of 1 root has 2 canals which open in a common apical foramen.

Many types of canal configurations.



The pulp chamber is wide B-P with 2 distinct pulp horn.

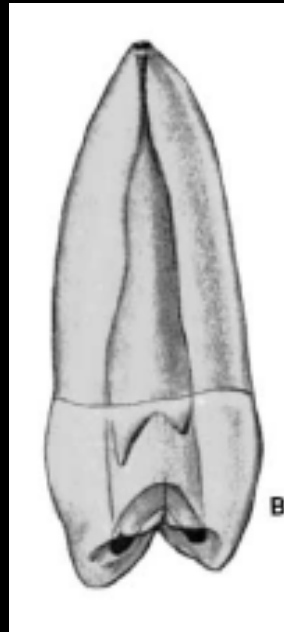
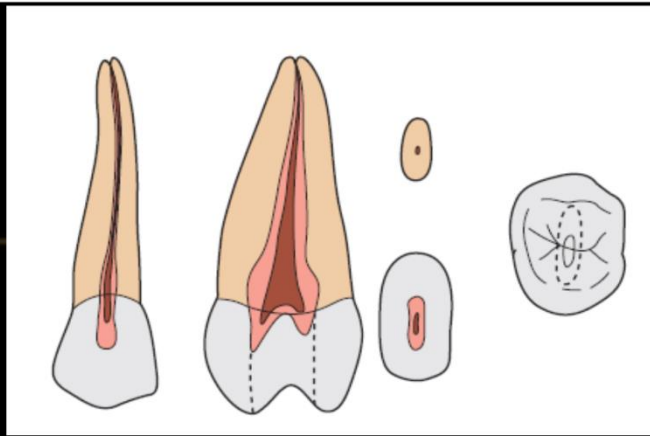
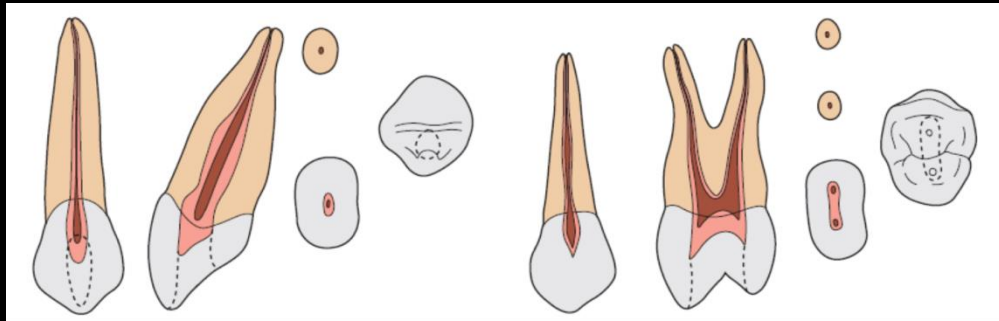
M-D, the pulp chamber is much narrower.

MAXILLARY SECOND PREMOLAR

The typical *second* premolar has 1 root and 1 canal and sometimes has 2 roots and 2 canals.

The pulp chamber is wider B-P and narrower M-D and has 2 well define pulp horns.

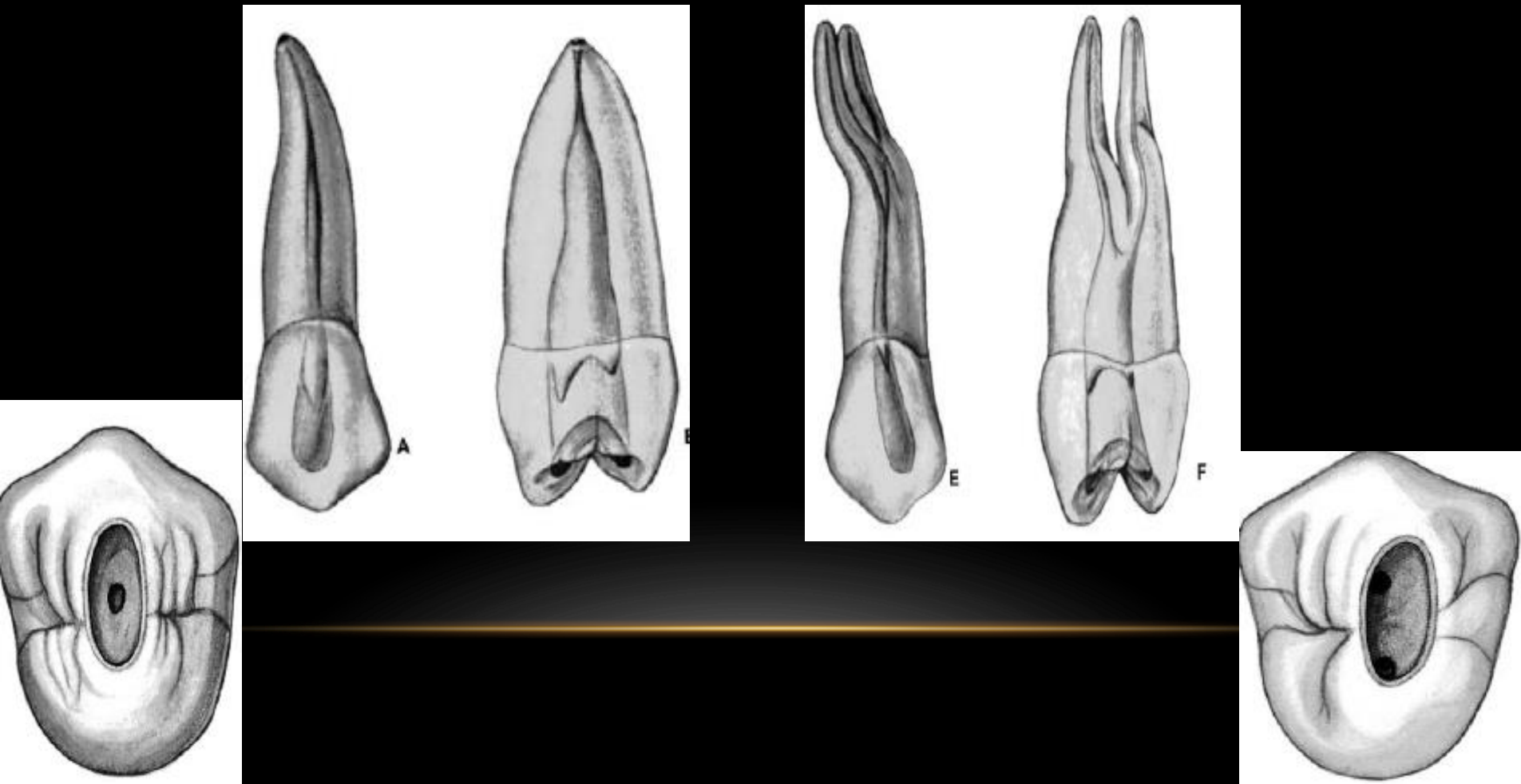
The canal orifice is directly in the centre of the tooth.



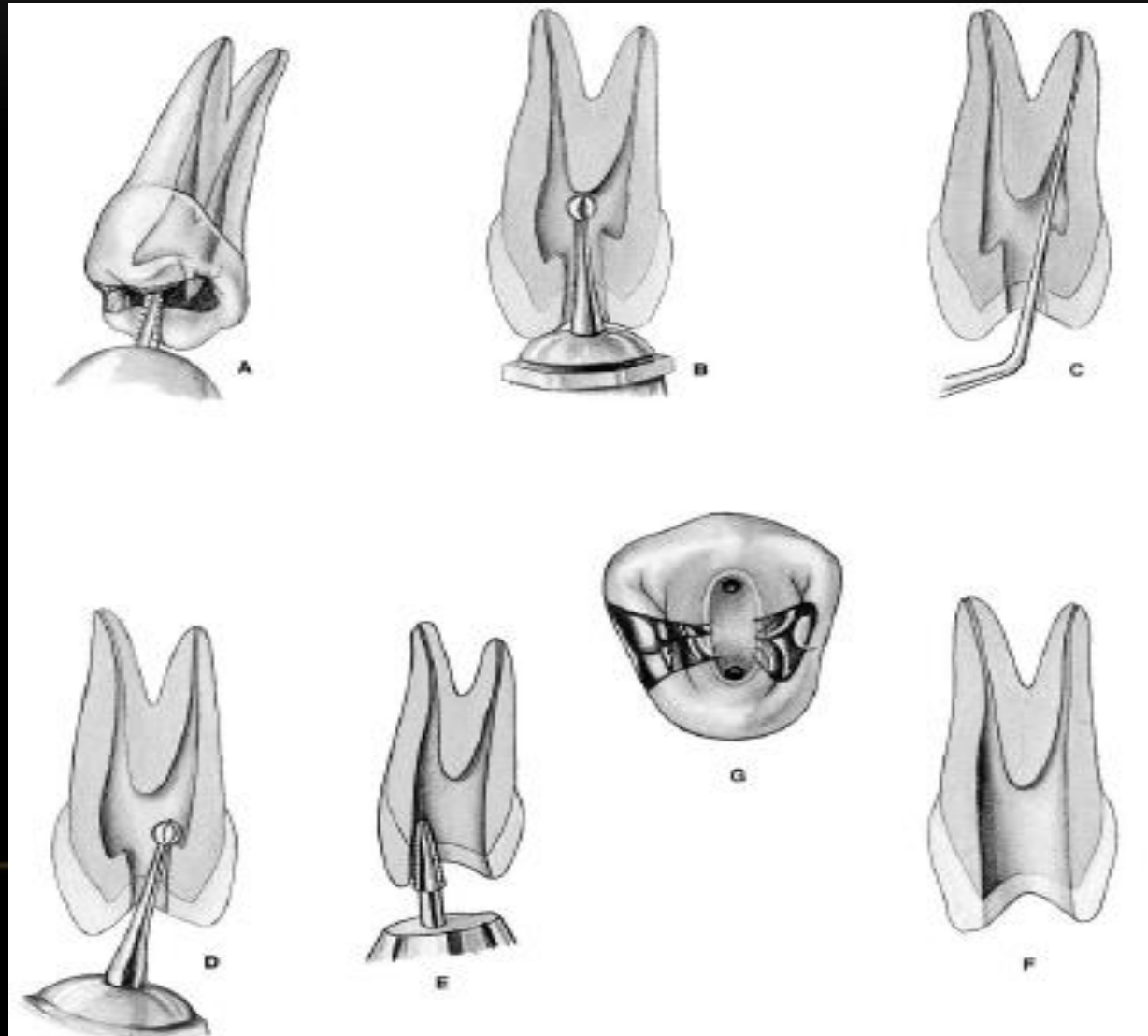
ENDODONTIC ACCESS OF MAXILLARY SECOND PREMOLAR TEETH

Young tooth

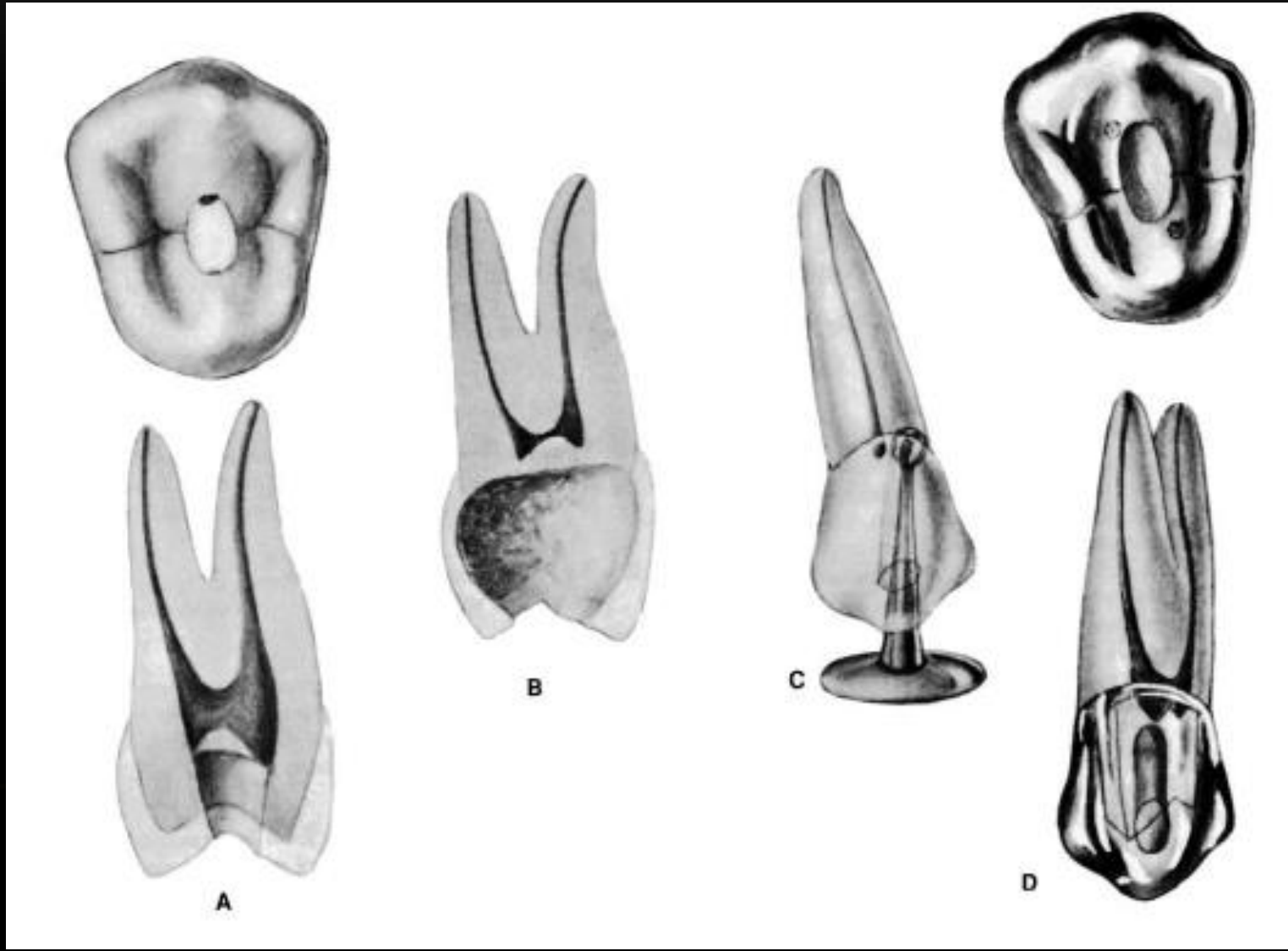
Adult tooth



ENDODONTIC ACCESS PREPARATION OF MAXILLARY PREMOLAR TEETH



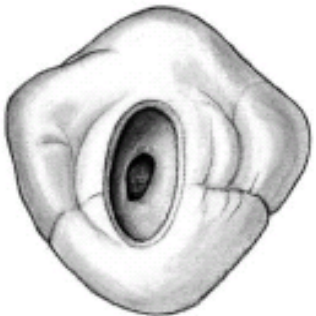
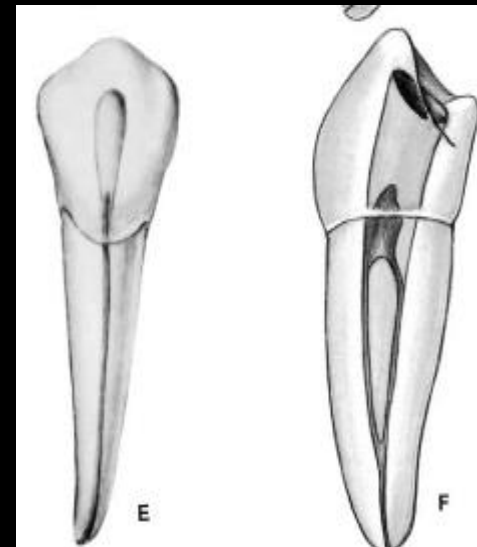
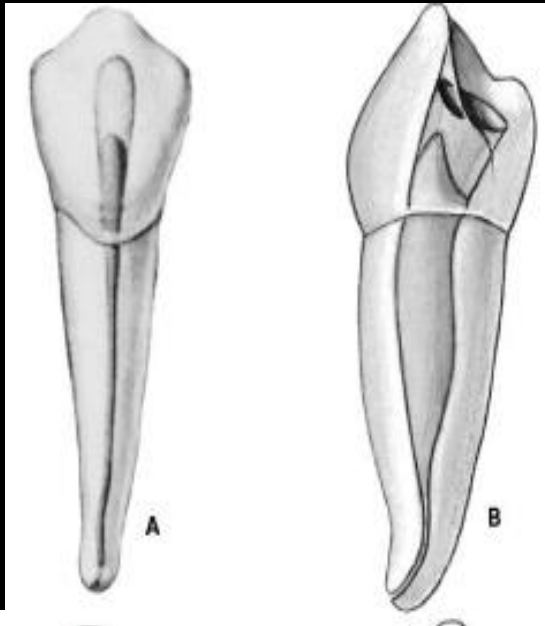
MAXILLARY PREMOLAR TEETH ERRORS IN CAVITY PREPARATION



ENDODONTIC ACCESS PREPARATION OF MANDIBULAR PREMOLAR TEETH

Young tooth

Adult tooth

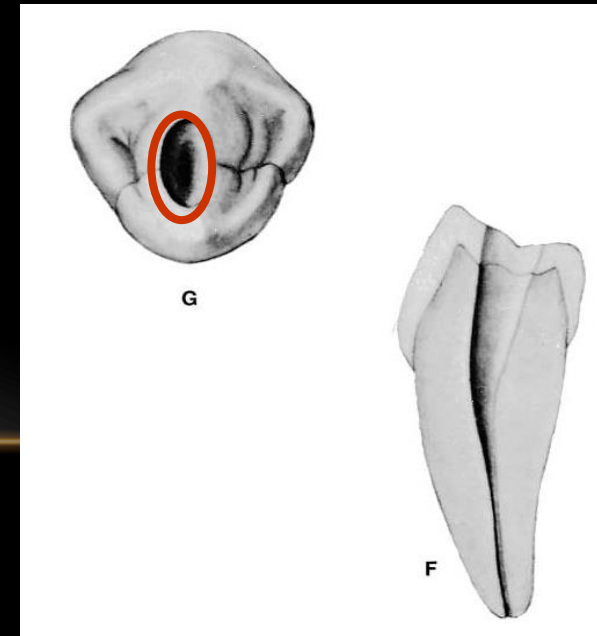


ACCESS CAVITY

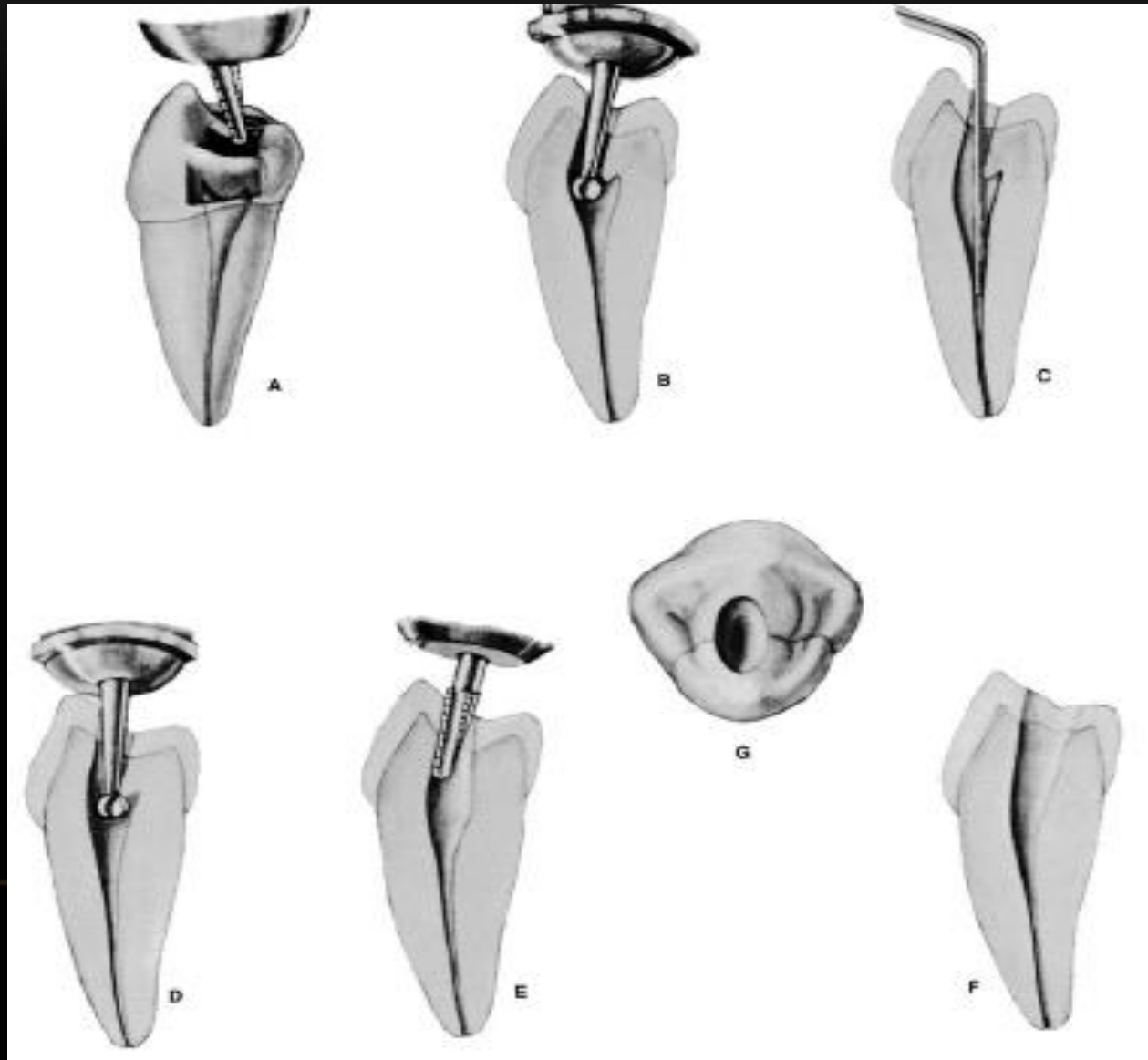
Because of:

- 1-The inclination of the crowns of mandibular teeth.
- 2- The smaller lingual cusp, the access opening should be placed buccal to the central fissure.

The preparation is made **oval**, corresponding to the shape of the root and canal.



ENDODONTIC PREPARATION OF MANDIBULAR PREMOLAR TEETH



MANDIBULAR PREMOLAR TEETH ERRORS IN CAVITY PREPARATION

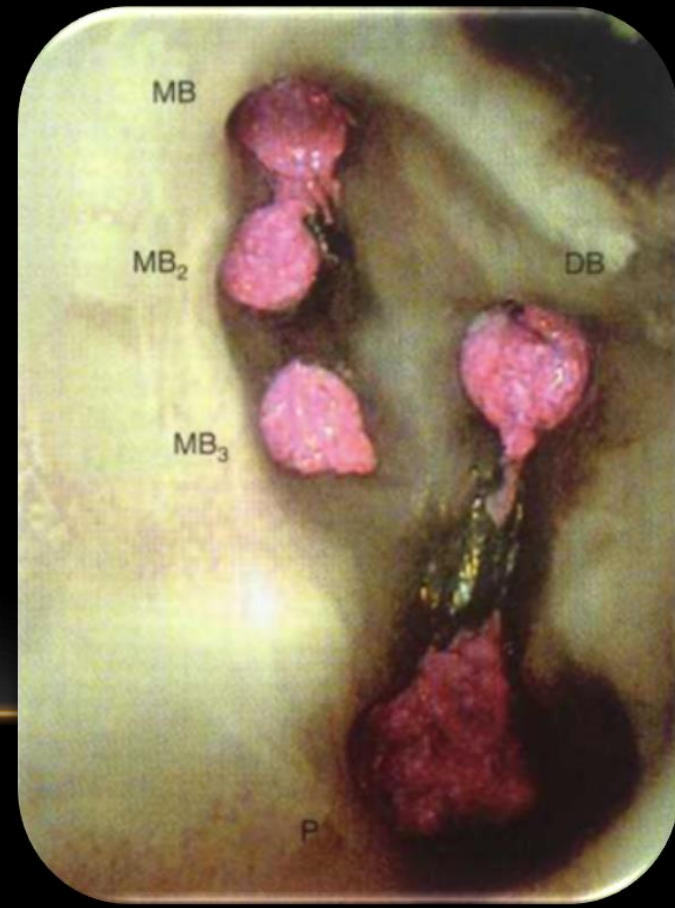
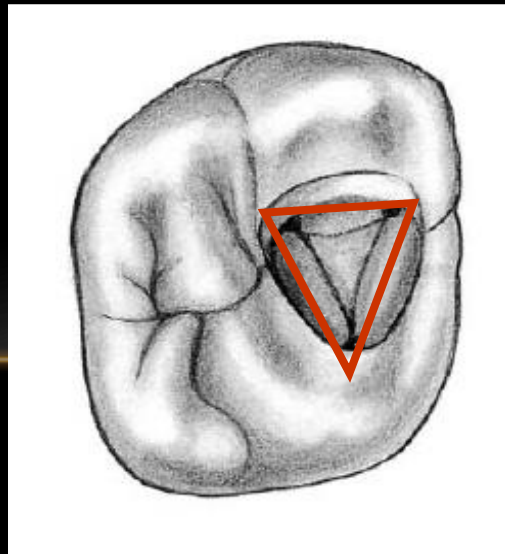
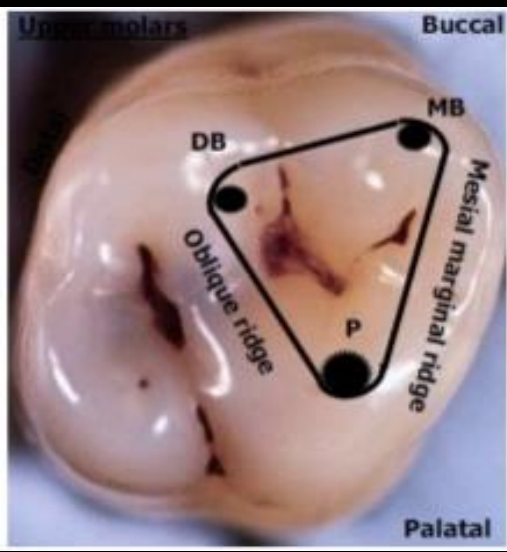


ENDODONTIC PREPARATION OF MAXILLARY AND MANDIBULAR MOLAR TEETH



MAXILLARY FIRST MOLARS

1. Generally three rooted with 3 canals. Additional canals MB1,2 & 3 is located in the MB root.
2. Large pulp chamber, **triangular in shape**, with the base toward the buccal and the apex toward the lingual surface.
3. Slightly curved buccal roots.
4. DP curvature of the MB root.
5. Apical-buccal curvature of the palatal root (55%)



It is very important to preserve the developmental lines that connect with the canal orifices. It's dark lines in the floor of the pulp chamber of teeth.



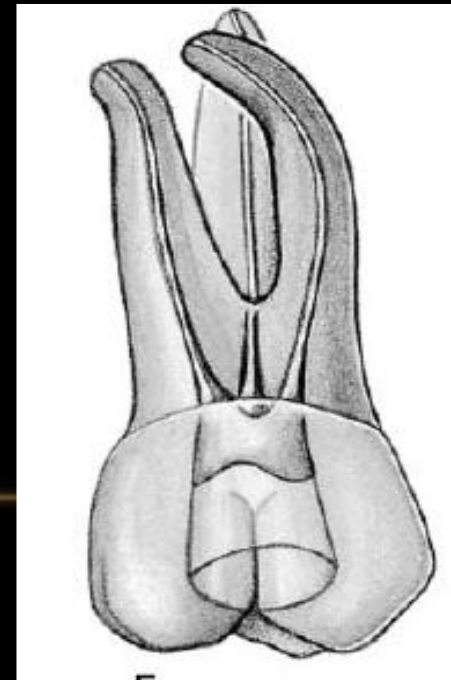
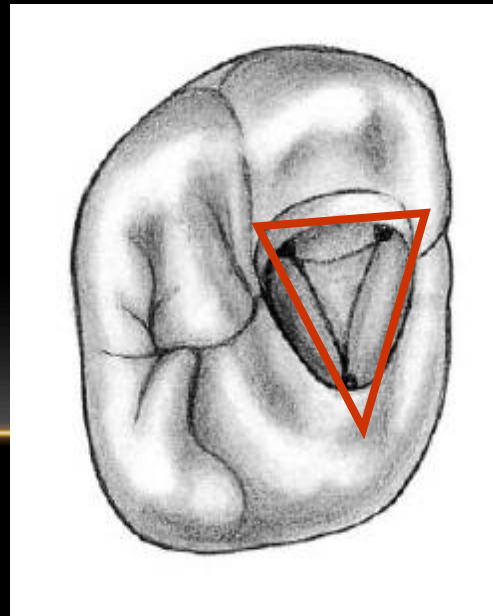
Maxillary First Molars

It has 3 or 4 pulp horn, the MB is the longest.

The floor of the pulp chamber is normally just apical to the cervix and is rounded and convex to the occlusal.

The **MB** canal opening is closer to the buccal wall than is the DB orifice, mostly 2 - 3 canal **MB1 + MB2 or MB3**.

The DB canal is closer to the middle of the tooth than to the distal wall.



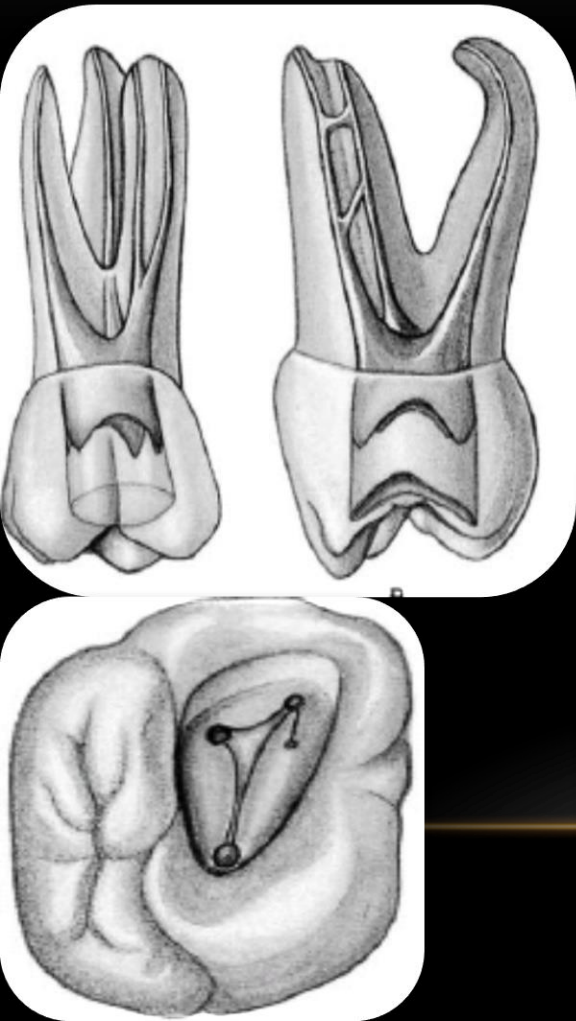
Magnification is very important to locate the canals.

**OPERATING
MICROSCOPE IS USED
TO LOCATE THE CANAL
ORIFICES**

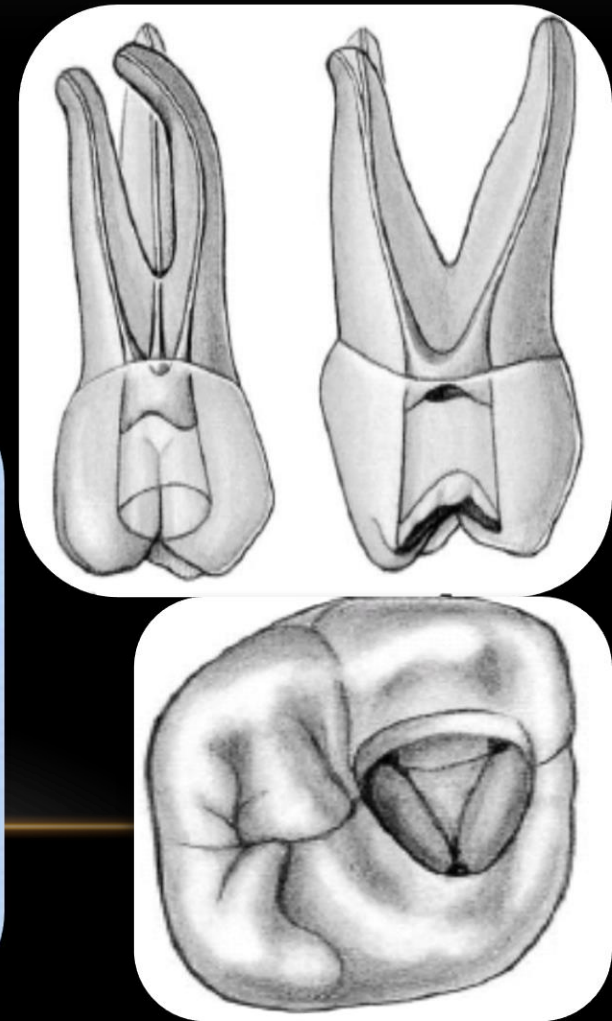


ENDODONTIC ACCESS PREPARATION OF MAXILLARY FIRST MOLAR TEETH

Young tooth



Adult tooth

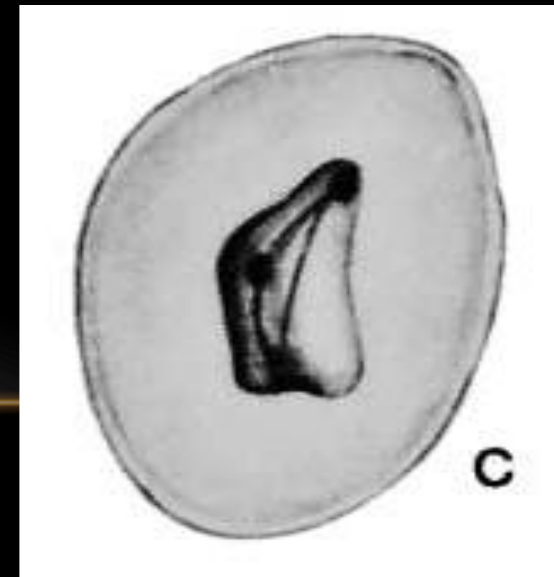
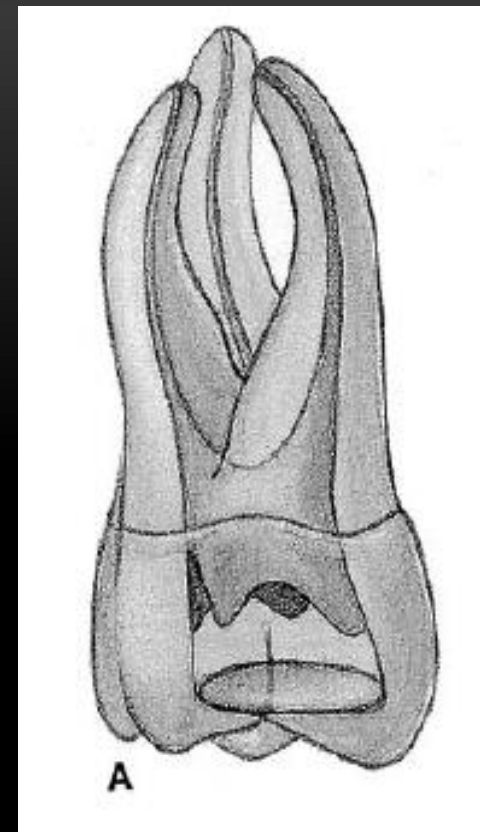


**Always seek for
accessory canal
in any tooth and
every were**

MAXILLARY SECOND MOLAR

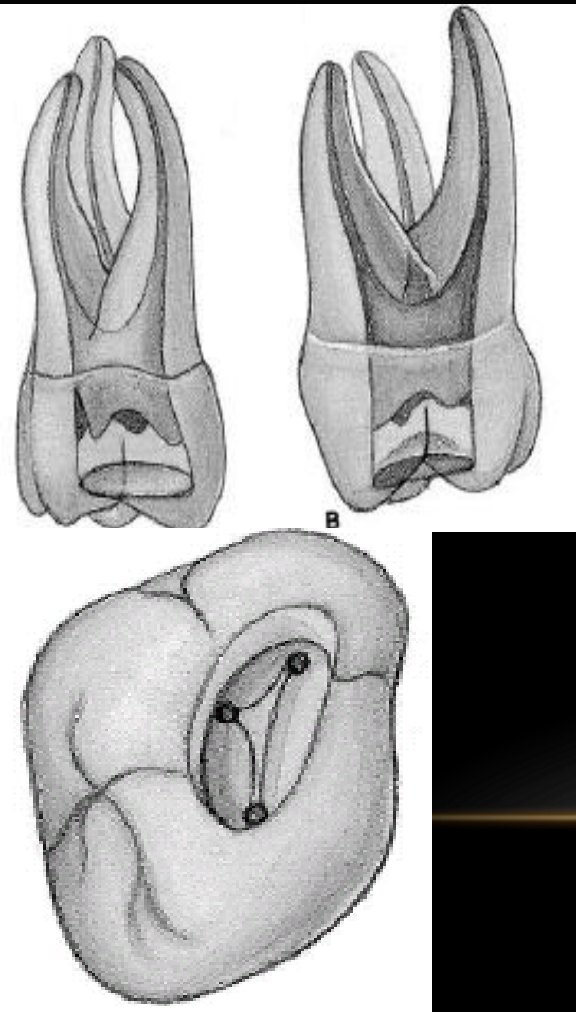
It is similar of the first molar:

- 1- Large pulp chamber.
- 2- Mesio Buccal, distobuccal, and palatal roots, each with one canal.
- 3- Gradual curvature of all three canals.
- 4- Flattened” triangular outline form.
- 5- The DB canal orifice is nearer the centre of the cavity floor.
- 6- Some time 2 canals

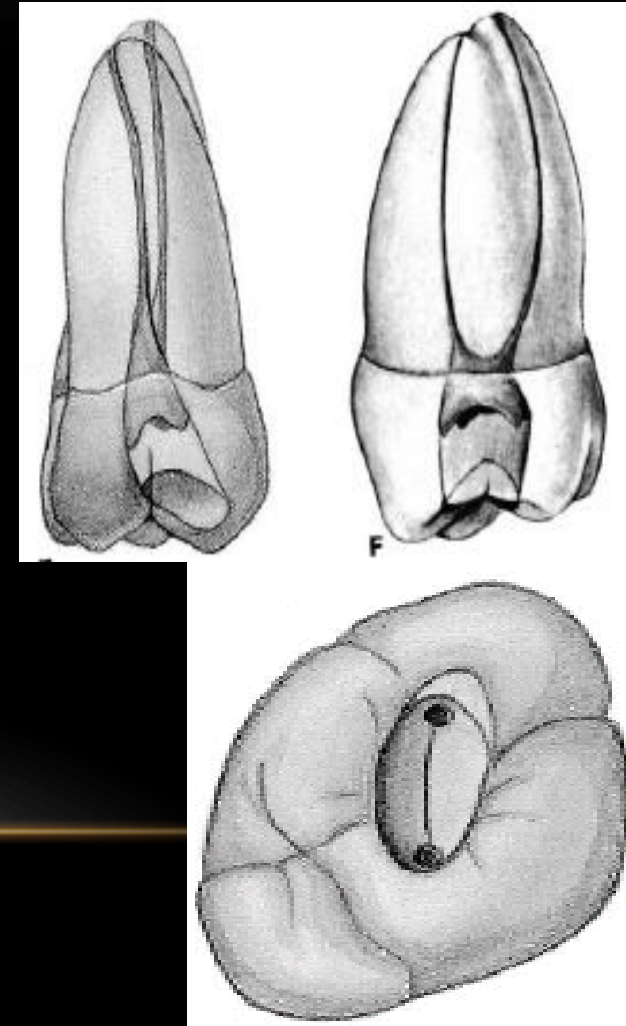


ENDODONTIC ACCESS PREPARATION OF MAXILLARY SECOND MOLAR TEETH

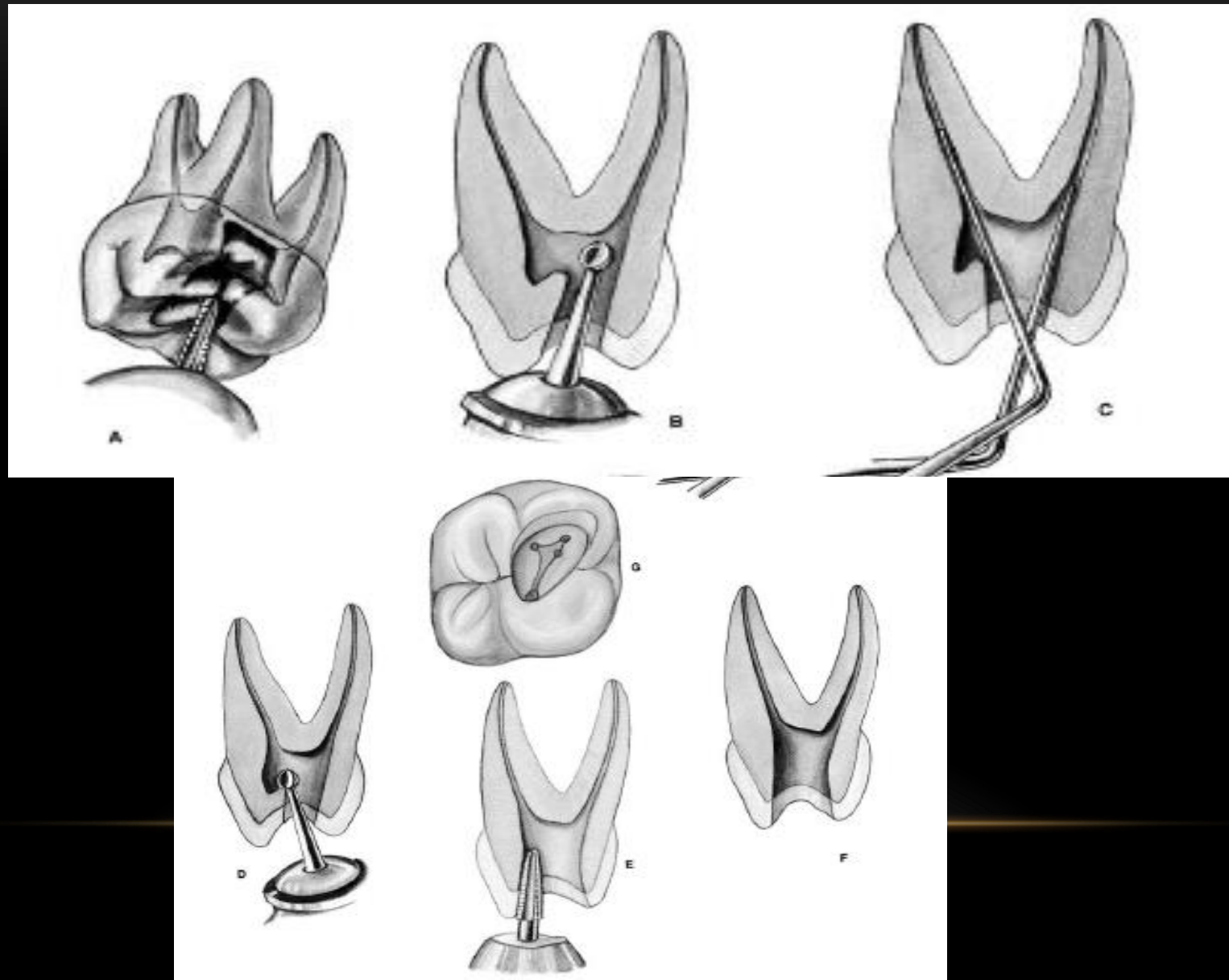
Young tooth



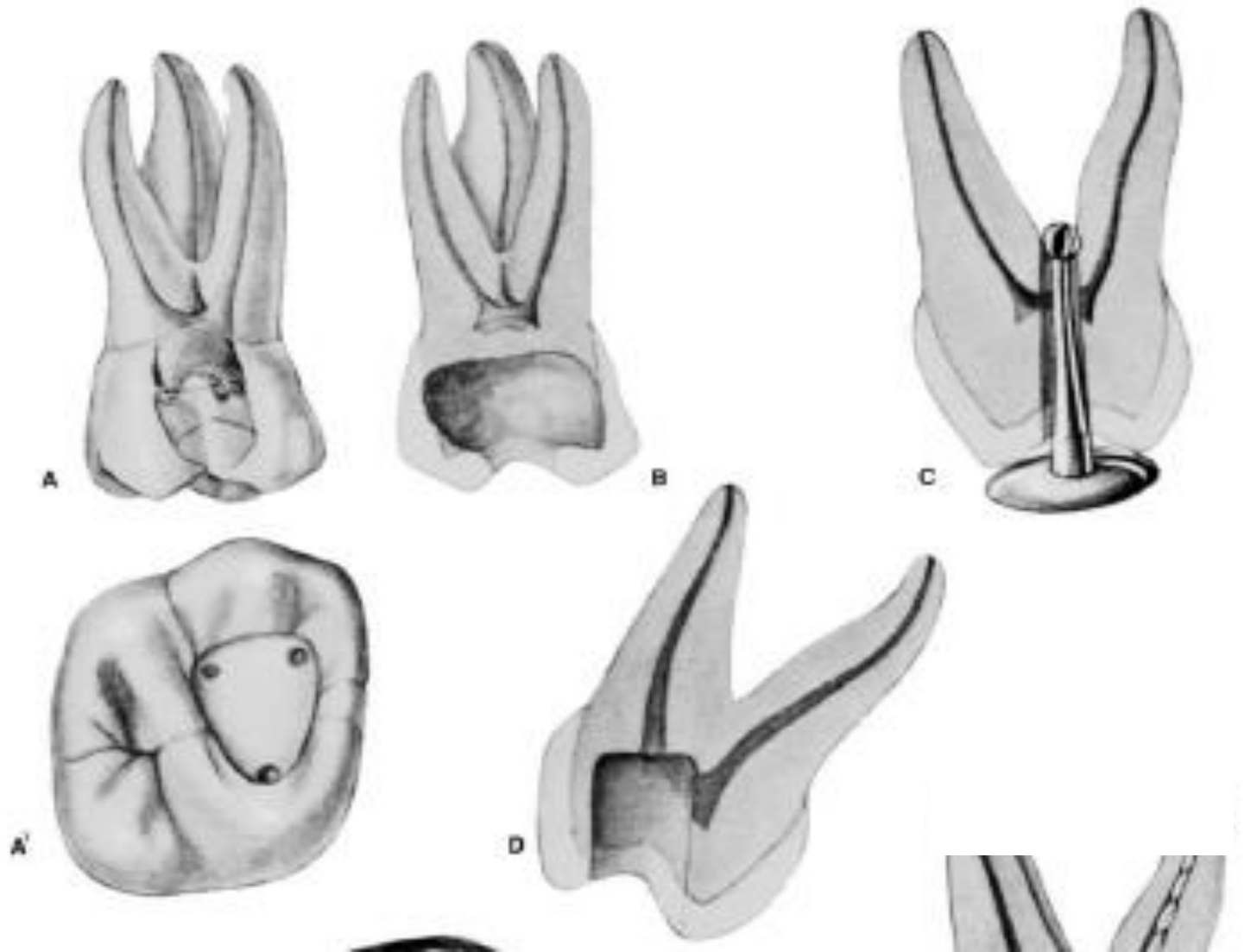
Adult tooth



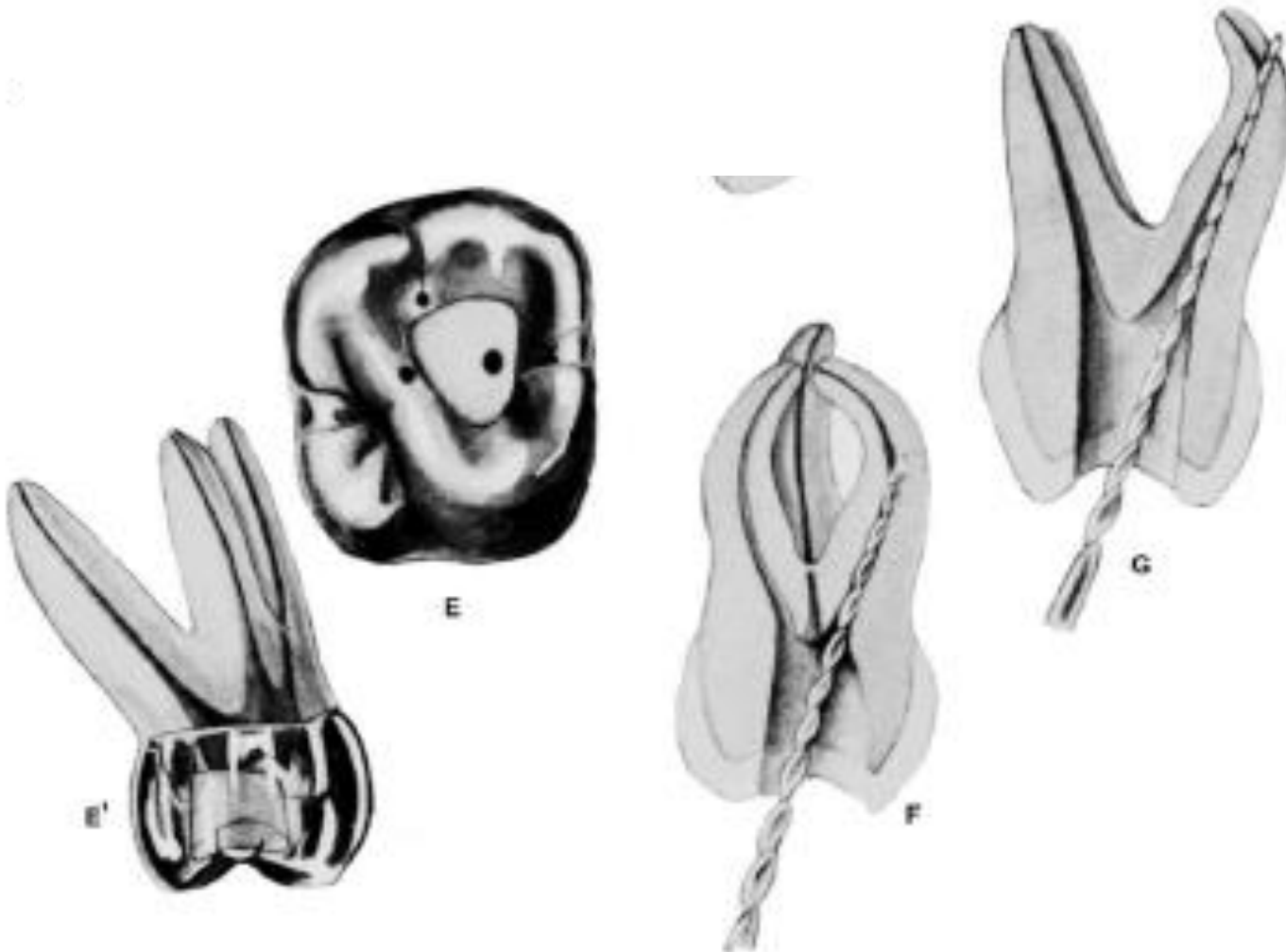
ENDODONTIC PREPARATION OF MAXILLARY MOLAR TEETH



MAXILLARY MOLAR TEETH ERRORS IN CAVITY PREPARATION



MAXILLARY MOLAR TEETH ERRORS IN CAVITY PREPARATION

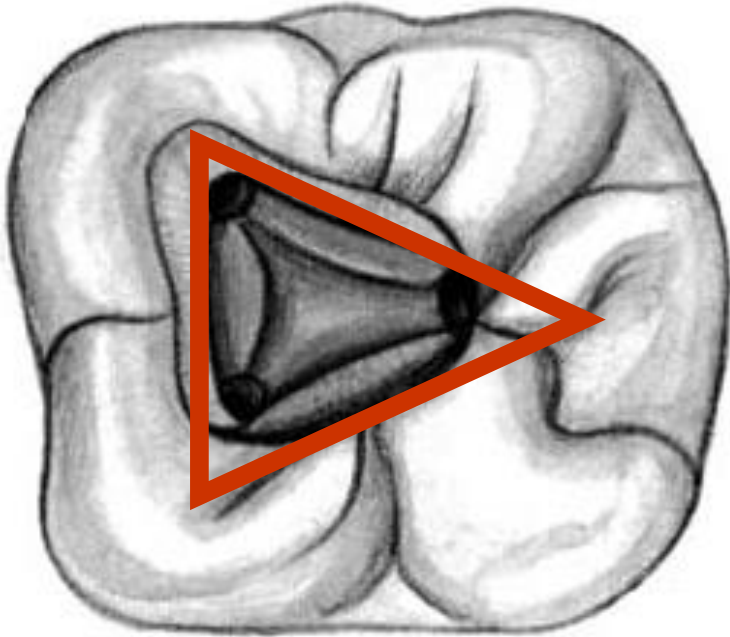


MANDIBULAR FIRST MOLAR

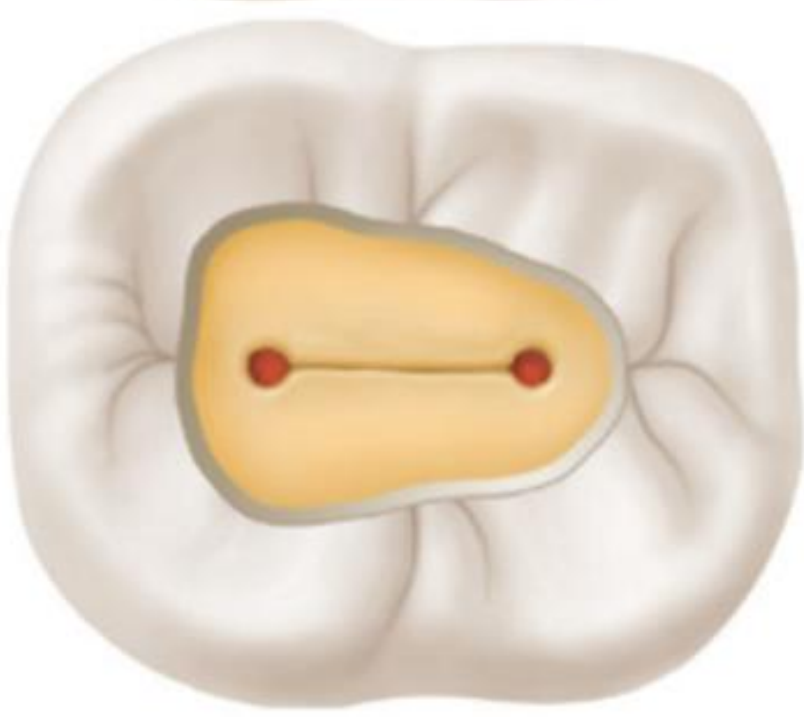
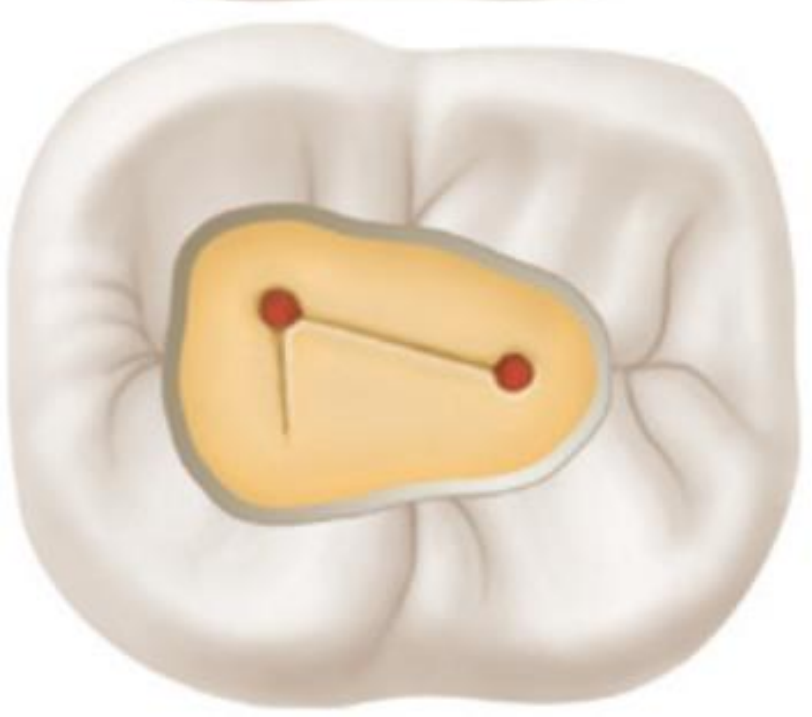
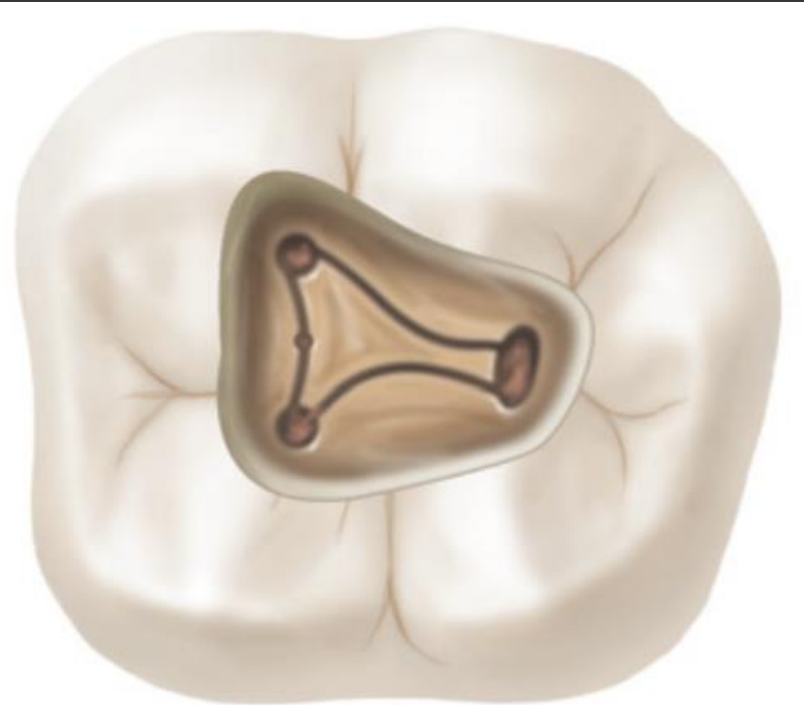
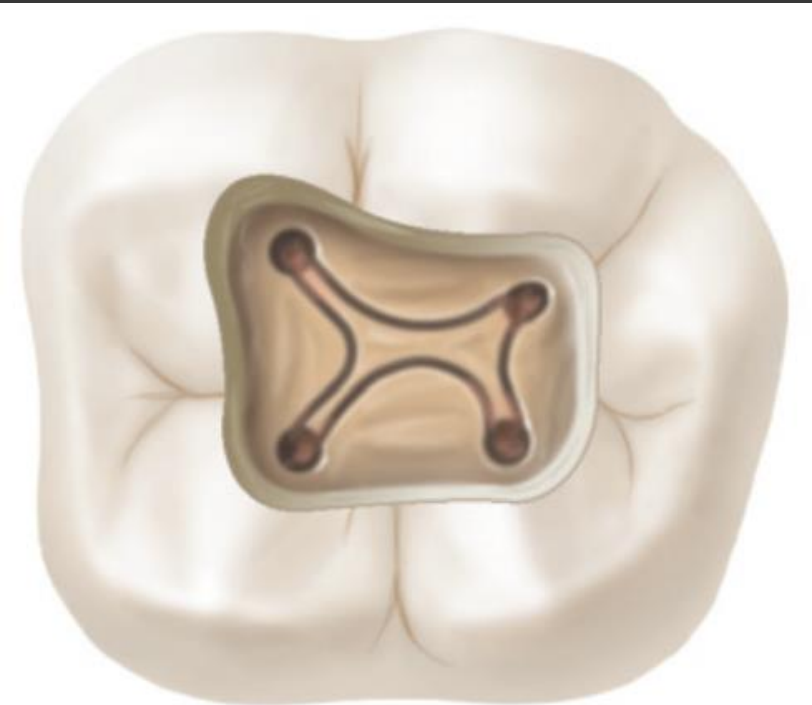
1. Usually has 2 roots one mesial and one distal.
2. The Distal root is smaller and vertical.
3. Distal curvature of the mesial root (84% of the time) which has two canals.
4. The distal canal is larger and more oval.
5. The MB is the most difficult canal to instrument because its tortuous path and narrow.



The cavity is primarily within the mesial half of the tooth but is extensive enough to allow positioning of instruments and filling materials.



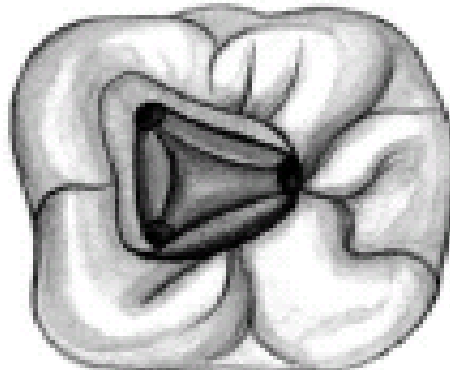
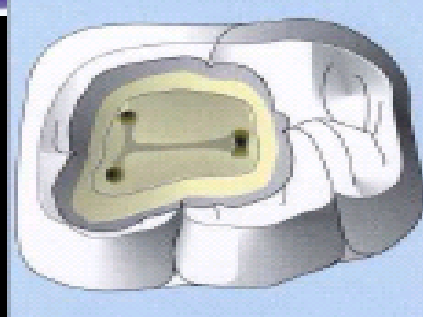
Triangular outline form in case of 3 canals, with the base toward mesial and the apex toward the distal surface. square to rectangular in case of 4 canals reflects the anatomy of the pulp chamber,



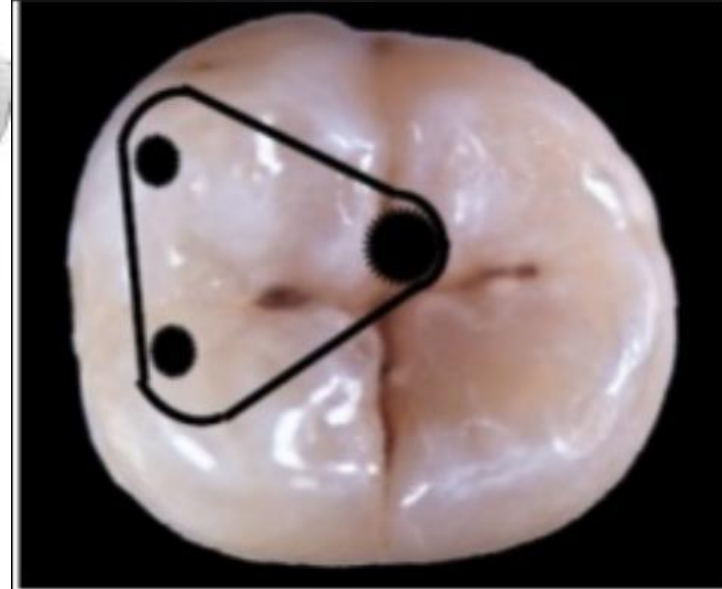
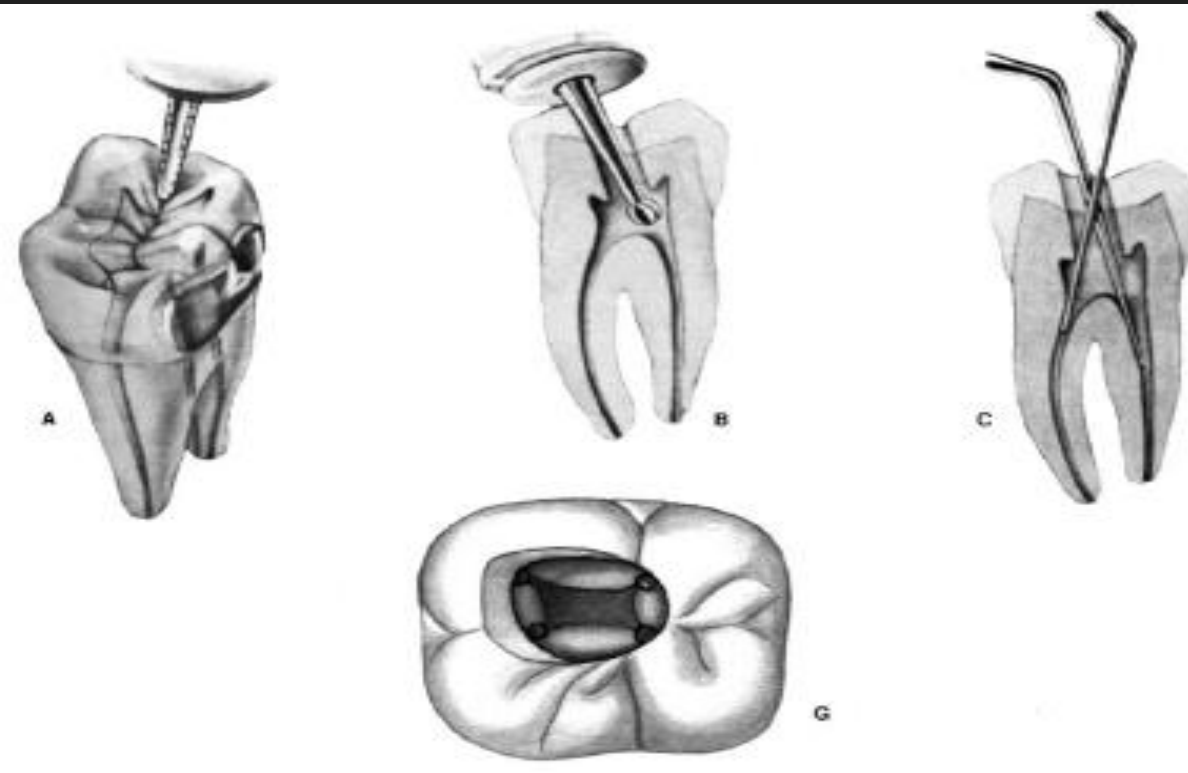
ENDODONTIC PREPARATION OF MANDIBULAR MOLAR TEETH

Young tooth

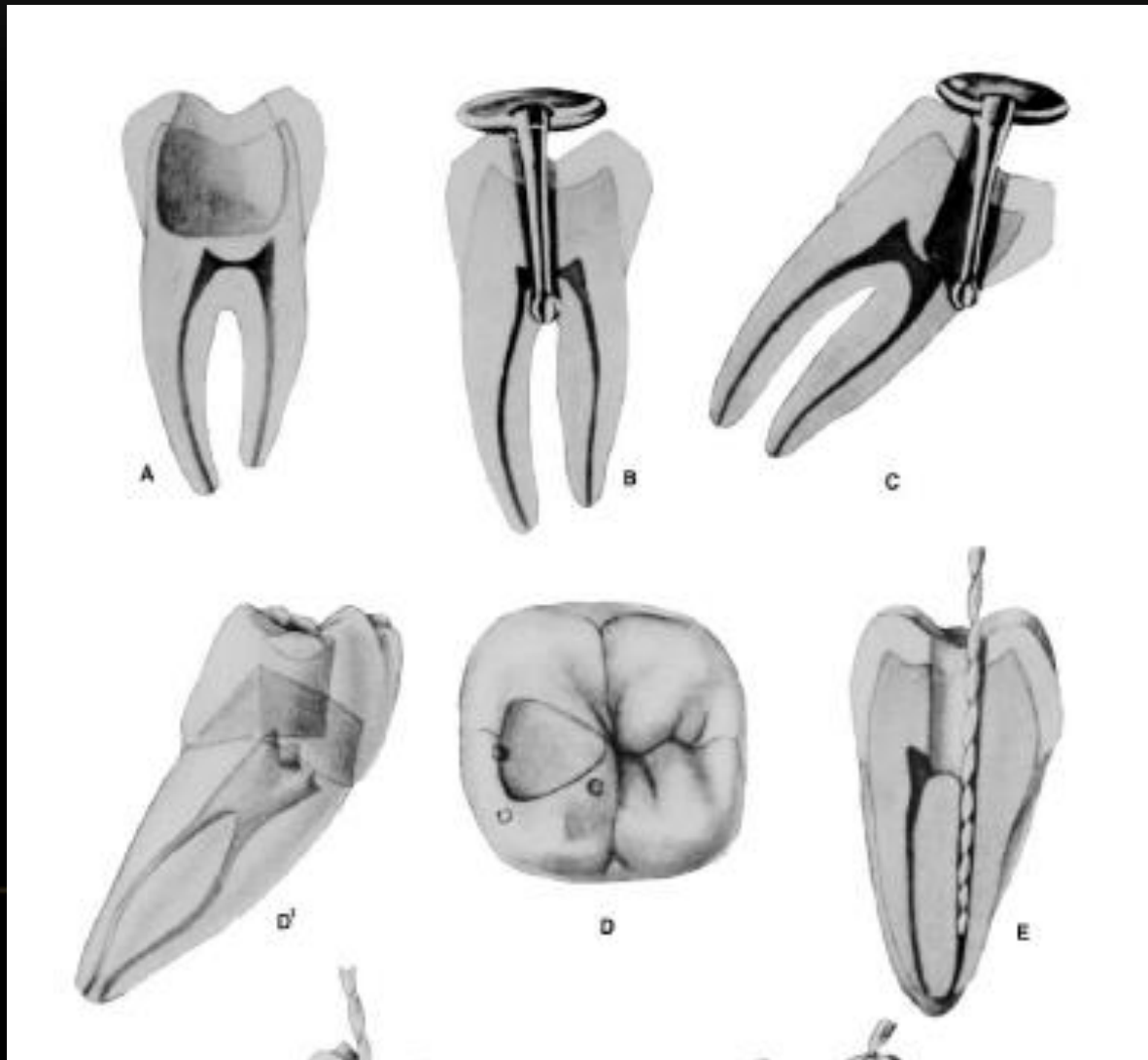
Adult tooth



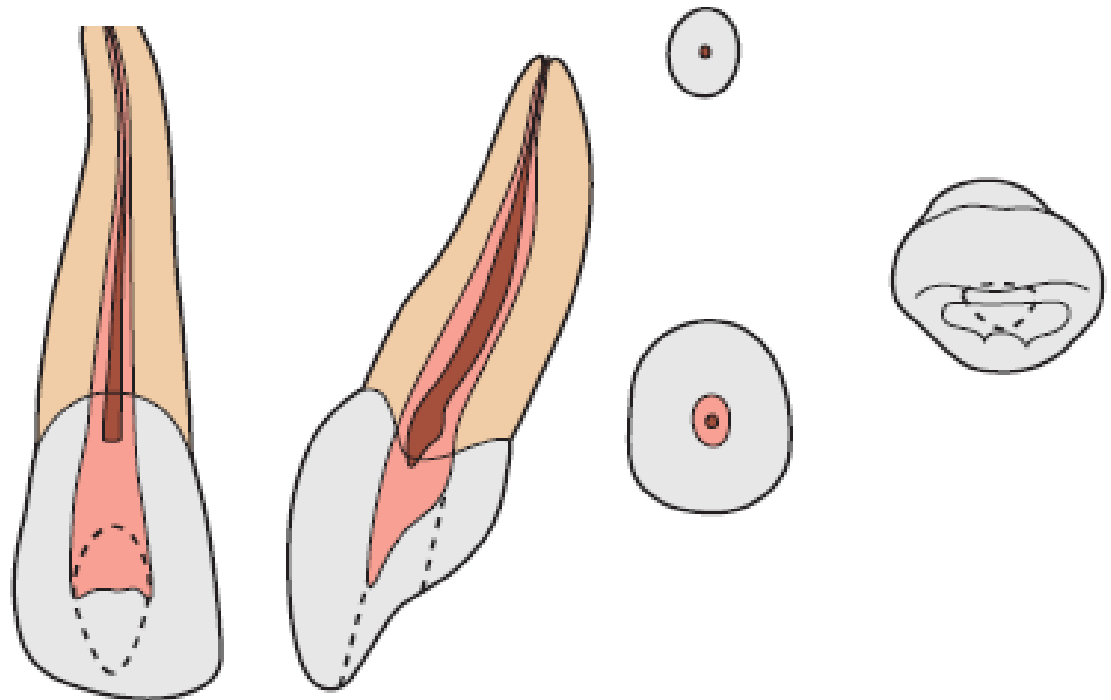
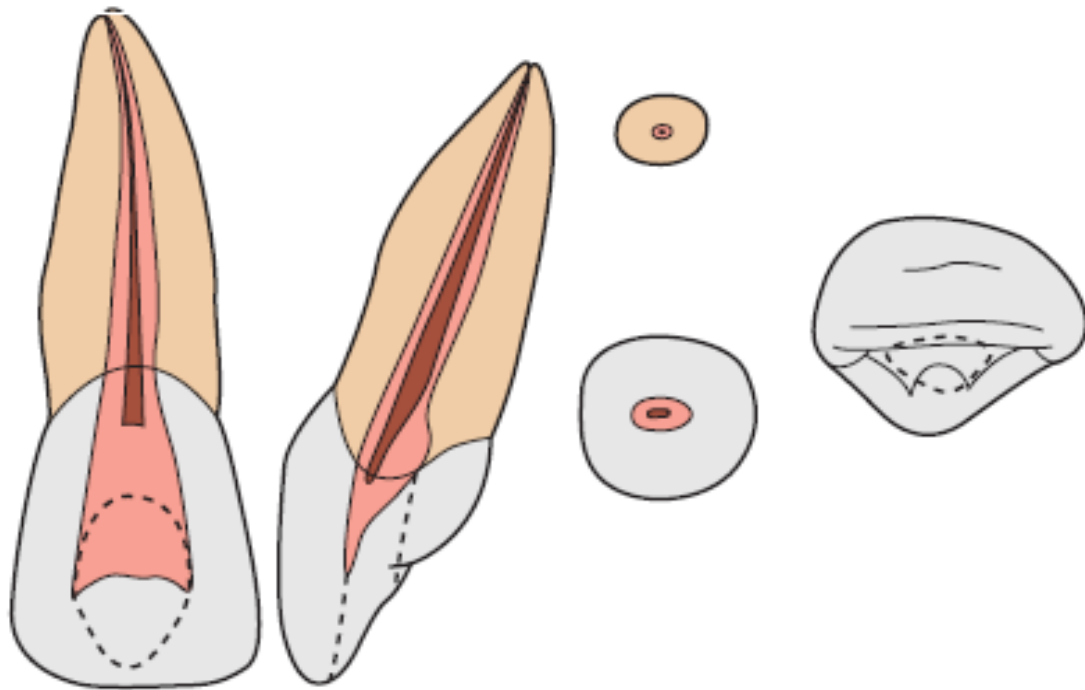
ENDODONTIC PREPARATION OF MANDIBULAR MOLAR TEETH

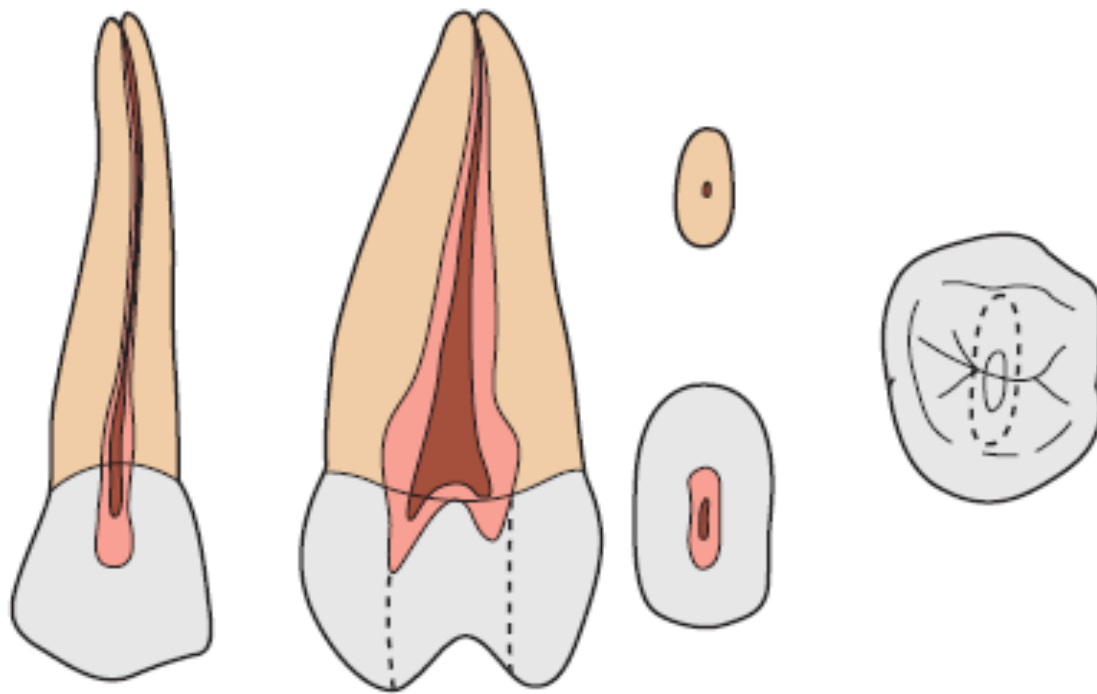
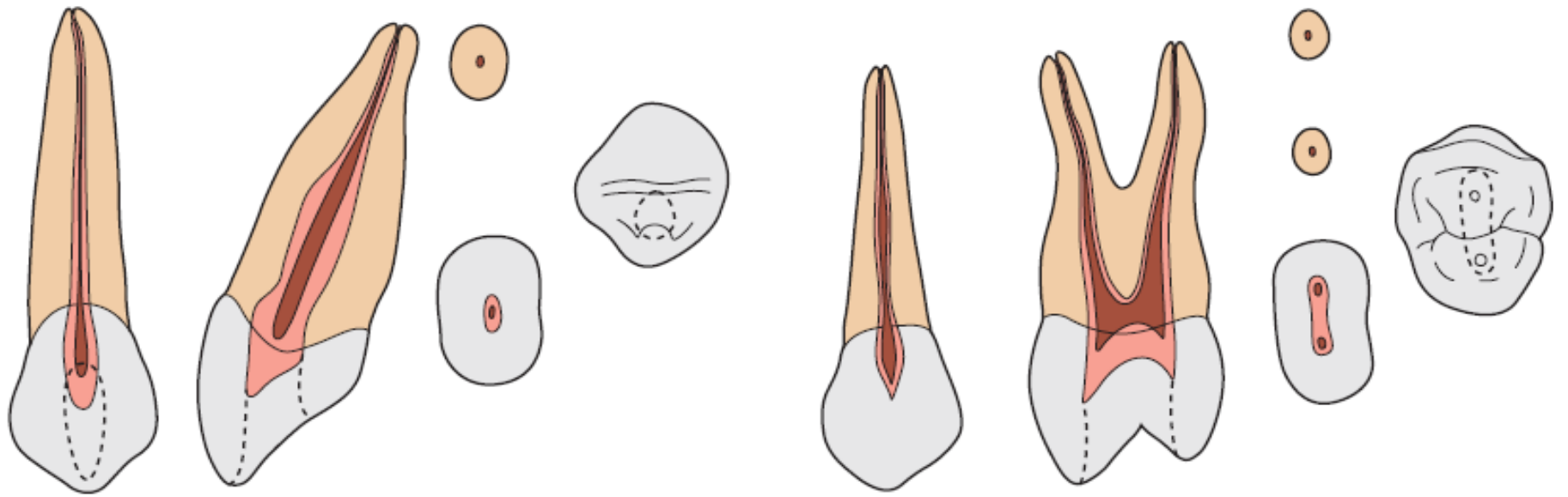


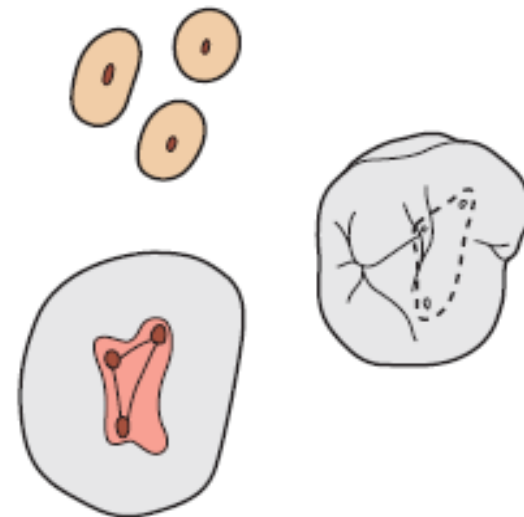
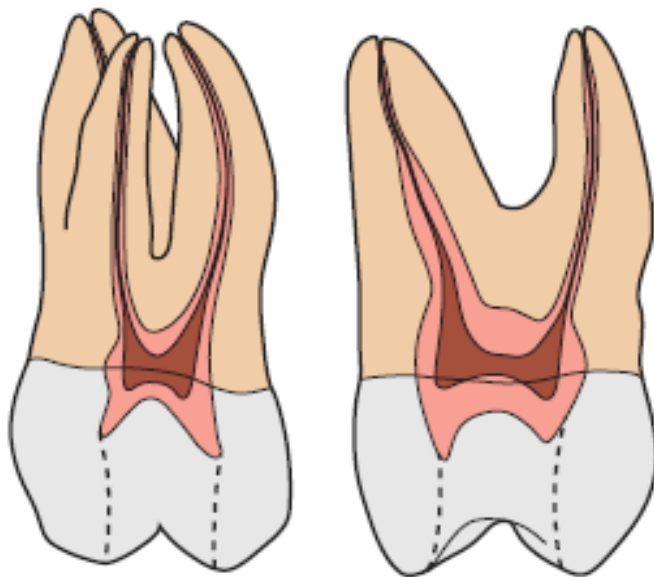
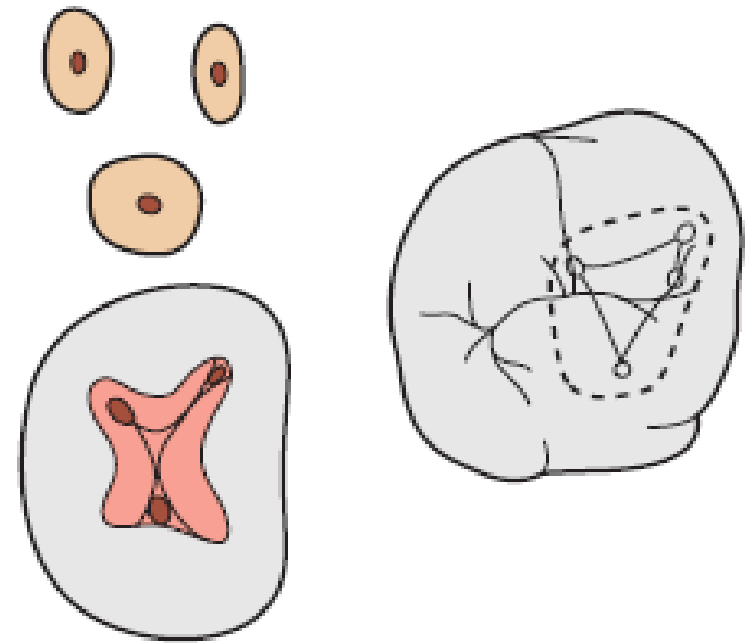
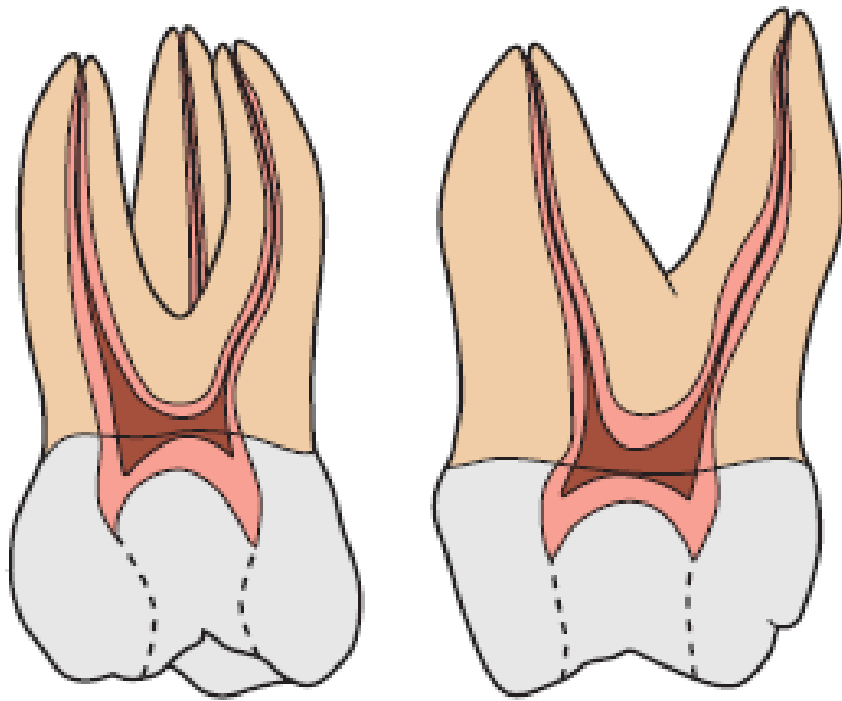
MANDIBULAR MOLAR TEETH ERRORS IN CAVITY PREPARATION

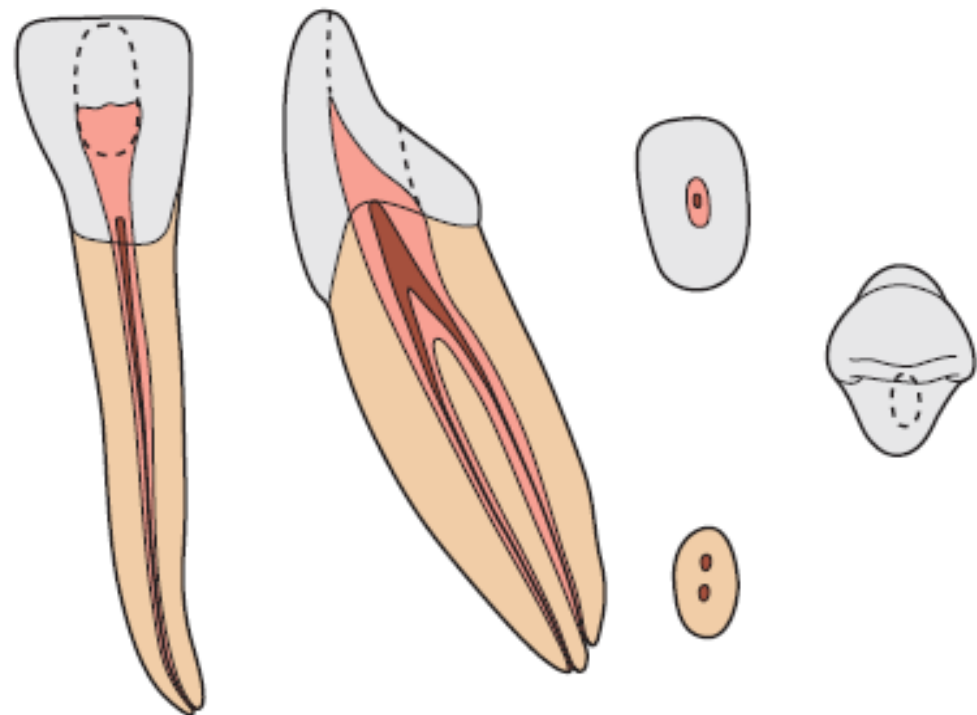
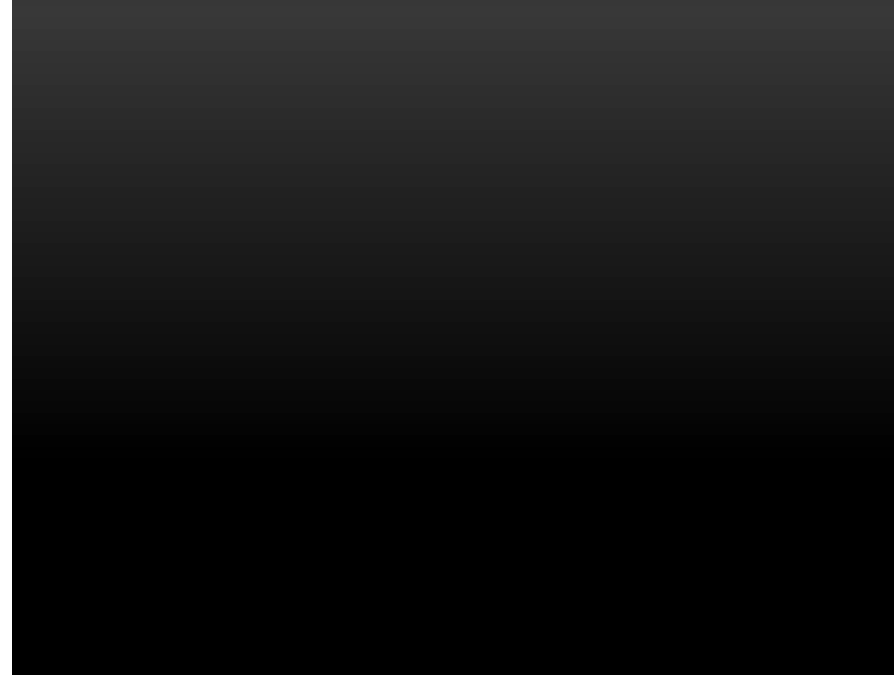
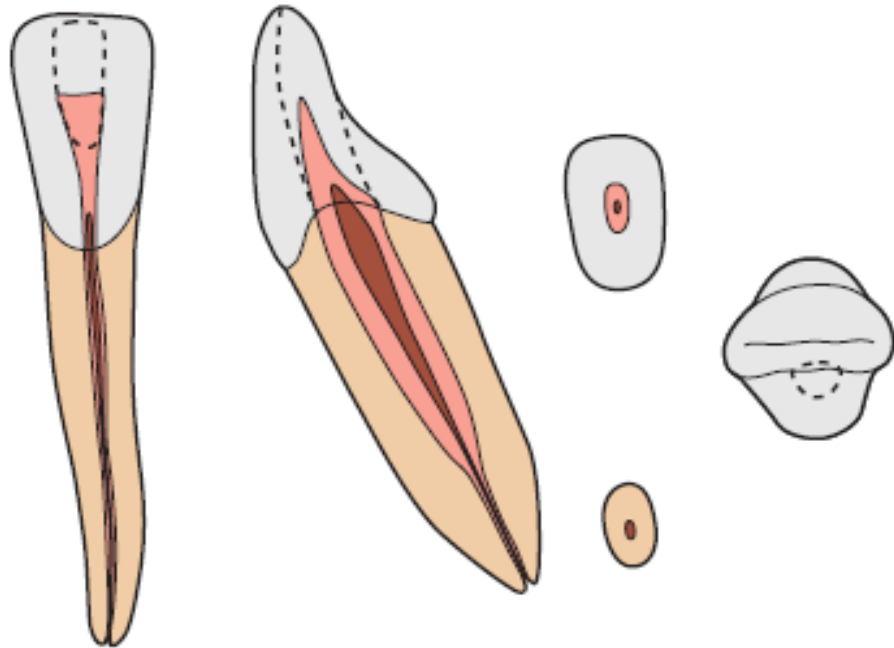


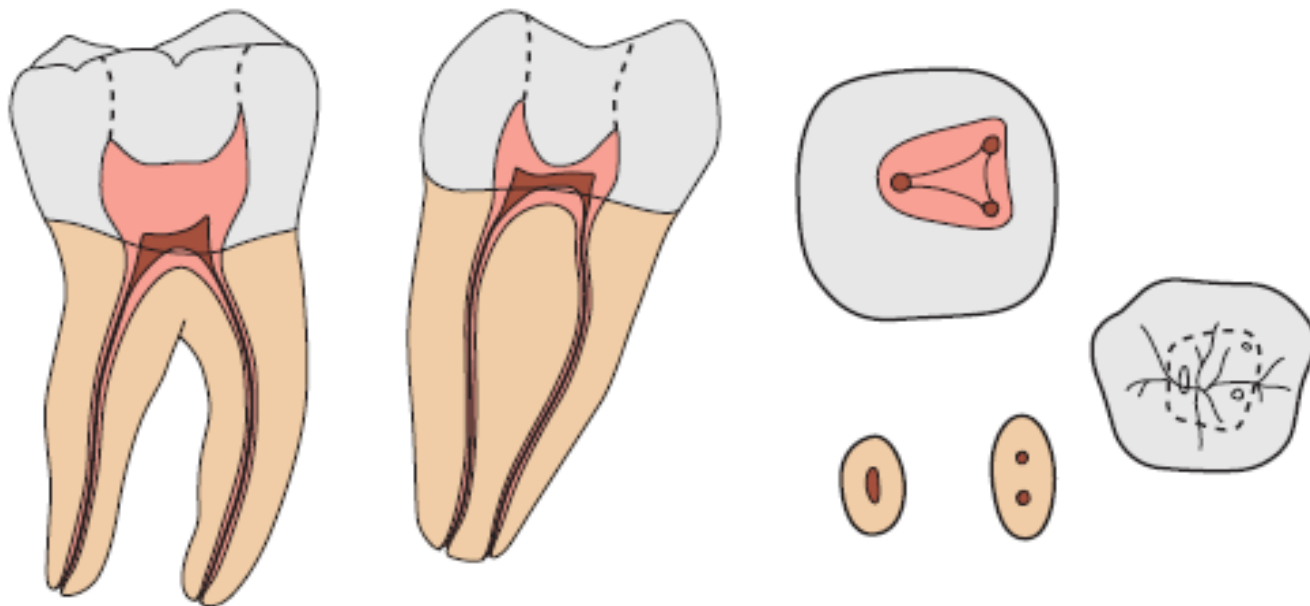
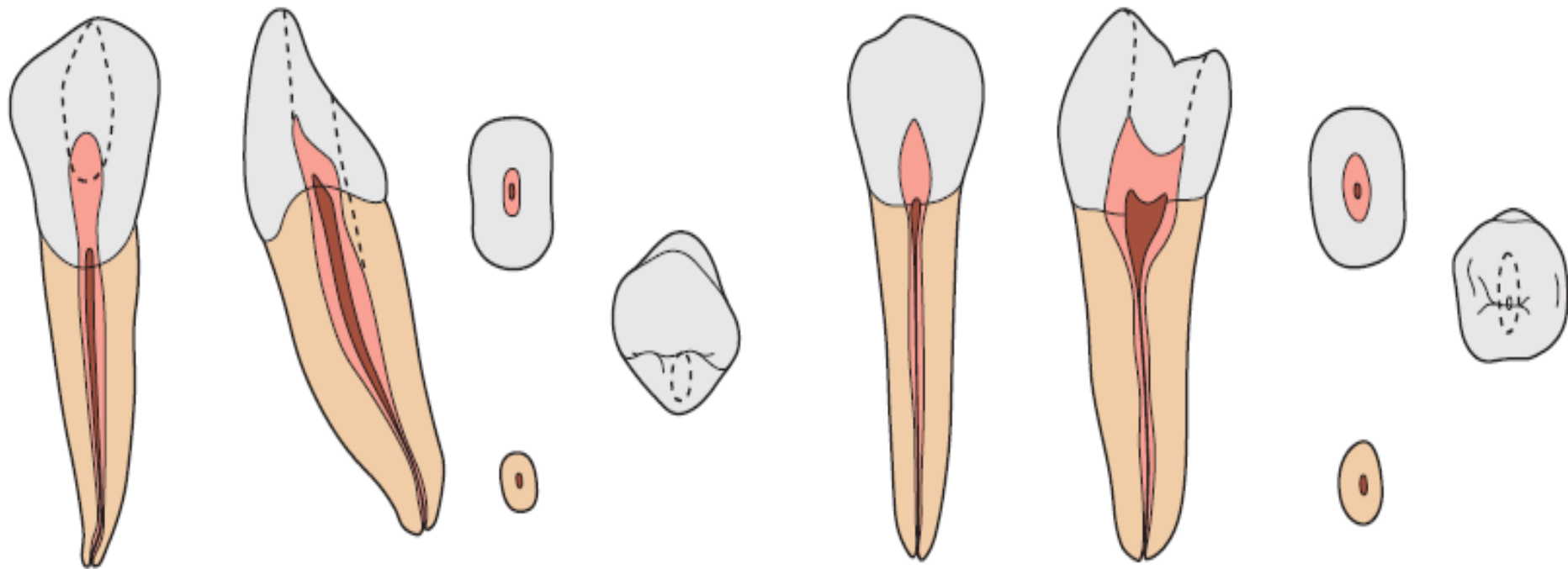
FIGURES OF ACCESS OPENING











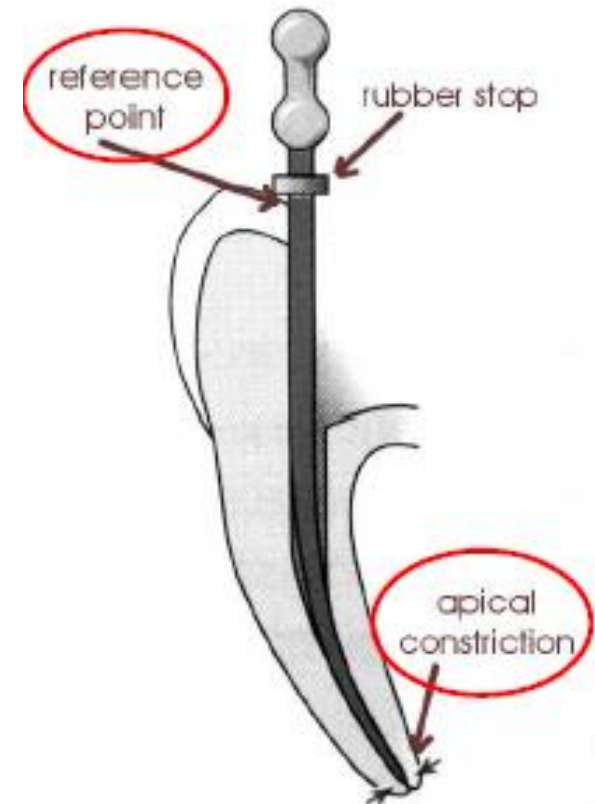
Working Length Determination

Dr. Nawfal Zakarea

PhD., MSc., Cons. Dentistry

Working length- definition:

1. The distance from a coronal reference point to the point at which canal **preparation** and **obturation** should terminate.
2. The first step in cleaning and shaping is working length determination.



importance:

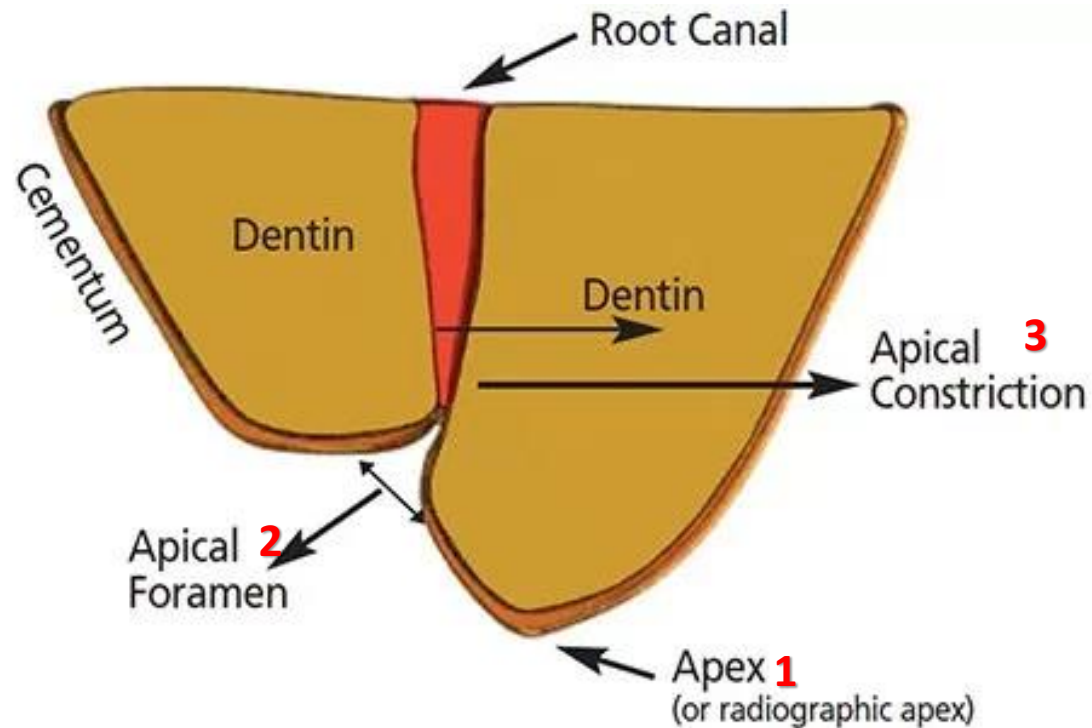
- 1) Determine the **instrument** length in the canal.
- 2) Limits the depth to which the **canal filling** maybe placed.
- 3) Limits the **postoperative pain & discomfort** as instrumentation shorter than the apical constriction leaves uncleaned space, while beyond the apical constriction irritate the periapical tissues, violate the apical zone and affect the compaction of the filling material against the apex.
- 4) Determination the **success** of treatment.

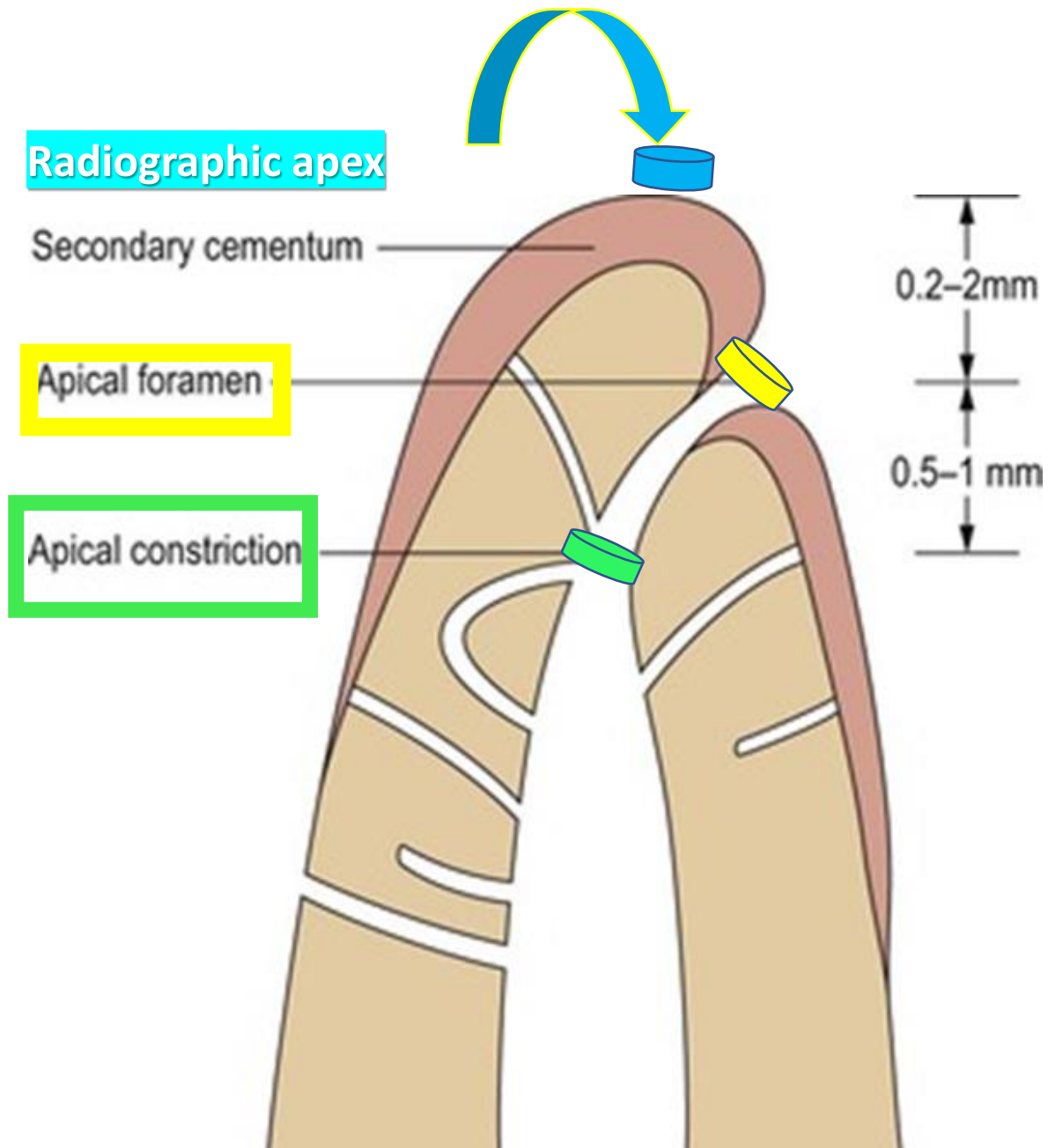
Anatomy of the Apex

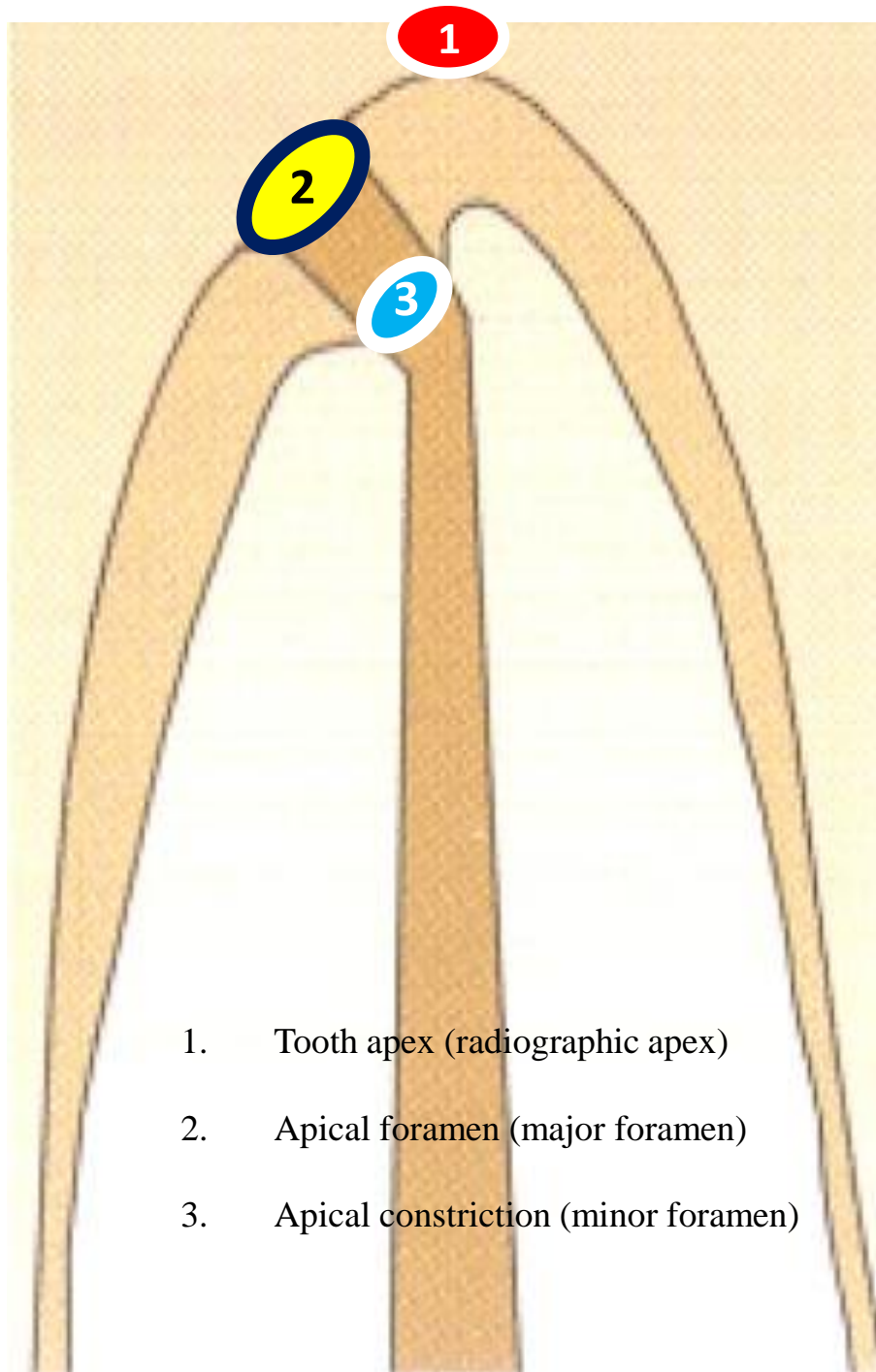
1. Tooth apex (radiographic apex)

2. Apical foramen (major foramen)

3. Apical constriction (minor foramen) very near CDJ







1. Tooth apex (radiographic apex)
2. Apical foramen (major foramen)
3. Apical constriction (minor foramen)

The terminal part of a tooth root exhibits 4 distinct landmarks:

1- Apical Foramen AF: is the main apical opening of the root canal

2- Apical Constriction AC: is defined as the apical part of the root canal with the narrowest diameter near CDJ.

3- Radiographic Apex RA: is the root end as identified radiographically.

Reference points

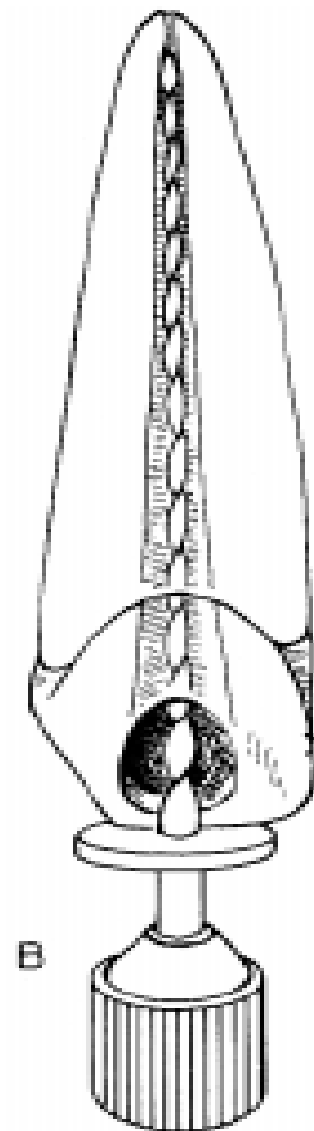
It is the site on the occlusal or incisal surface from which measurements are made.

This point is used throughout canal preparation, irrigation & obturation.

The measurement should be made from reference point on the crown, in close proximity to the straight-line path of the instrument, a point that can be identified and monitored accurately.

A definite, repeatable plane of reference to an anatomic land mark on the tooth is necessary.

usually the incisor edge in anterior teeth , the cusp tip in posterior teeth).

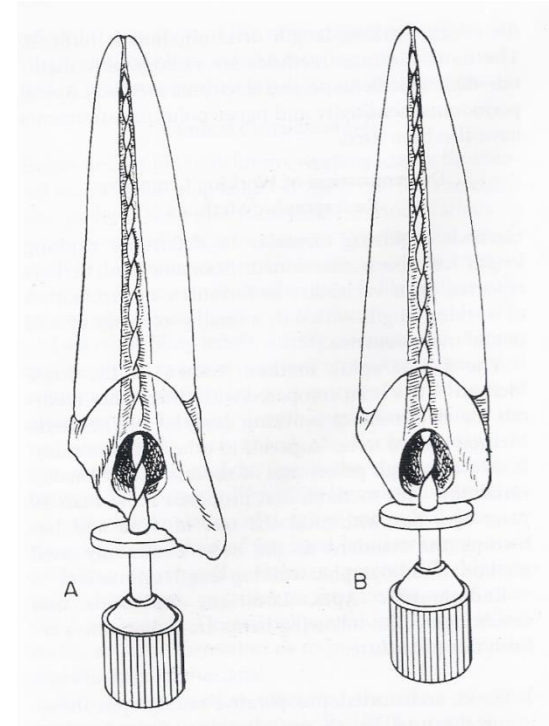


Reference points

Don't use weakened enamel walls, temporary filling or diagonal lines of fracture as a reference site for length of tooth measurement.

Diagonal surfaces should be flattened to give an accurate site of reference.

Weakened cusps or incisal edges are reduced to a well – supported tooth structure.



Requirements of W.L. Determination

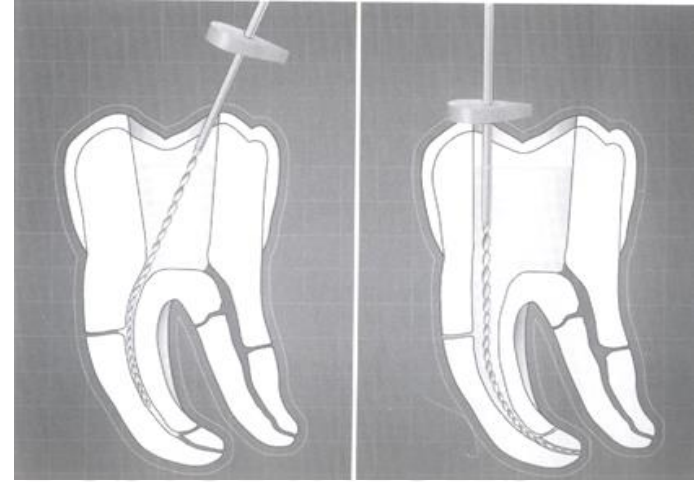
1. Knowledge of pulp anatomy and **average length** of each individual root.
2. Good, undistorted **preoperative radiographs** (parallel technique) showing the total length and all roots of the involved tooth.
3. Straight line access.
4. Small stainless steel **K-files (8,10)** facilitate the process and the exploration of the canal.
5. A definite, repeatable plane of reference, it should be noted on the patient record.

Techniques of W.L Determination

- 1. Radiographic methods**
- 2. Electronic apex locators**
- 3. Digital (finger) tactile methods**
- 4. Paper point evaluation**
- 5. By apical periodontal sensitivity**

Factors improve the W.I. determination

- 1. Cervical widening (Coronal preflaring)**
- 2. Dry canal**
- 3. Proper file size selection**
- 4. Good apex locator type**
- 5. Removal of any metallic (amalgam)**
- 6. Sharp radiographic image**



Rubber stopper

1. Should be thick enough
2. Perpendicular to the file
3. Not loose
4. With notch or guide to determined the direction of curvature and amount of rotation

One stopper
is used →



← Two stoppers are used



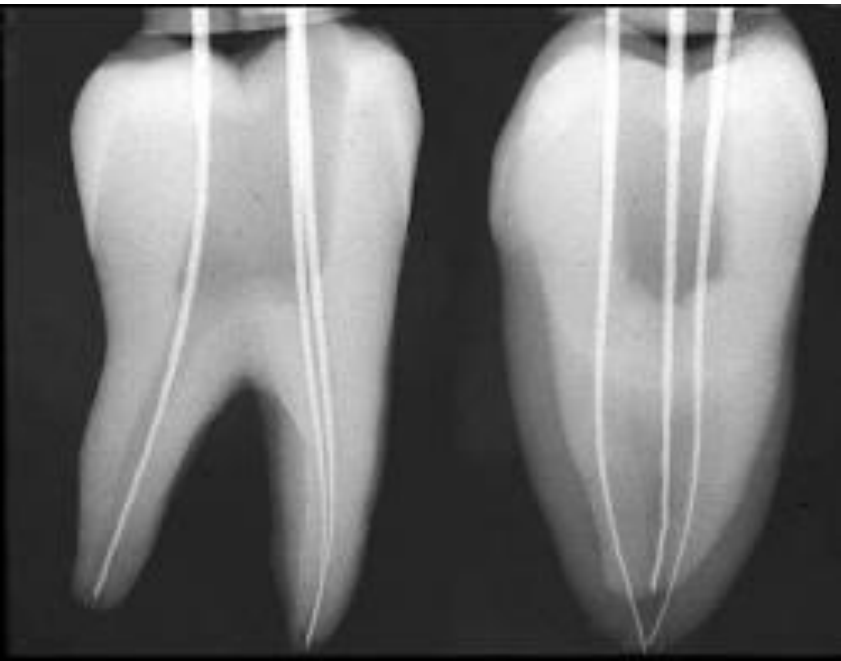
Correct



Incorrect

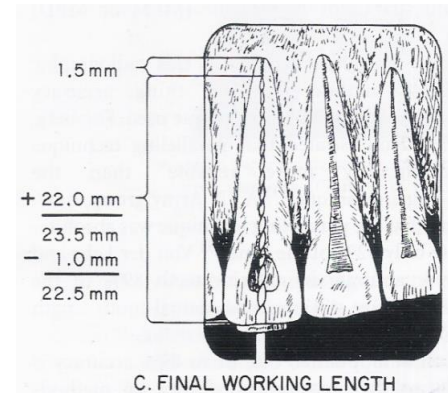
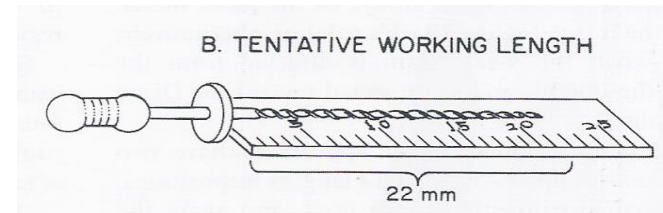
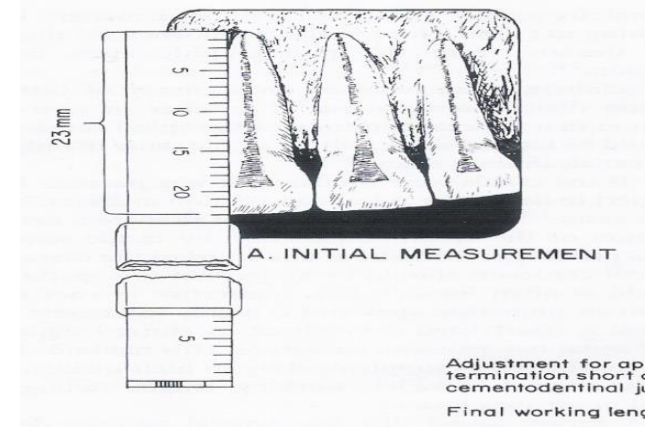
1. Radiographic Methods

- Preoperative radiograph
- Radiograph of the tooth with endodontic instrument placed to its tentative working length.
- Parallel technique is preferable over bisecting technique



Ingles Method

1. Measure the tooth on the preoperative Radiograph to the radiographic apex. **23mm**
2. Subtract at least 1.0mm " safety allowance " for possible image distortion. **22mm**
3. Set the endodontic ruler at this tentative working length and adjust the stop on instrument at that level. **22mm**
4. Place the instrument in the canal until the stop is at the plane of reference. **22mm**



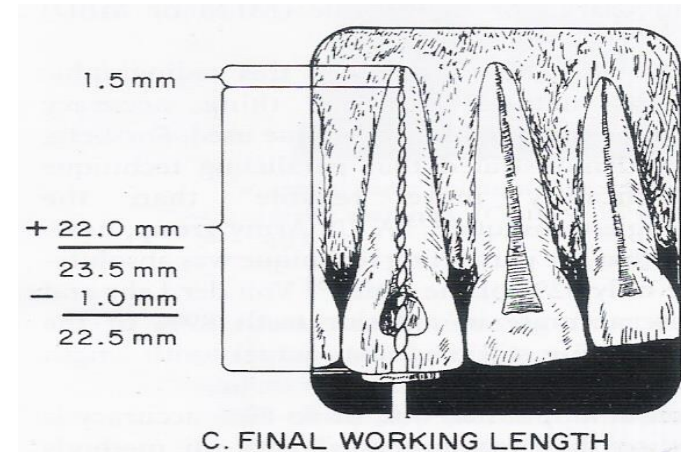
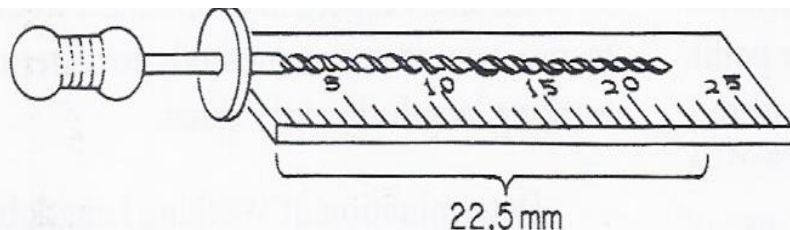
5. On the radiograph, measure the difference between the **end of the instrument** and the **end of the root 1.5mm**

and add this amount to the original measured length of the instrument. **23.5mm**

6. From this adjusted length of tooth, **subtract a 1.0 mm "Safely factor"** to conform with apical termination of the root canal at the apical constriction. **22mm**

7. Set the endodontic ruler at this new corrected length (**22mm**) and readjust the stop on the exploring instrument.

FINALLY, the file tip shorter 1mm from radiographic apex



2. Digital tactile method

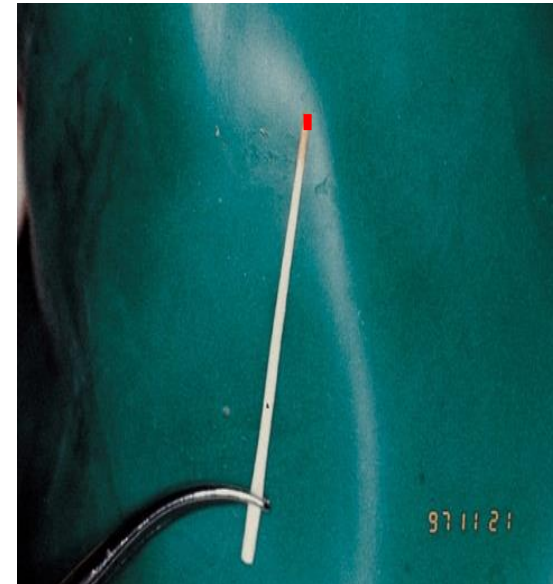
- Although it may appear to be very simple, its accuracy depends on sufficient experience.
- The clinician should be able to literally feel the foramen by tactile sense.
- Confirmation may be done either by the radiographic or electronic method.



3. Paper point evaluation

In a root canal with an immature (wide open) apex, the most reliable means of determining WL is to gently pass the blunt end of a paper point into the canal after profound anesthesia

The moisture or blood on the portion of the paper point that passes beyond the apex is an estimation of WL or the junction between the root apex and the bone.



4. By apical periodontal sensitivity

- By insertion of a small instrument beyond the apical constriction to stimulate pain.
- **It is not an accurate method and has the following disadvantages:**
 1. In inflamed tissue hydrostatic pressure developed may cause moderate to severe instant pain.
 2. When pain is afflicted in this manner, little useful information is gained by clinician, and **considerable damage is done to patient's trust.**
 3. Sometimes remnants of pulp tissue could induce pain leading to underestimation of working length.

5. Electronic apex locators

- In today's practice - one of the most important and essential instrument in endodontic practice
- These devices all attempt to locate the apical constriction, the cemento-dentinal junction, or the apical foramen.
- The most accurate one is **ROOT ZX II** by MORITA company



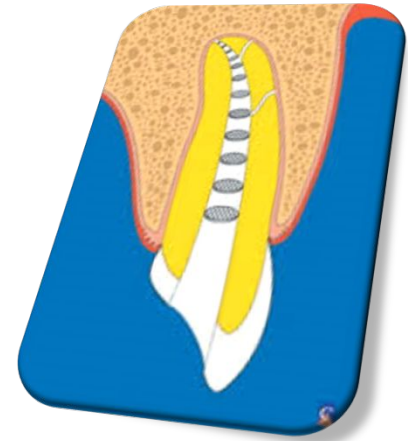
Root Canal bio-mechanical Preparation

Dr. Nawfal Zakarea

BDS., MSc., PhD., Cons. Dentistry

Basic Objectives in Cleaning and Shaping **MECHANICAL OBJECTIVES**

- 1. Continuous tapering conical shape**
- 2. Narrow apically and widest coronally**
- 3. Apical foramen as narrow as possible**
- 4. Avoid transposition of apical foramen**
- 5. Creation of sufficient space for obturating material**
- 6. Give disinfecting irrigants access to the apical canal space**
- 7. Maintain original canal anatomy**
- 8. Maintain original canal curvature**
- 9. Remove infected soft and hard tissue**



Objectives

Biological

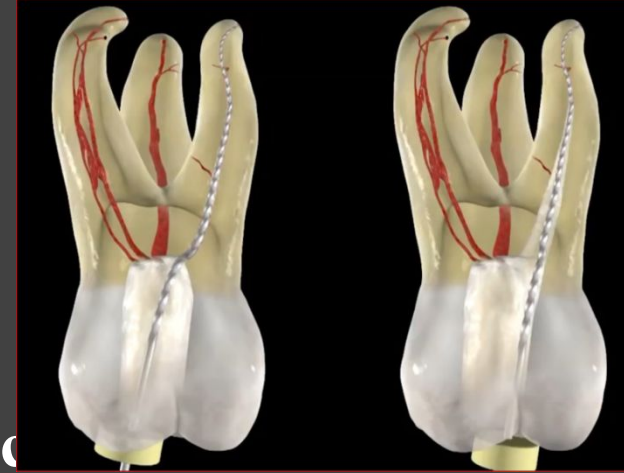
- 1-Completely debride the pulp space from:
- 2-Pulp tissue
- 3-Bacteria or fungi
- 4-Endotoxins
- 5-Removal of smear layer

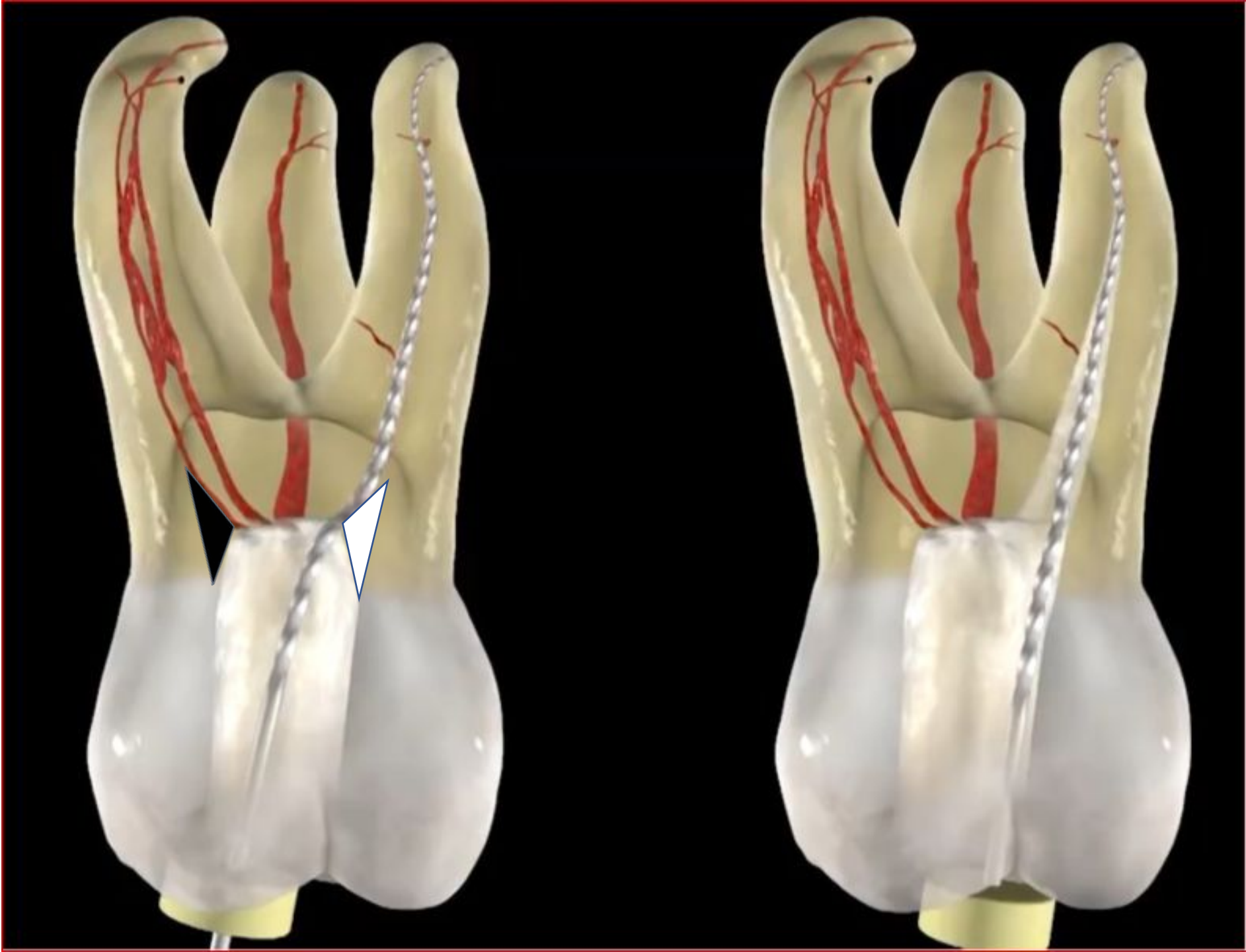
Mechanical

- 1-Continuously tapered preparation
- 2-Maintain original anatomy
- 3-Maintain the position of the apical foramen
- 4-Maintain apical construction as small as particularly possible

PRINCIPLES OF CANAL INSTRUMENTATION

1. Straight line access of the file
2. Pre-curving the apical part of file
3. Always use Irrigate between each file size
4. Always use recapitulation
5. Removal of dentinal debris by activation of irrigants by ultrasonic
6. Always use EDTA 17% paste coating the file before insertion
7. Never apply apical pressure
8. Never engage the file to dentin
9. Examination of file shape before use it
10. Measure the length of file after removal from the canal



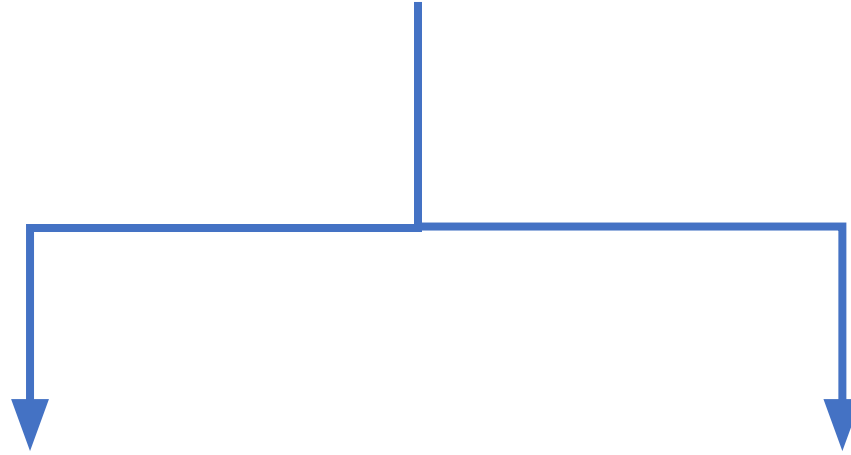


Advantages of CORONAL PRE-flaring

1. REDUCED POTENTIAL OF EXCTROSION DEBRIS BEYOND APEX
2. APICAL GAUGING MORE ACCURATE
3. PREVENTS PREMATURE BINDING OF INSTRUMENTS TO CANAL WALLS
4. BETTER IRRIGATION
5. BETTER VISION
6. BETTER SENSATION (because in narrow canal it is impossible to scout the canal)
7. INCREASE THE ACCURACY OF W.L. DETERMINATION
8. REDUCE THE RISK OF BROKEN FILE
9. ICREASES THE VOLIUM OF IRRIGANT (reduce the blockage possibility & control the instrumentation)



Techniques of Root Canal Preparation



Initial apical prep:

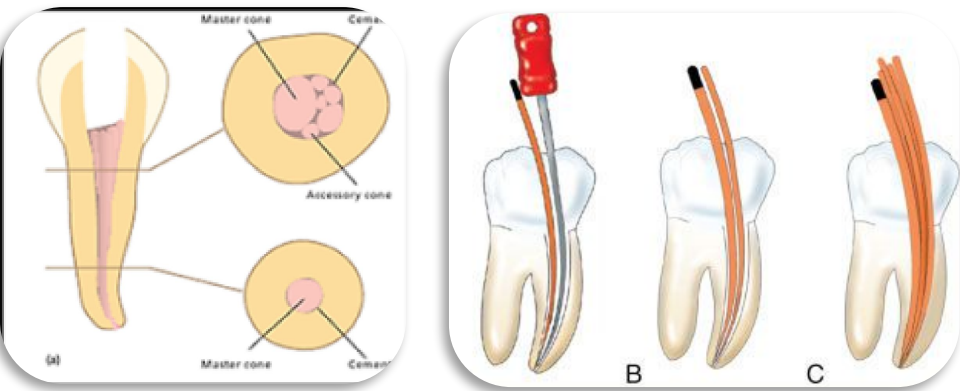
1. Standardized tech.
2. Step back tech.

Initial coronal prep:

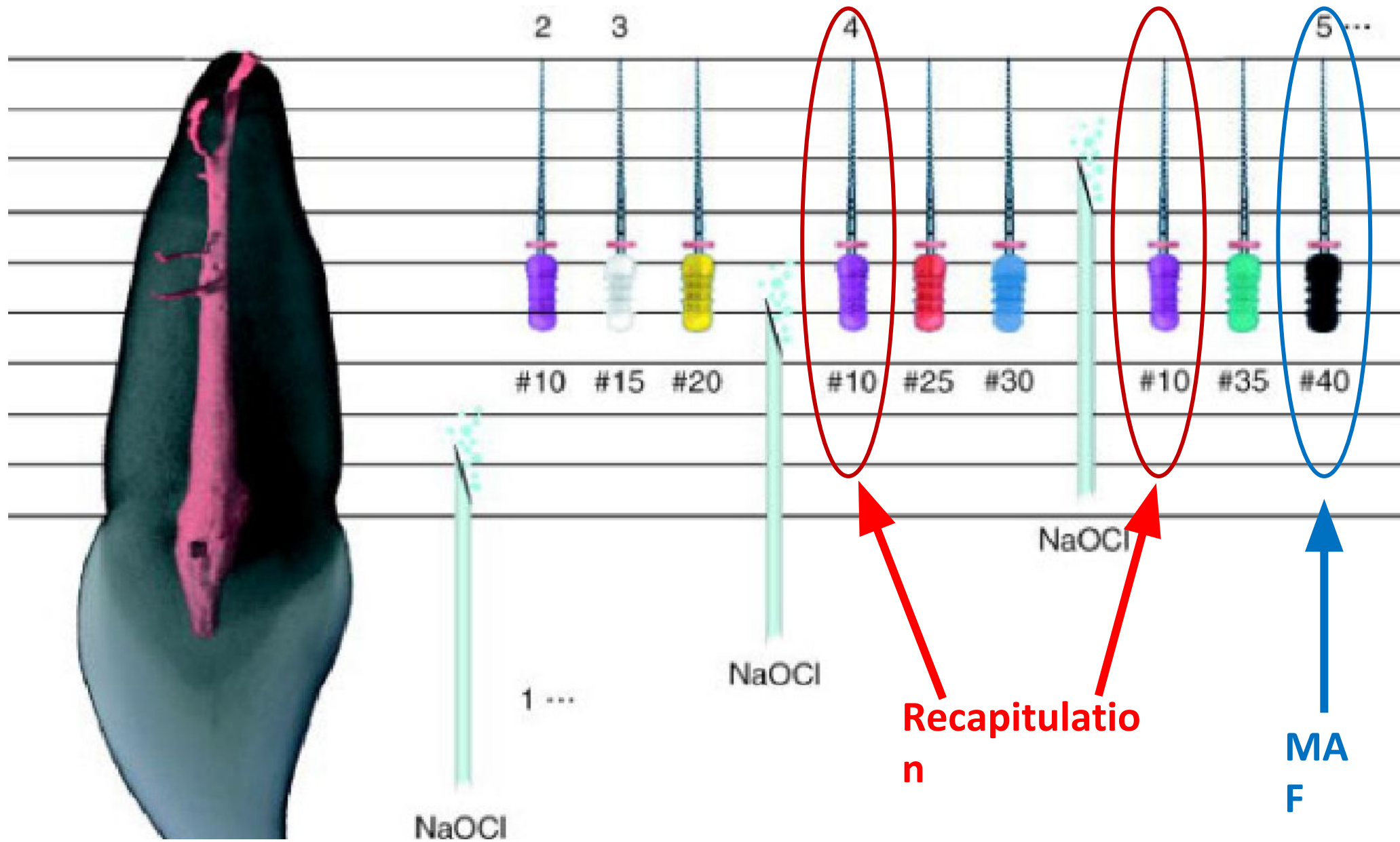
Stepdown tech.

Standardized Technique

1. This technique use the ISO hand instrument in an ascending manner (15, 20, 25, 30.... etc.) sequentially, all inserted to full working length.
2. It result in a canal shape like the shape of the last instrument used.
3. Canal enlarged until **clean dentin chips** are seen on the apical few mm of the instrument in case of necrotic canal, but in vital tooth **3 sizes larger than the first file that fit snugly to full working length** .
4. Obturation with lateral condensation technique.



Standardized tech. Illustration



- *Recapitulation:*

Is the re-insertion of small files during canal preparation to full working length in order to keep the apical area clean and patent, this should be done along with irrigation size 10 file.

- *Patency:*

Means the maintenance of the apical portion of the canal free of debris (dentin chips) by recapitulation with a small file (size 8 or 10) **through** the apical foramen, it keeping the foramen open in case drainage is needed from the periapical tissues.

- *Master Apical File (MAF):*

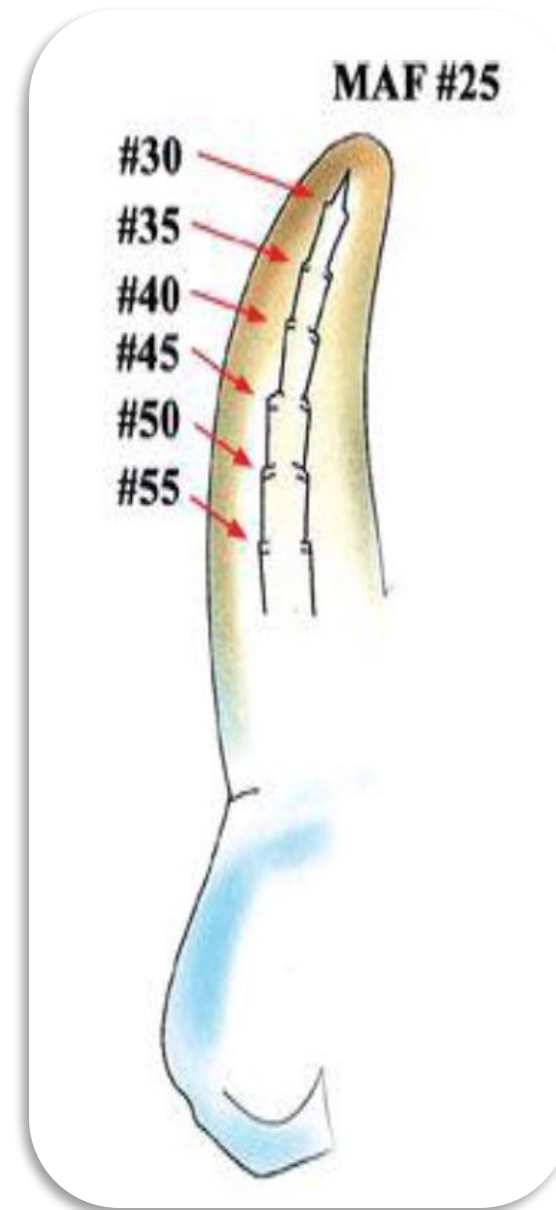
Is the largest file used to the full working length of a completely prepared root canal.

DISADVANTAGES

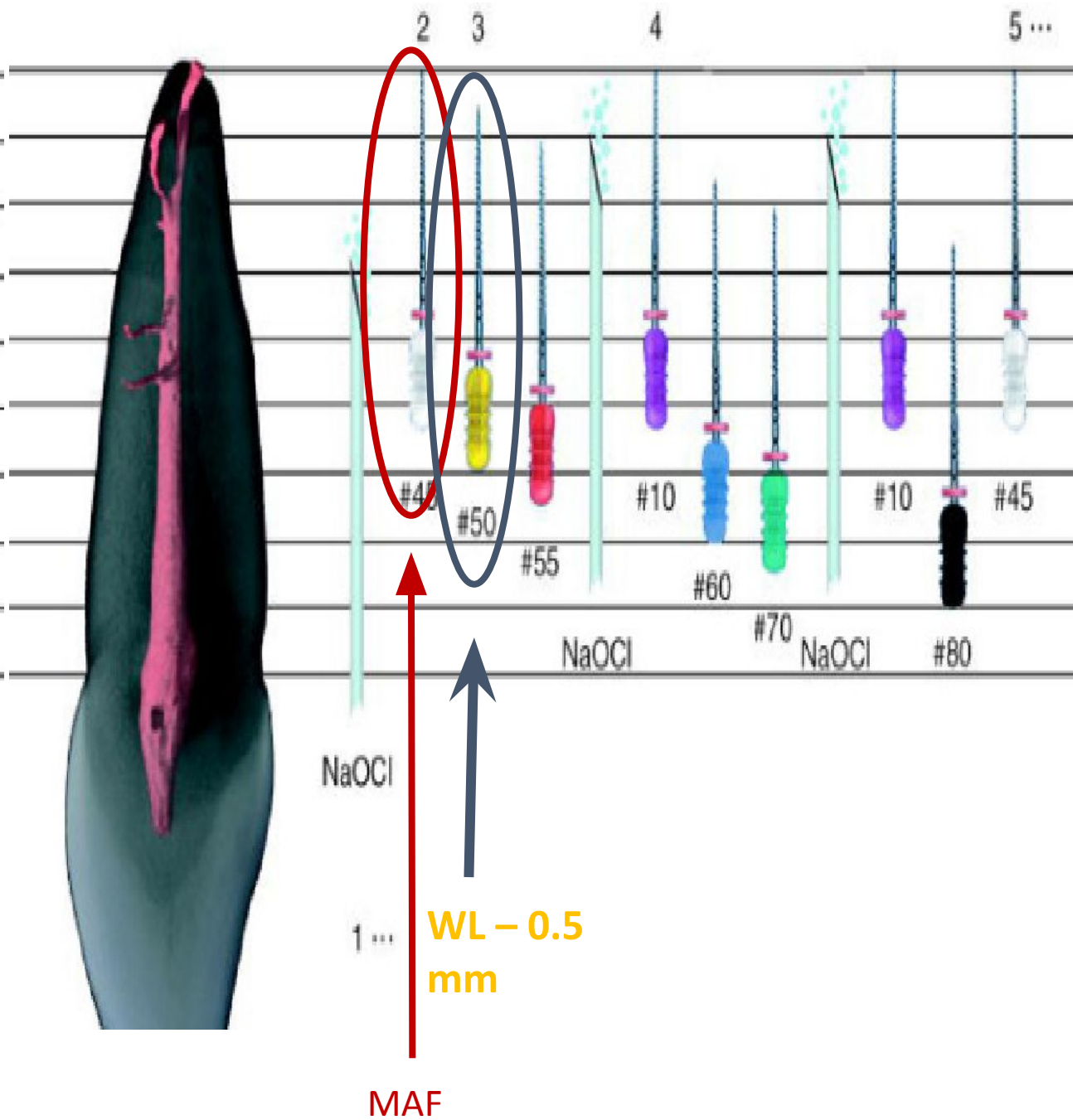
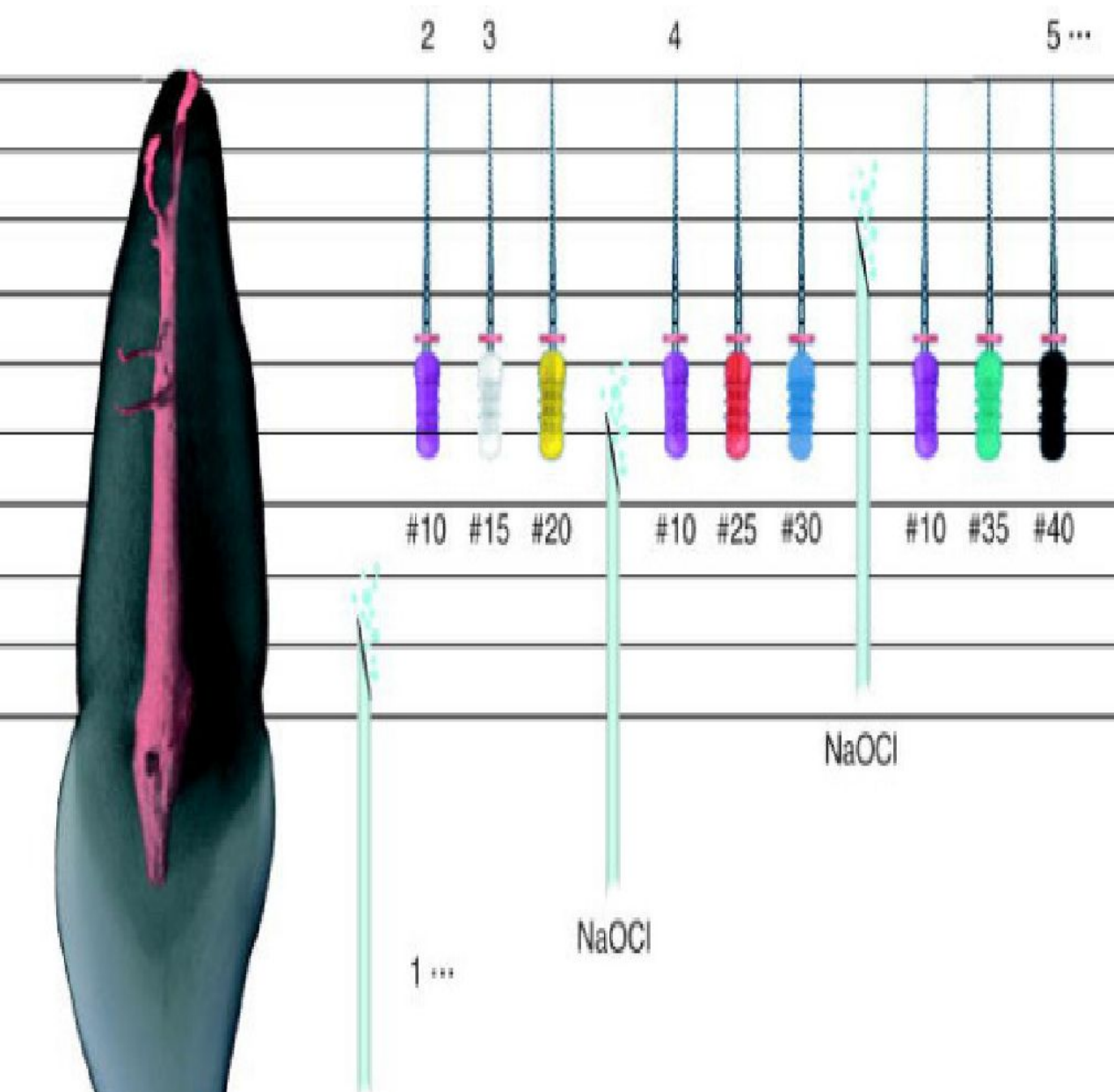
1. In small curved canals it could lead to many procedural errors due to compaction of dentin debris
2. Chances of pushing debris into peri-radicular tissues
3. In oval shaped canals a large areas of the canal could be left un-instrumented

Step-back technique

1. The step-back technique reduces procedural errors and improves debridement.
2. After determining the master apical file, the succeeding larger files are shortened by 0.5 or 1.0 mm increments from the previous file length.
3. This step-back process creates a flared, tapering preparation while reducing procedural errors.
4. The **step-back preparation is superior to standardized technique** in debridement and maintaining the canal shape.









WL=21
SIZE=25



WL=20.5
SIZE=30



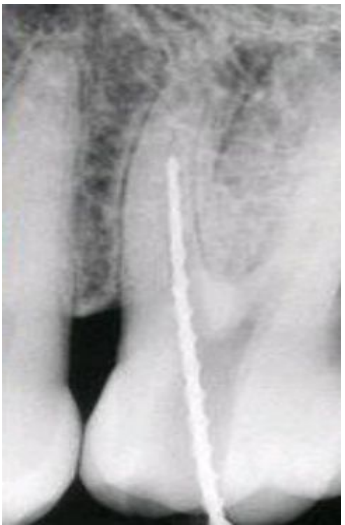
WL=20
SIZE=35



WL=19.5
SIZE=40



WL=19
SIZE=45



WL=18.5
SIZE=50



WL=18
SIZE=55



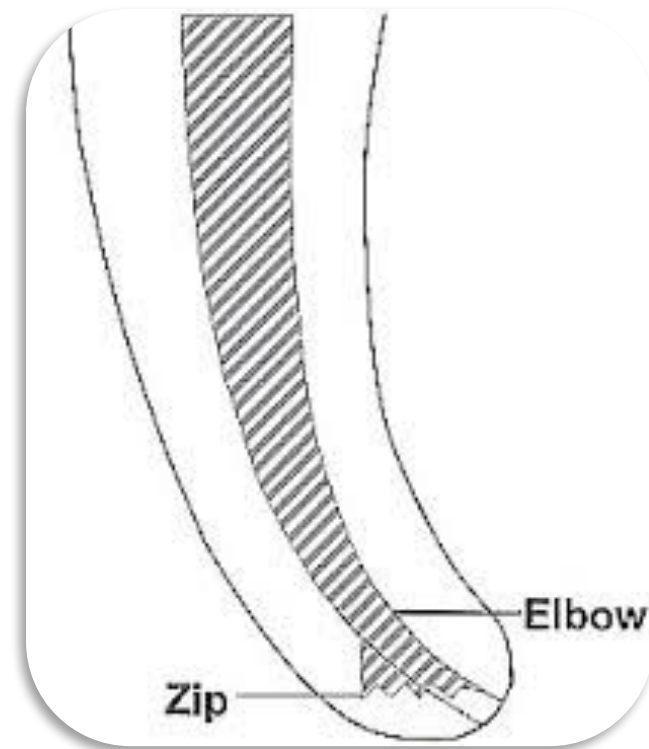
WL=17.5
SIZE=60



WL=17
SIZE=70

Advantages of step-back tech

1. **Better tactile awareness.**
2. **Keeps apical preparation small in its original position.**
3. **Greater taper coronally compared to standard prep so more dentin removal and cleaner walls.**
4. **Avoids zipping.**



Disadvantages of step-back tech

- 1. Chances of pushing debris into peri-radicular tissues (post operative pain & tenderness).**
- 2. Working length likely to change as canal curvatures are eliminated.**



Step-Down (Crown-down) Technique

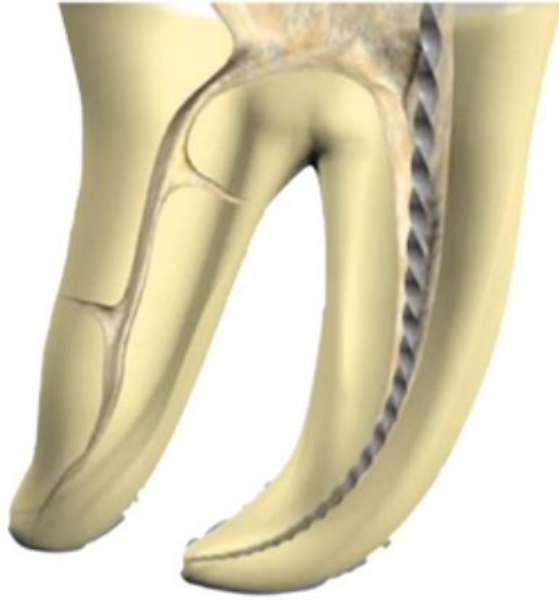
- 1. The step-down technique is advocated for cleaning and shaping procedures as it removes coronal interferences and provides coronal taper.**
- 2. With the pulp chamber filled with irrigant or lubricant, the canal is explored with a small instrument to assess patency and morphology (curvature). The working length can be established at this time.**
- 3. The coronal one third of the canal is then flared with Gates-Glidden drills or rotary files of greater taper 10%.**



Step-Down technique



Flaring-Preflaring



Working Length-Glide Path



shaping



finishing

- 4. A large file (such as No. 70) is then placed in the canal, and a watch-winding motion is used until resistance is encountered.**
- 5. Sequentially smaller files + 1MM till working length**
- 6. The process is repeated with sequentially smaller files until the apical portion of the canal is reached.**
- 7. The working length is checked again.**
- 8. The apical portion of the canal can now be prepared by enlarging the canal at the corrected working length.**



1 Gates-Glidden #4
irrigate

2 Gates-Glidden #3
irrigate

3 Gates-Glidden #2
irrigate

4 K-flex type file #60
irrigate

5 K-flex type file #55
irrigate

6 K-flex type file #45
irrigate

7 K-flex type file #40
irrigate

When 3mm from estimated
working length verify actual
working length with x-ray or
apex locator.

8 K-flex type file #35
irrigate

9 K-flex type file #30
irrigate with EDTA
irrigate & dry

Advantages of Crown-down technique

- 1. Elimination of microbes and infected dentin**
- 2. Less extrusion of debris apically**
- 3. Less postoperative pain and tenderness**
- 4. Better and deeper penetration of irrigant**
- 5. Less strain and physical load on the file apically**
- 6. Less possibility of ledge formation and file separation**
- 7. Provide more tactile sensation in the apical 1/3rd**
- 8. Less time for preparation**
- 9. It also reduces the risk of compacting debris apically which may block the canal.**

Hand instruments manipulation

- 1. Filing**
- 2. Reaming**
- 3. Watch winding**
- 4. Circumferential filing**
- 5. Balanced force technique**

• *Filing:*

Is the push and pull movement of a file within the canal. **K-files** and **H-files** can be used for this movement, the later has better cutting efficiency.

• *Reaming:*

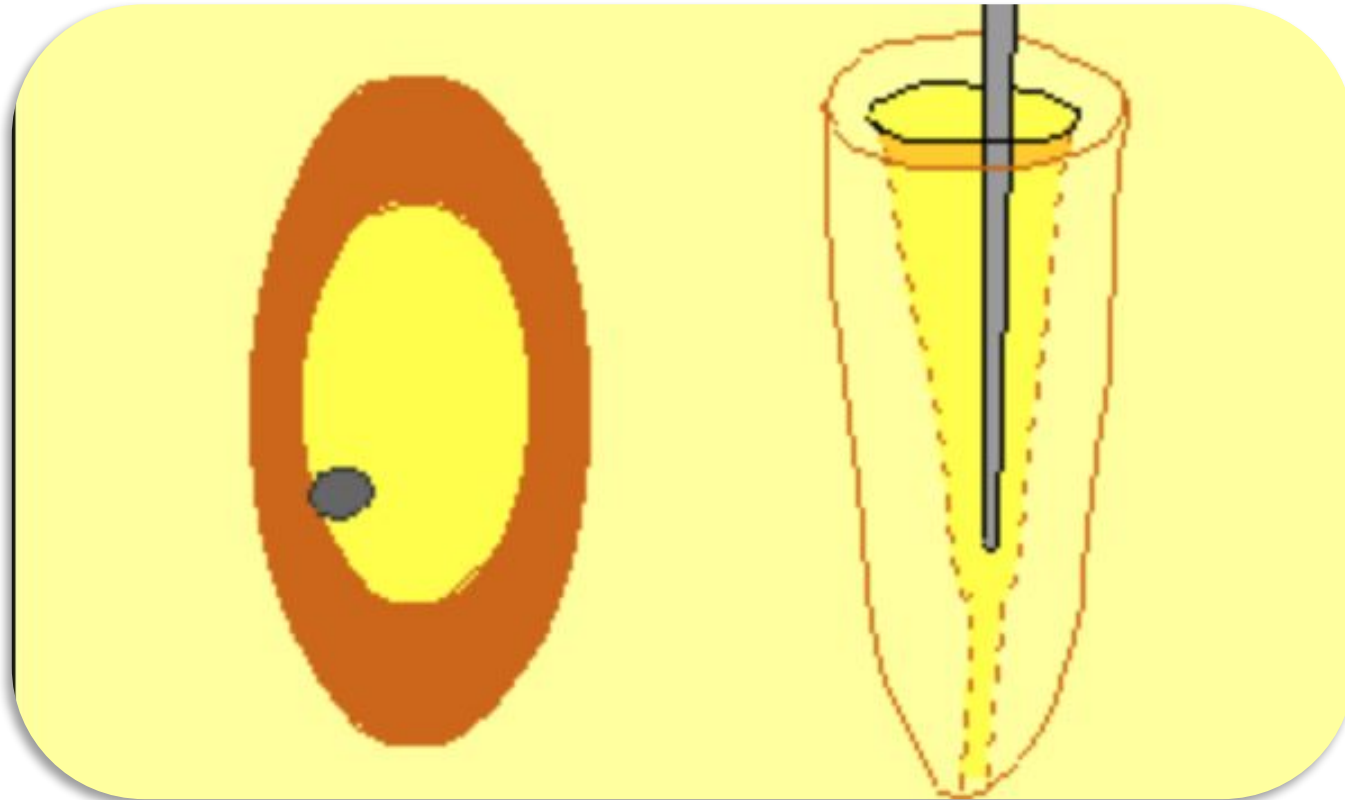
Is the quarter turn and pull movement, it could be used by **reamer** or **file**, the quarter turn is rotated clockwise to engage the canal dentin and the pull movement is to cut this dentin off the wall. H-file is contra indicated for reaming due to possibility of fracture.

• *Watchwinding*

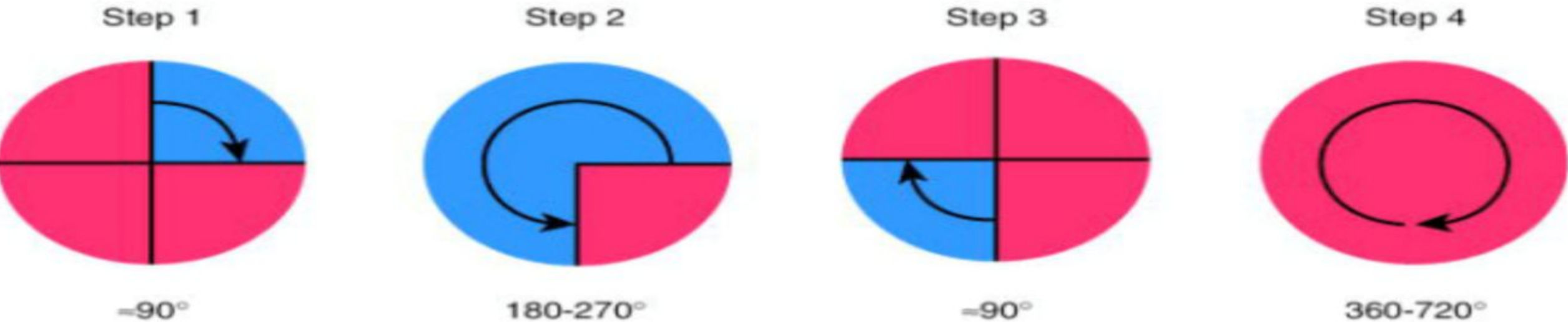
A continuous back and forth rotation with slight apical pressure, with this movement a **fine file** rapidly advances down a root canal. Only fine files should be advanced to the apex in this way as there is a danger of compacting pulpal debris ahead of the file.

- Circumferential filing:**

A push-pull filing movement circumferentially around the canal walls. K-files and H-files are used with an attempt to file on the outstroke only, to reduce the apical compaction of debris.



BALANCED FORCE TECHNIQUE (ROANE)



apical pressure.

- **Step 2:** The instrument is rotated counterclockwise 180 to 270 degrees; sufficient apical pressure is used to keep the file at the same insertion depth during this step. Dentin shavings are removed with a characteristic clicking sound.
- **Step 3:** This step is similar to step 1 and advances the instrument more apically.
- **Step 4:** After two or three cycles, the file is loaded with dentin shavings and is removed from the canal with a prolonged clockwise rotation.

•*Advantages of Balanced Force Tech:*

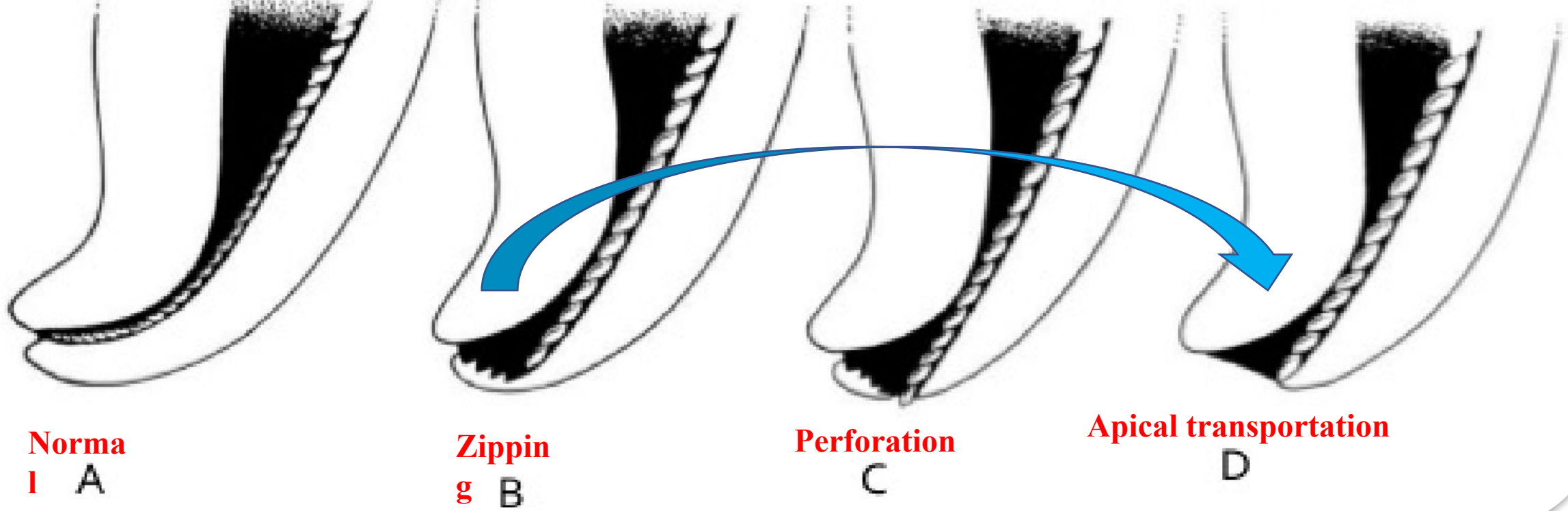
1. Preparation is centered better around original canal outline.
2. Allows for larger apical preparation.
3. Less need to precurve the file.

Disadvantages:

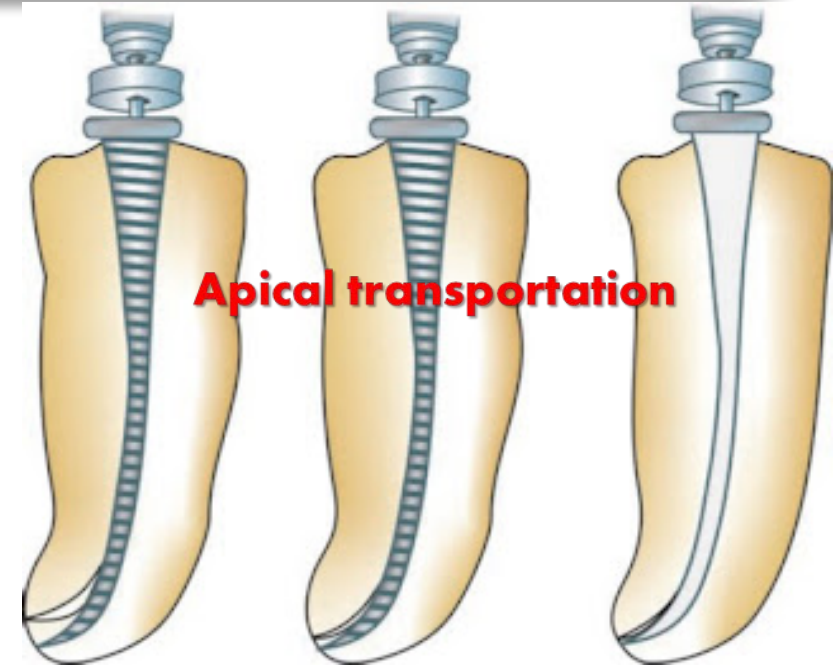
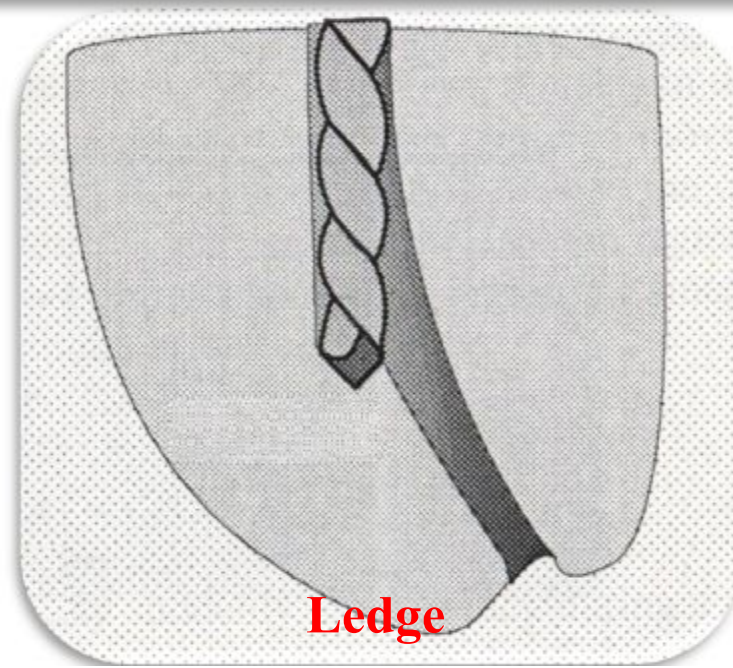
1. Instrument breakage
2. Canal stripping

Procedural Errors during instrumentation

- a. Canal blockage and Loss of working length.**
- b. Separated instrument**
- c. Over-instrumentation**
- d. Ledge formation.**
- e. Zipping**
- f. Perforation**
- g. Canal transportation**



ledge

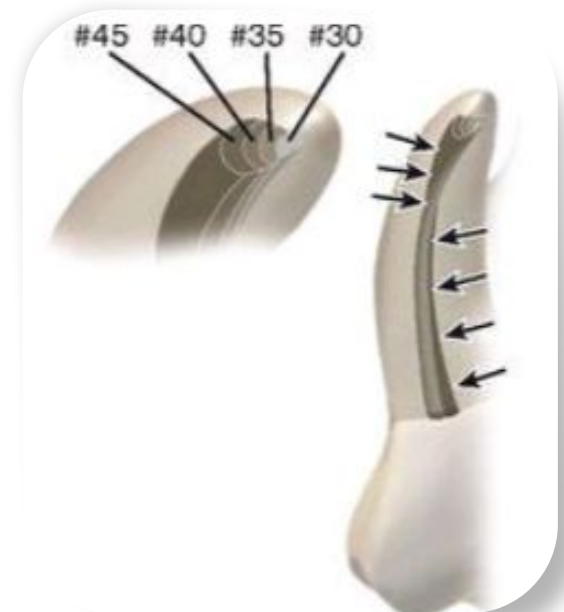


zipping

Zippering in Endodontics:

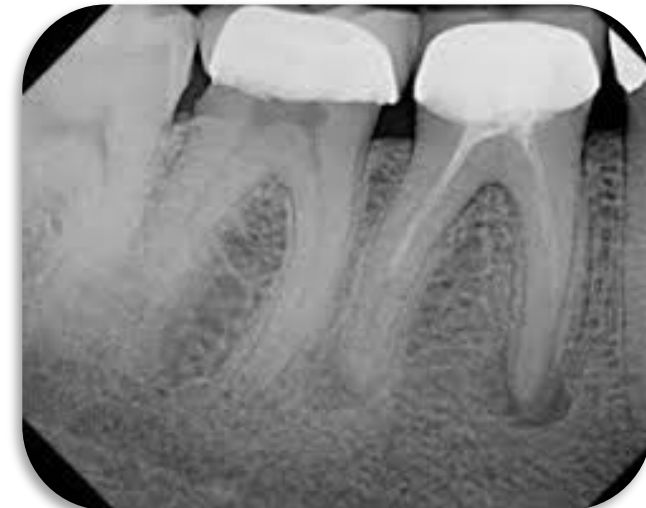
Is defined as the apical transportation of a curved canal caused due to improper shaping technique.

When a curved canal is filed with pressure against the outer side of the curvature.



Loss of working length

1. It is a very common and frustrating error usually noted on a master cone radiograph
2. It is actually secondary to other procedural error.
3. This error could be overcome by good recapitulation and irrigation



Ledge formation

Ledge is an iatrogenically created irregularity in the root canal that impedes access of instruments to the apex.

Causes

- 1. Inflexible instrument in curved canals.**
- 2. Over enlargement of the curved canals.**
- 3. Not extending the access sufficiently (out line error)**
- 4. Incorrect assessment of root canal direction**
- 5. Incorrect working length determination**
- 6. Forcing a large instrument in the canal**
- 7. Using non curved stainless steel instrument in a curved canal**
- 8. Failure to use the instruments in sequential order**

Prevention of ledge formation

- 1. Careful instrumentation**
- 2. Appropriate preoperative and working length radiographs**
- 3. Copious irrigation**
- 4. The use of pre-curved S/S in small sizes files or NiTi files in in large sizes**
- 5. Sequential instrumentation**
- 6. Careful attempting to remove blockage**

perforation

An artificial opening in a tooth or its root created by boring, piercing, cutting or pathologic resorption, which results in communication between the pulp space and the periodontal tissues.

Causes:

- 1. Caries**
- 2. Resorptive defects**
- 3. Iatrogenic events**

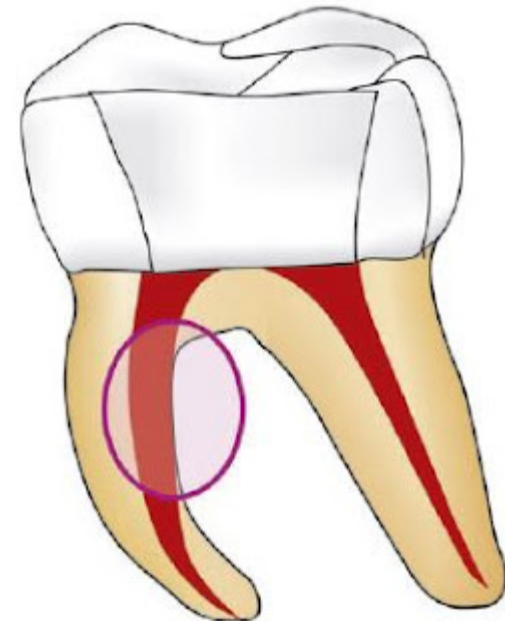
Perforation:

Identification of perforation :

- 1. Sudden appearance of fresh bleeding from the canal.**
- 2. Pain during canal preparation.**
- 3. Sudden loss of apical stop.**

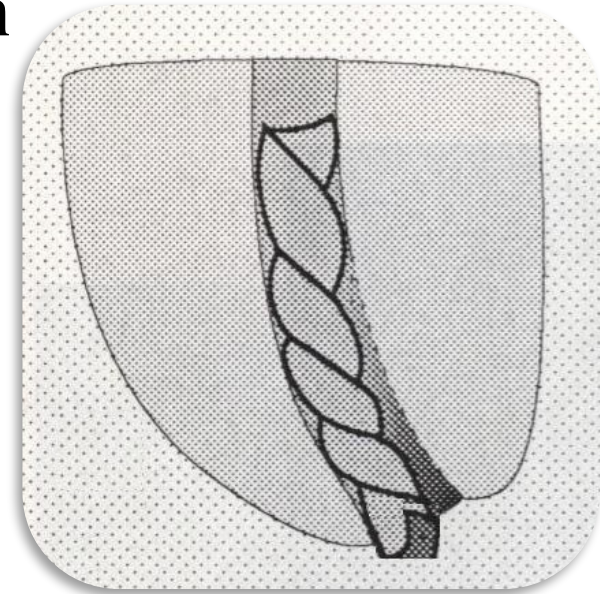
CLASSIFICATIONS OF PERFORATION

1. **APICAL THIRD**
2. **MIDDLE THIRD**
3. **CERVICAL THIRD**
4. **BIFORCATION PERFORATION**
5. **PERFORATION VIA APICAL FORAMEN**
6. **STREP PERFORATION**



Over instrumentation

- **This occurs due to wrong working length estimation.**
- **It can be treated by re establishing the apical constriction by enlarging the canal on a shorter length and maintaining the funnel shape, then very carefully obturate the canal on the corrected working length preventing any extrusion.**
- **Apical barrier with Mineral Trioxide Aggregate (MTA) is another option to solve over instrumentation**



1. Tissue emphysema caused by over extrusion of hydrogen peroxide H_2O_2 irrigant into the tissues or pushing air in to the canal.



2. Sodium hypochlorite accident



3. Instrument Aspiration and Ingestion supine position without rubber dam



Pre-Curving



Pre-Bending



Orifice Modification = Coronal Flaring

Orifice Modifier

- Enlarging Canal Orifice
- Achieving Coronal Flaring To gain SLA to the rest of RC.

 From Shahad Zahed to Everyone



RECAP “Clinical Steps”

- 1- Isolation.
- 2- Access Cavity Preparation.
- 3- Patency #10.
- 4- Coronal Flaring.
- 5- W.L. Determination.
- 6- Glide Path.
- 7- Shaping.
- 8- Disinfection (Irrigation).
- 9- Obturation.
- 10- Coronal Restoration.

Root Canal obturation

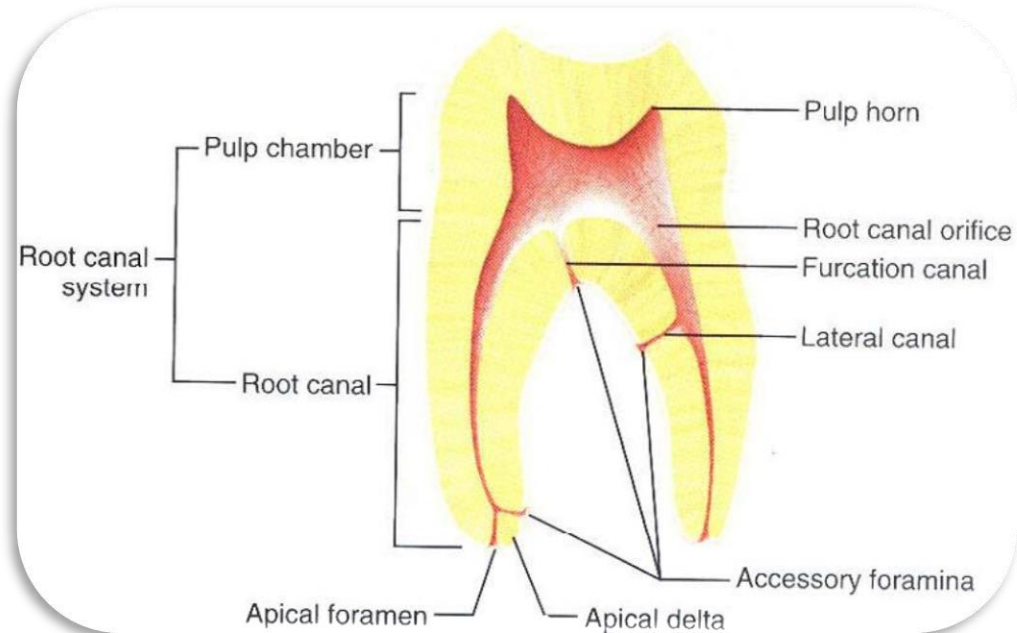
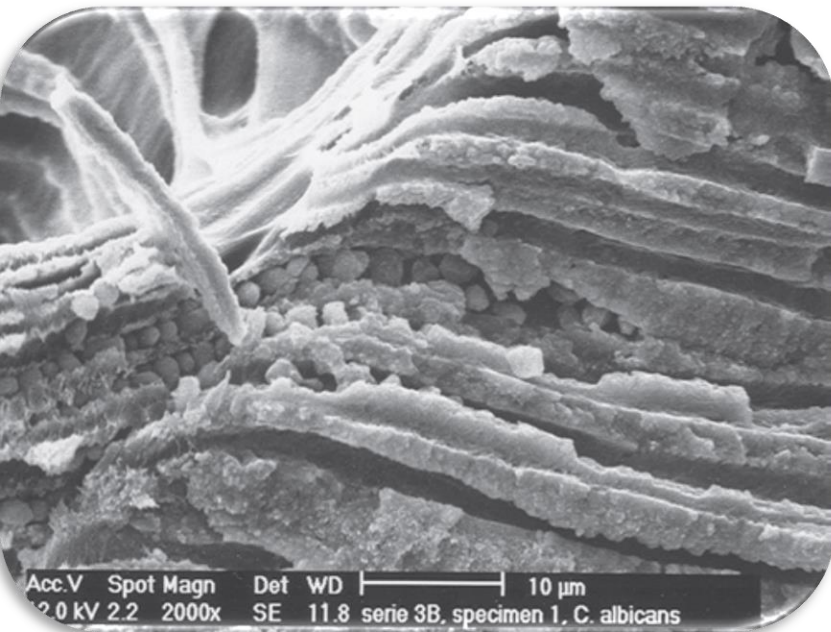
Nawfal Zakarea

MSc., PhD., Conservative Dentistry

Objectives of Root Canal Filling

1. To prevent microorganisms which remain **in the root canal system** after preparation from proliferating and passing into the periapical tissues **via the apical foramen** and other pathways.
2. To seal the pulp chamber and root canal system from leakage via the **coronal restoration** in order to prevent passage of microorganisms and/or toxins along the root canal and into the periapical tissues **via the apical foramen** and other pathways.

3. To prevent percolation (leakage) of **periapical exudate** and possibly microorganisms into the pulp space **via the apical foramen** and other pathways.
4. To prevent percolation of **gingival exudate** and microorganisms into the pulp space **via lateral/furcation canals opening** into the gingival sulcus or through exposed, patent dentinal tubules around the neck of the tooth.



Factors Affecting the Quality of Obturation

- 1. The complexity of root canal system.**
- 2. The quality of root canal instrumentation.**
- 3. The materials used for obturation.**
- 4. The technique used for obturation.**
- 5. The skills of the operator.**
- 6. The quality of the coronal restoration.**

Materials Used for Obturation

Core Materials

Solid

Gutta percha

Silver points

Semi-solid (paste)

Zinc-oxide
eugenol paste

Sealers

ZOE based sealers

Resin sealers

Ca(HO)₂ based
sealers

Glass ionomer
sealers



SEALERS

A root canal sealer is used in combination with the core root canal filling material, e.g. gutta-percha. The primary role of the sealer is to obliterate the microspaces between the root canal wall and the core material.

Almost all of today's root canal filling techniques use a sealer to enhance the seal of the root canal filling.

Functions of Sealers

1. cementing (luting, binding) the core material to the canal
2. filling the discrepancies between the root canal walls and core material
3. acting as a lubricant
4. acting as an antimicrobial
5. acting as a marker for accessory resorptive defects, root fractures and other spaces into which the main core material may not penetrate.

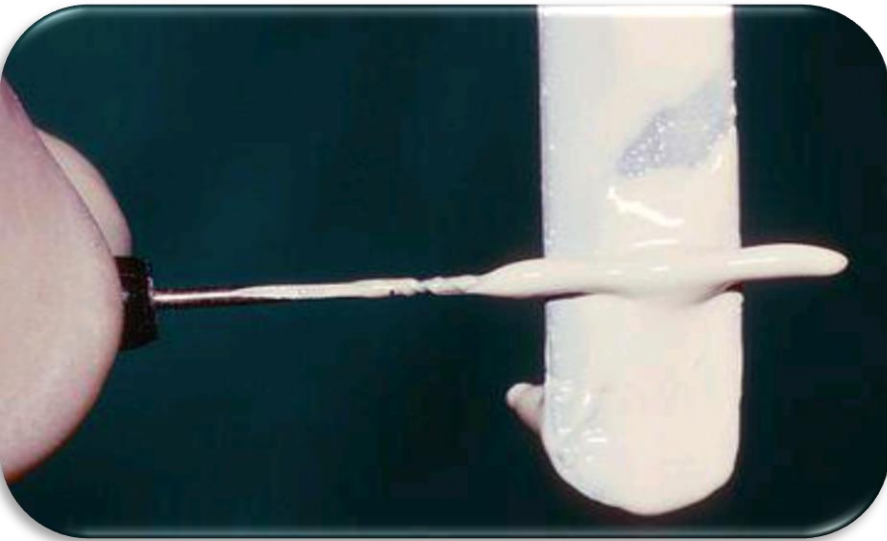


Types of Sealers

- 1) Zinc oxide–eugenol sealers**
- 2) Calcium hydroxide based sealers**
- 3) Resin sealers**
- 4) Glass ionomer sealers**
- 5) MTA based sealer**
- 6) Bioceramic sealer**

Sealer Application

1. By hand file rotated counter clock wise
2. By rotary instrument called Lentulo Spiral
3. By syringe injection
4. Sonic or ultra sonic



Gutta-percha

- **Gutta-percha has been used to fill root canals for over 130 years and is the most widely used and accepted root filling material.**
- **Gutta-percha endodontic filling points were found to contain approximately:**
 - **20% gutta-percha (matrix)**
 - **66-80% zinc oxide (filler)**
 - **11% heavy metal sulfates (radiopacifier)**
 - **3% waxes and/or resins (plasticizer).**
- Alfa

GUTTA-PERCHA OBTURATION TECHNIQUES

Cold technique

Single cone

Lateral condensation

Heat softened gutta-percha

Intra-canal heated

Extra-canal heated

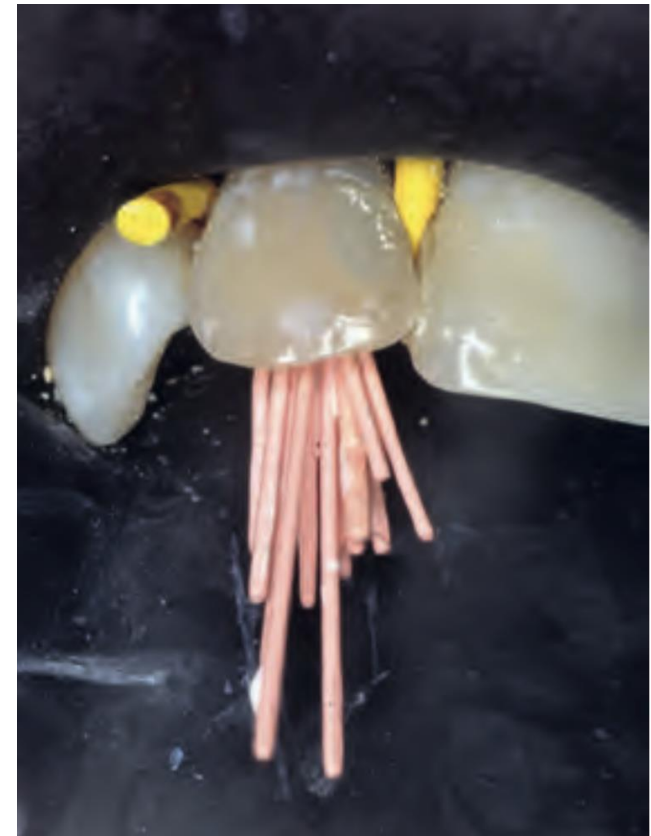
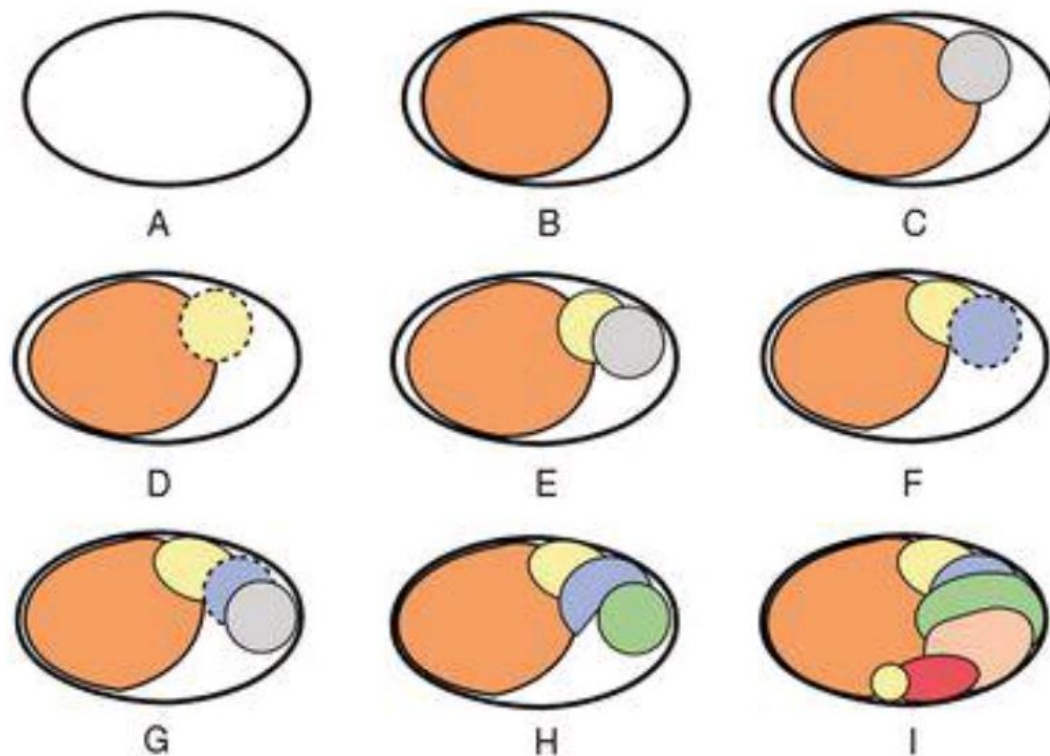
Solvent softened gutta-percha

Single cone technique

- It include filling root canals with a single, full length gutta-percha cone and sealer.
- This technique is indicated with standardized canal preparation technique, and when the root canal was prepared to a round cross-sectional shape of a standard size with reamers and filled with a gutta-percha cone of the **same diameter**.

Lateral condensation technique

It involves the placement of a **master** (primary) gutta-percha cone to the terminus of the preparation (WL) followed by placing additional (**accessory**) gutta-percha cones alongside



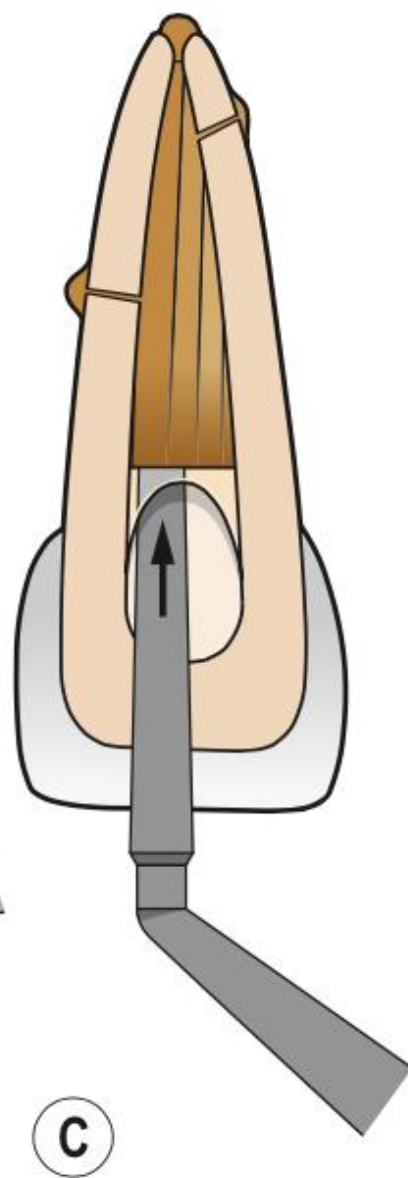
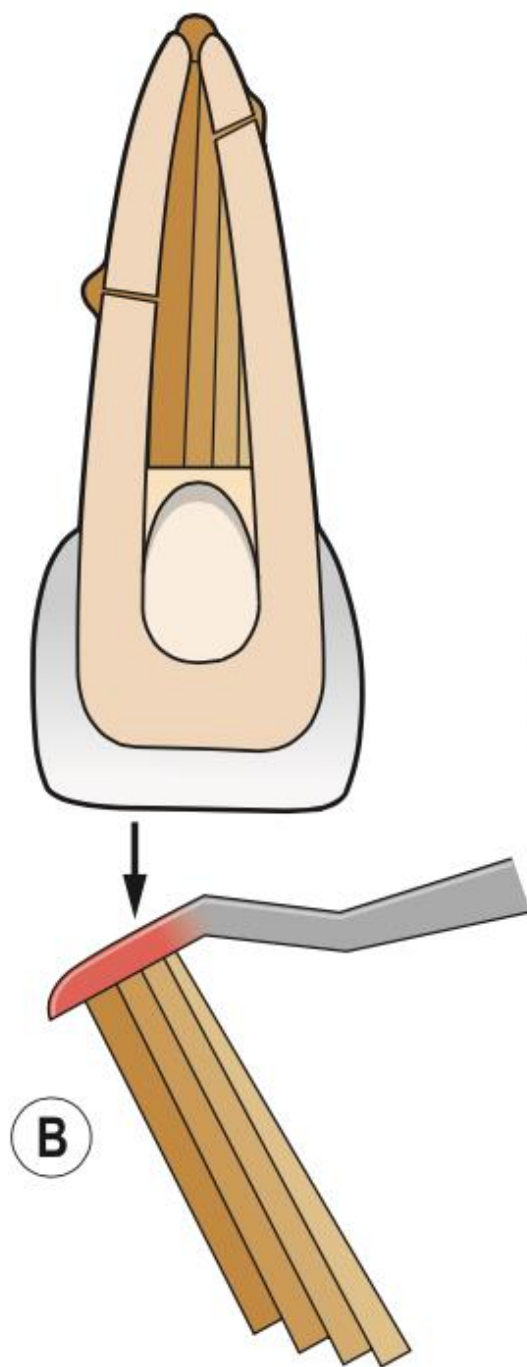
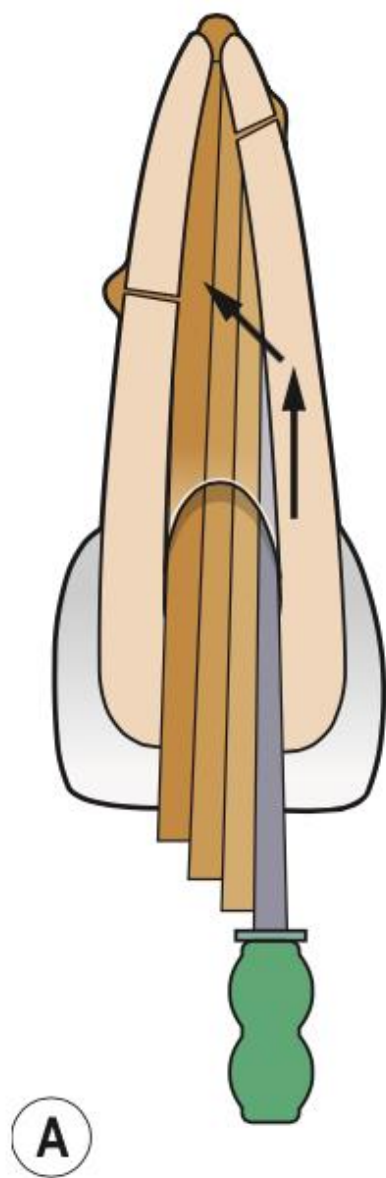
Master Cone Insertion

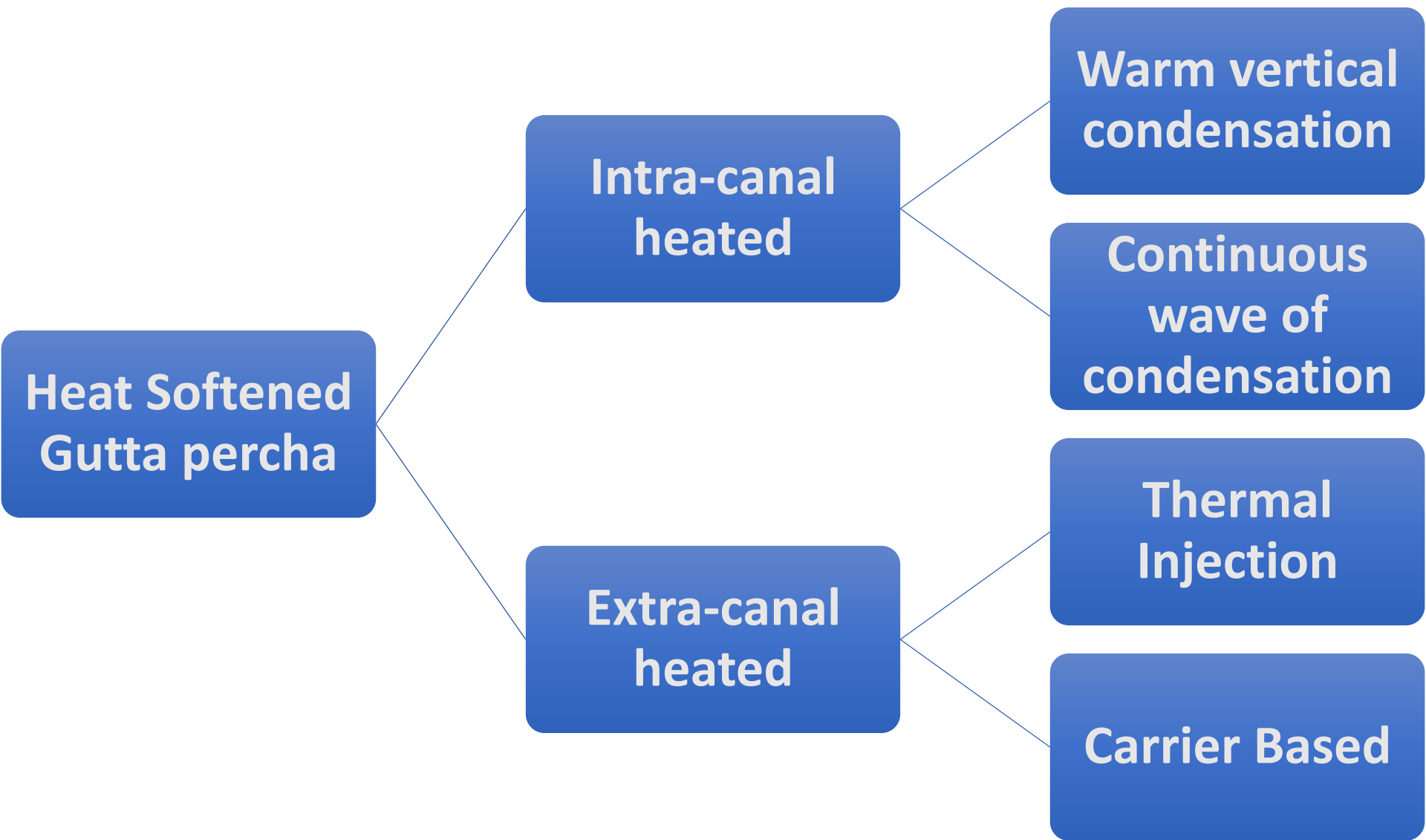
- **Master cone size should be the same as the MAF.**
- **Three requirements should be available in this cone:**
 - 1. Full working length insertion.**
 - 2. Resistance to withdrawal (tug-back action).**
 - 3. Radiographic fitness.**

Completion of lateral condensation

- 1. Master cone is removed after checking and the canal is dried carefully with paper points.**
- 2. Sealer is smeared onto the canal wall thoroughly**
- 3. The master cone should be coated lightly with sealer and then inserted immediately to the full working length.**
- 4. The spreader is then placed alongside the gutta- percha cone and pushed apically with controlled force until it reaches the appropriate depth, 1-2mm from the end-point of preparation (WL).**

5. The first accessory gutta-percha cone is inserted into the space created by the spreader.
6. The spreader is then cleaned and reinserted immediately into the canal. **It should not go down to the full working length.**
7. The second accessory gutta-percha cone is inserted into the space created.
8. The sequence of spreader application and cone insertion continues until the canal is full.
9. The excess gutta-percha emerging from the canal entrance should be removed with a hot instrument and condensed vertically with a plugger to promote a satisfactory seal.





Warm Vertical Condensation

- **Aim:**

To fill the canal with heat-softened gutta-percha packed with sufficient vertical pressure to force it to flow into the entire root canal system, including accessory and lateral canals

- **Requirements:**

A flared canal preparation with a definite apical stop. The flared nature of the canal is necessary to accommodate the plugger used to condense the gutta-percha and facilitate the flow of the material apically.

- **Advantages:**

The method produces homogeneous, compact fillings with gutta-percha flowing into irregularities, apical deltas and lateral canals.

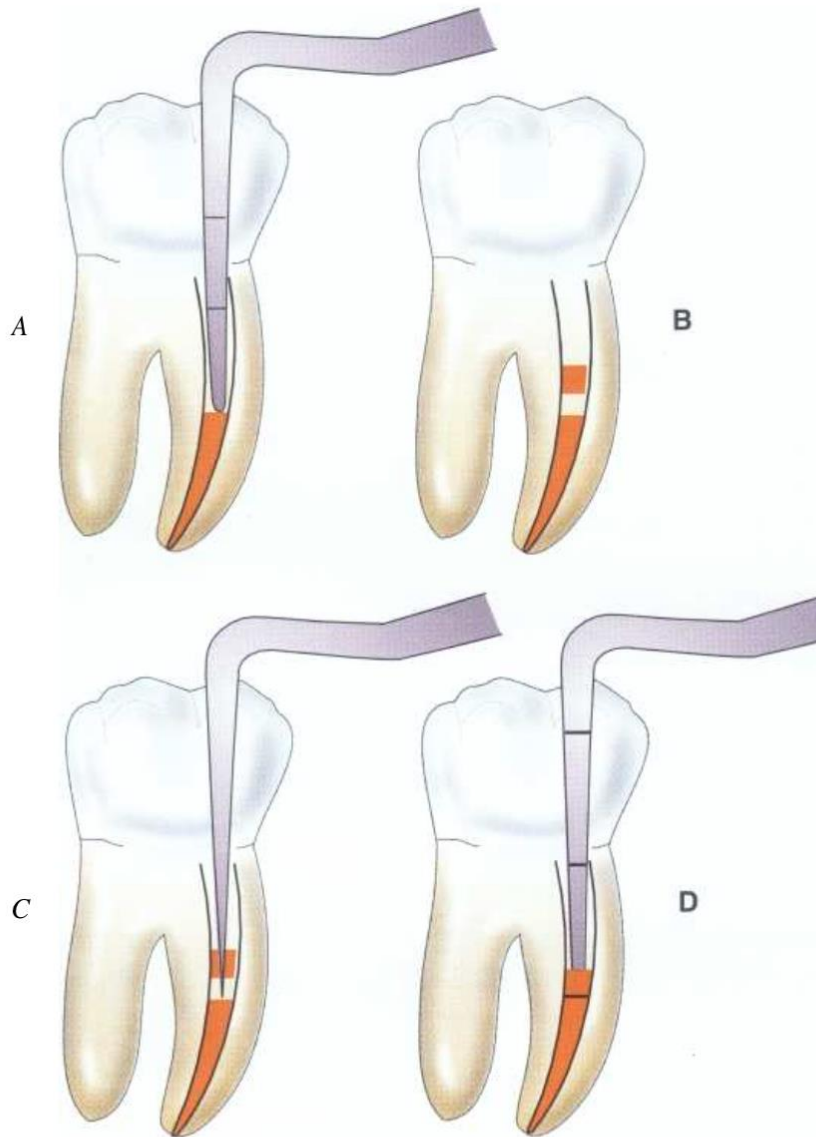


FIGURE 7-19 Diagram of the warm vertical condensation technique. A, After a heated spreader is used to remove the coronal segment of the master cone, a cold plugger is used to apply vertical pressure to the softened master cone. B, Obturation of the coronal portion of the canal is accomplished by adding a gutta-percha segment. C, A heated spreader is used to soften the material. D, A cold plugger is then used to apply pressure to the softened gutta-percha.

Technique:

- 1. The technique consists of fitting a gutta-percha cone with a taper similar to the canal, short of the apex 1-2mm.**
- 2. Heat application using a flame-heated carrier.**
- 3. The gutta-percha is softened by the heat and becomes plastic.**
- 4. Plugger then placed in the canal with apical pressure to produce a hydraulic force that moves the gutta-percha apically, against the canal walls, and into canal irregularities such as accessory canals.**
- 5. Gutta-percha is then added in small increments, and each increment of gutta-percha is heated and softened and packed vertically until the entire canal is filled.**

Continuous Wave of Condensation

- It is a modification of warm vertical condensation, this technique uses the **System B** type heat sources.
- There are two stages, **down-packing** and **back-packing**.

200 C



Down-packing:

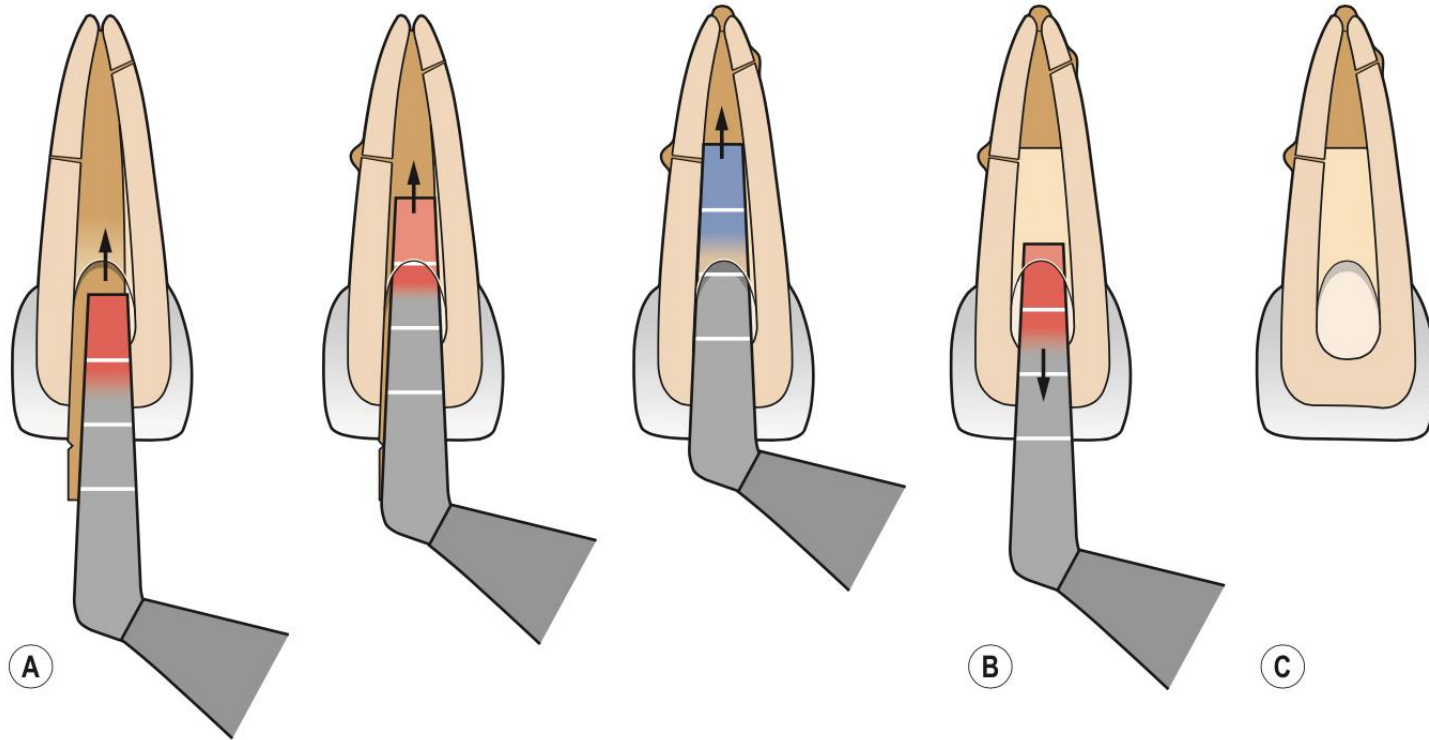
Heat is carried along the length of the master gutta-percha cone starting coronally and ending in apical third via heater. The apical and lateral movement of thermo-softened gutta-percha is referred to as a 'wave of condensation'. Down-packing is completed in a single continuous vertical movement. **Step 1**

Back-packing:

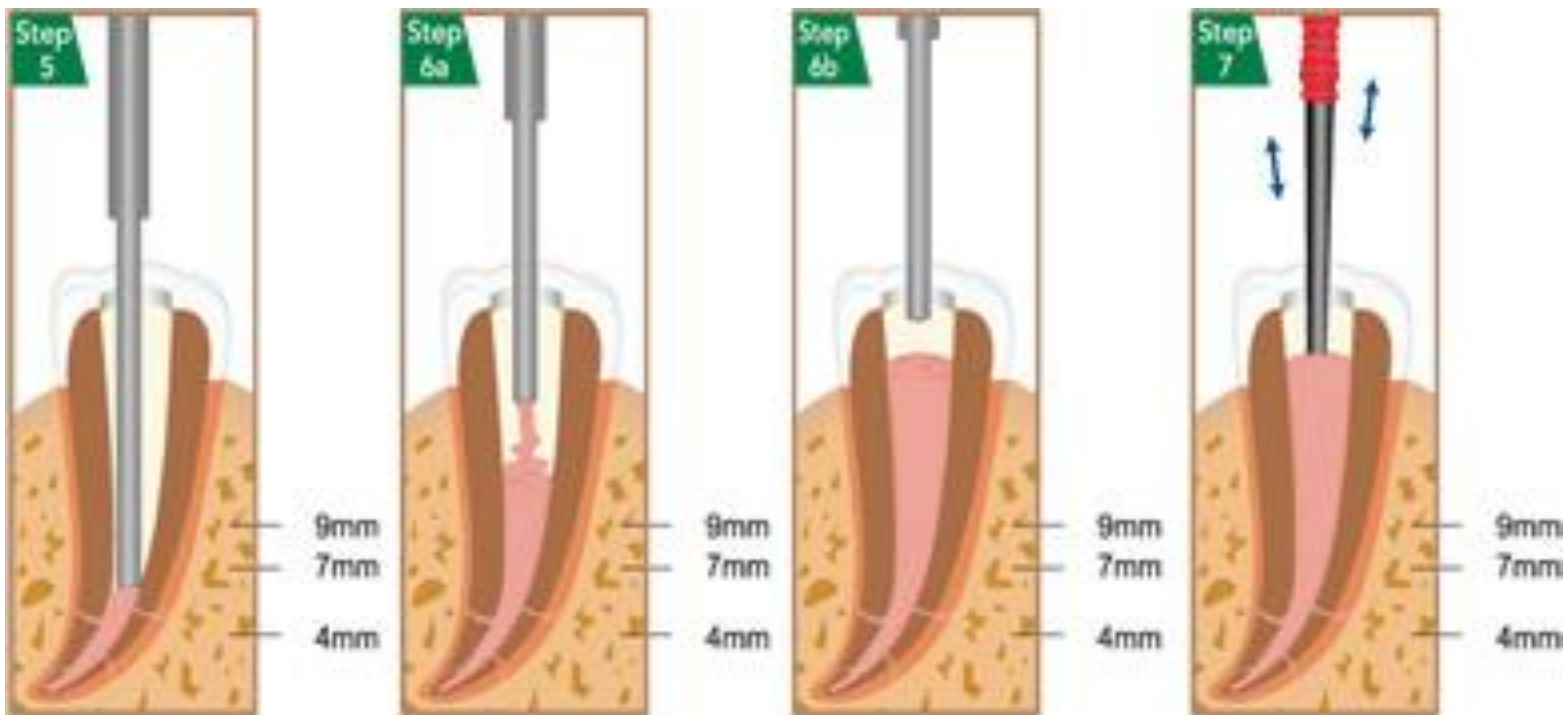
Involves filling the middle and coronal portions of the canal and can be accomplished using thermoplasticized gutta-percha devices, e.g. injection delivery systems.

- **Step 2**

Down Packing



Back-Packing



Extra-canal Heated Gutta-Percha:

A- Thermal Injection

The technique involves heating gutta-percha to a molten state and then injecting it into the root canal. The delivery device deposits the softened gutta-percha into the canal; vertical condensation is required to ensure adaptation of the gutta-percha to the canal wall.

In a commercially available delivery system (Obtura), the gutta-percha is heated to **160°C** and delivered through the needle tip at approximately **60°C**.



Carrier Based Gutta-percha

- **Thermafill:** is manufactured with a metal or plastic core to which the manufacturer applied a coating of gutta-percha. The carrier is flexible yet it provides rigidity for the overlying gutta-percha. The “obturators” are tapered and standardized so the carriers correspond to the size of instruments (MAF).
- After canal preparation, the canal is dried and lightly coated with sealer. The appropriate size obturator is heated in a special oven and firmly placed to working length. The carrier is then sectioned 1 to 2 mm above the orifice to the canal.

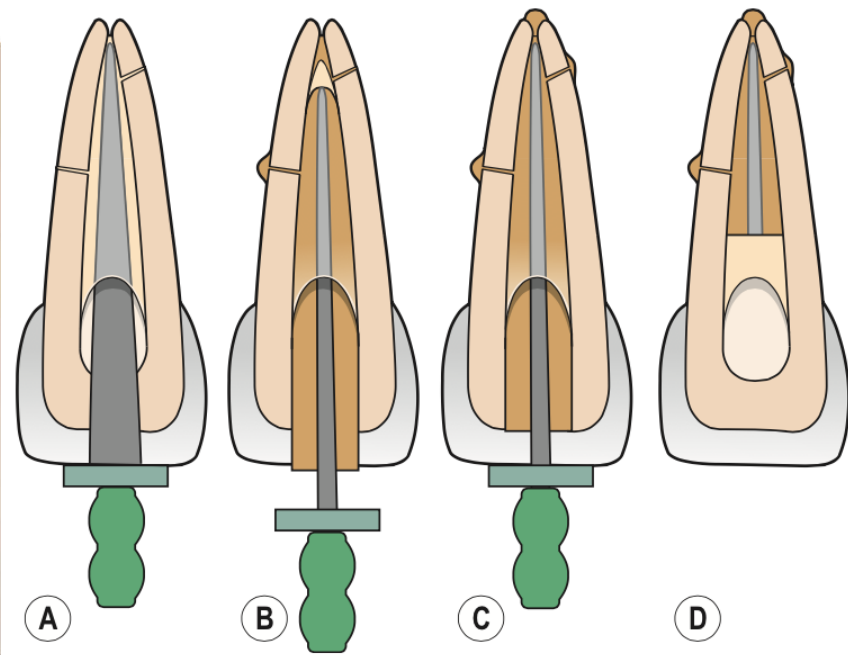


Figure 9.11 Precoated carrier obturation. (A) Verification of size with a blank carrier. (B) Selected carrier conditioned in oven, placed into canal and (C) inserted to length. (D) Excess removed (based on an original drawing by M Monteith).

