Operative Dentistry

Lecture (8) Dr. Raghad Noori

Pulp Irritants and inflammatory conditions of the pulp

Pulp

Dental pulp consists of 75% water and 25% organic material, this organic material composed of collagen fibers and ground organic substances. Function of organic material is supporting nervous, cellular and vascular components of the vital tooth. Vascularization of the pulp is through the apical foramen which present at the root apex. Pulp tissue is enclosed in a hard dentinal structure of the tooth.

Functions of the pulp

1- **Formative:** Generates primary, secondary, and tertiary dentin (dentinogenesis)
2- **Nutritive:** Provides the vascular supply and ground substance transfer medium for metabolic functions and maintenance of cells and organic matrix
3- **Sensory:** Transmits afferent pain sensation (nociception)
4- **Protective:** Coordinates inflammatory, antigenic, neurogenic, and dentinogenic responses to injury and noxious stimuli.
5- **homeostasis and clearance** of noxious and antigenic substances through the vascular and lymphatic systems and through defense cells, such as macrophages and leukocytes.

Pulp irritants

Response of the pulp to external irritation usually accompanied by changes in the dentin (such as sclerosis of dentin, reparative dentin…. etc.), therefore it has been agreed to consider the pulp and dentin as a one organ called the pulp-dentin complex which responds to tooth pathology through pulpal immune-inflammation defense systems and dentin repair/ formation.

Like other soft tissues, the pulp reacts to irritants with an inflammatory response. The pulp irritants can be classified according to the cause of irritant into:

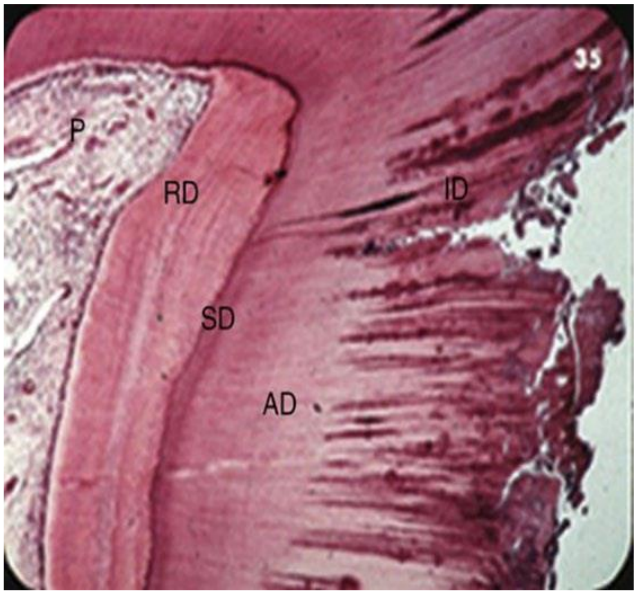
I- Bacterial
II- Physical
III- Irradiation
IV- Chemical

I- Bacterial irritant

1-Caries

Carious enamel and dentin contain numerous bacteria such as *Streptococcus mutans*, *Lactobacilli*, and *Actinomyces*. The population of microorganisms decreases to few or none in the deepest layers of carious dentin. Microorganisms in caries produce toxins that penetrate into the pulp through the dentinal tubules. As a result of the presence of microorganisms and their by-products in the primary dentin, cells in the pulp (mainly odontoblasts)) will respond to this irritation by releasing of cytokines and chemokines that initiate inflammatory reaction and beginning of infiltration of chronic inflammatory cells primarily. As the carious lesion progresses deeper into the dentin and bacterial irritation increase, the pulpal reaction increases as well and the concentration and character of the inflammatory cells' infiltrate change to a more sever type of inflammation.

The outward flow of fluid through the dentinal tubules during the primary irritation does not prevent bacteria or their toxins from reaching the pulp and increasing the pulpal inflammation. The extent of the pulpal inflammation beneath a carious lesion depends on the depth of bacterial invasion as well as the degree to which dentin permeability has been reduced by dentinal sclerosis and reparative dentin formation as well as the duration of the irritant.



2-Contamination of an exposed pulp by microorganisms

When actual pulpal invasions by bacteria and/or their toxins occur, severe inflammation occurs and is infiltrated locally by polymorphonuclear leukocytes to form an area of liquefaction necrosis at the site of exposure. Pulpal tissue may stay inflamed for long periods and may undergo necrosis eventually or become necrotic quickly which make the tooth non-vital requiring root canal treatment.



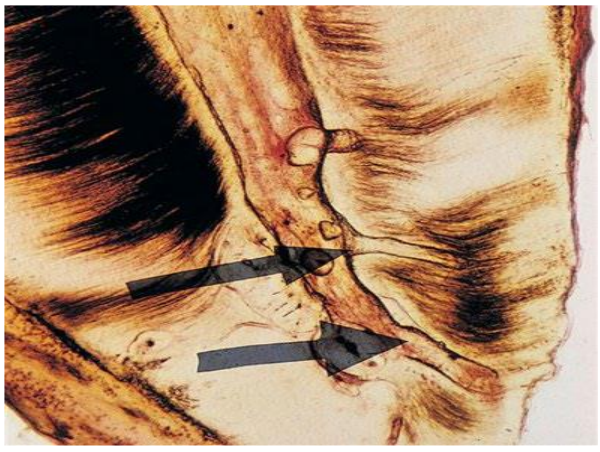
3-Periodontal disease

Periodontal disease may extend to the pulp through the accessory canals (arrows in the figure), the apical foramen, and open dentinal tubules. The inflammatory changes of the pulp occur when teeth have many accessory canals or when periodontal disease has progressed to the apex.

Many studies concluded that the accumulative effect of periodontal disease has a damaging effect on the pulp, as indicated by the presence of pulp calcification, inflammation, or resorption, but total pulp disintegration is certainty only when the apical foramen is infected.

Some studies found that periodontal disease does not have a direct inflammatory effect on the pulp; the initial effect of periodontal inflammation may be degenerative.

Root curettage can result in pulp devitalization. During curettage of a periodontal lesion that extends around the apex of a root, the pulp vessels may be severed and the pulp devitalized.

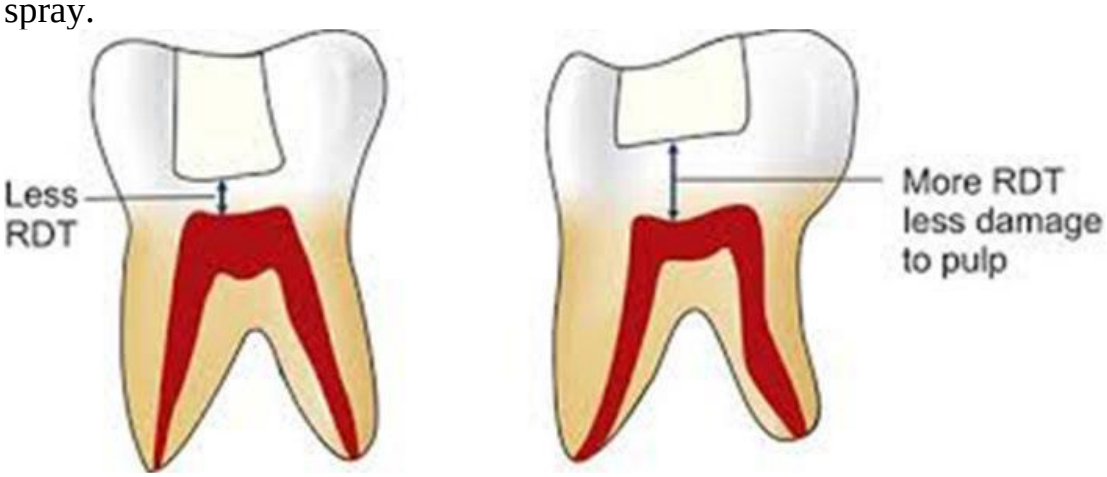


II-Physical irritant

1. Mechanical Irritation

A. Tooth preparation (caries removal or crown preparation)

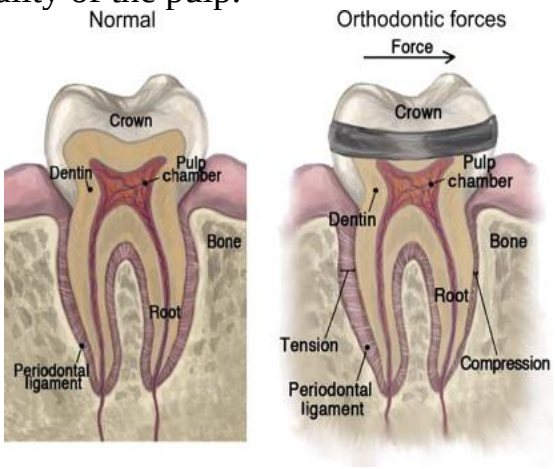
Pulp trauma results when the pulp is closely approached or the dentin is extensively removed during cavity preparation. Over-cutting during cavity preparation, whether a pulp is exposed or not is one of the greatest damages to the pulp. Not only the depth of cavity affects the pulp, but also the width of the cavity has the same importance. Pulpal damage is roughly proportional to the amount of tooth structure removed as well as to the depth of removal. The remaining dentin thickness (RDT) after deep cavity preparation play a significant role in health of the pulp. Also, it has been noted that there is an inverse relationship between remaining dentine thickness and the pulp injury. This is very important, especially in the case of the acid etching procedure, which is important to increase the longevity of restorations. In teeth with remaining dentine thickness less than 300 μm, acid etching may lead to severe irritation to odontoblasts and persistent inflammation in the pulp due to high permeability caused by acid etching. Also, operative procedures without water coolant cause more irritation than those performed under water spray.



B- Orthodontic movement

The force of movement during orthodontic treatment creates a disturbance in the circulation of the pulp that is similar to those found in the periodontally involved teeth.

If the force is beyond the limitation of the physiologic tolerance, blood vessels in the periodontal ligaments may rupture with a resultant hemorrhage which lead to loss of the nutritional supply to some pulp cells. If hemorrhage occur from larger vessels of the pulp the entire pulp become necrotic. In addition, sometimes orthodontic movement may initiate resorption of the root apex, usually without a change in the vitality of the pulp.



C- Tooth fracture (acute trauma)

Tooth fracture occurs by either direct trauma to the tooth or indirect trauma to the jaw, in addition severe occlusal pressure on a tooth with a large filling can cause fracture of the tooth. Fracture is usually associated with a bacterial invasion that follows the accident. Untreated bacterial invasions will decrease any possibility of sustained vitality of the pulp. If the fracture occurs through the root, this will lead to a disturbance in the vascular supply that often lead to the loss of vitality of the injured pulp.





D- Attrition

Attrition is a mechanical wear of the incisal or occlusal tooth structure as a result of functional or para-functional movements of the mandible (tooth grinding, or bruxism usually due to stress). Pulp inflammation or necrosis related to the incisal wear is seldom, pulp has the ability to lay down dentin, but when a sever worn of the tooth occurs (when attrition exceed the rate of deposition of reparative dentin), pulp exposure with an observable incisal opening could be seen. the pulp may be devitalized at an earlier time and the attrition finally reach the chamber. Sometime the tooth required to be crowned to overcome this problem.



E- Abrasion (chronic trauma)

Abrasion is defined as the loss of tooth structure by mechanical or frictional forces, these lesions are commonly caused by excessive tooth brushing, but repeated and excessive forces by other materials and appliance, such as dental floss, tooth picks, or removable appliances, may also produce such defects. These lesions can progress rapidly if they occur at the cement-enamel junction (CEJ) because the enamel is thin and the mechanical forces can wear the dentin and cementum away quickly. Also, it can be so severe that may invade the pulp space. The lesions commonly caused by horizontal brushing and appear as V-shaped notches on the labial surface.



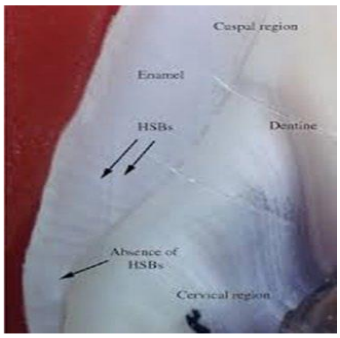
F- Abfraction

Abfraction is a type of noncarious cervical lesion characterized by the loss of tooth tissues with different clinical appearances.

The theory of abfraction sustains that tooth flexure in the cervical area is caused due to the occlusal compressive forces and tensile stresses, resulting in microfractures of the hydroxyapatite crystals of the enamel and dentin with further fatigue and deformation of the tooth structure. Abfraction lesions are also thought to be facilitated by the thin structure of the enamel and the low packing density of the Hunter-Shreger band at the cervical area.

It is important to determine and eliminate the cause (attrition, abrasion, or abfraction). If the tooth is hypersensitive, it could be relieved by desensitizing agents, topical fluoride, fluoride rinse, dentinal bonding agents, or restoration.





2- Thermal Irritation

It is commonly believed that the various dental procedures, such as tooth preparation, composite resin polymerization, finishing and polishing procedures can cause rise in the intrapulpal temperature. It was reported that an intrapulpal temperature increase of 5.5oC for 10 seconds can cause histological damage in the pulp tissues (irreversible pulpitis or even pulp necrosis).

Two new methods for tooth preparation are available:

-Laser

-Kinetic cavity preparation (air abrasion)

- ✓ **Laser device**: is a device which produces beams of very high intensity light. There are several types available based on the wavelengths. Laser used for soft and hard tissues, for soft tissue they can produce completely blood free incision followed by rapid healing. The use of a variety of lasers, including CO2, Er:YAG, and free electron lasers (FEL) on tooth structure has demonstrated minimal pulpal response, comparable to that of high-speed rotary instrumentation.

  The effect of laser depends on 1- power of beam 2- extent to which beam absorbed. When we used laser for cutting of enamel and dentin the

process would generate heat, which might affect the pulp so it should be used in pulsating manner (not continuously).

- ✓ **Air abrasion:** this generates heat, difficult for operator to determine the cutting progress within the cavity preparation (loss of tactile sensation).
  Air abrasive equipment is being used for stain removal and cleansing pit and fissure before sealing. Animal studies have shown that air abrasion cavity preparation is no more traumatic to the pulp than rotary instrumentation.

III-Irradiation irritant

The pulp of teeth is affected in-patient who is exposed to deep radiation therapy for malignant growth in head and neck region. In time odontoblasts cell and other cells will be necrotic and the salivary gland will be affected and resulting in decreasing of the salivary flow.

IV- Chemical irritant

1-Erosion

Erosion is being defined as the loss of tooth structure due to chemical action. Thus, erosion of facial or lingual tooth structure may create lesions. These lesions can be a prominent in patient with oral habits such as constant citrus ingestion, continues exposure to airborne acids, or gastrointestinal problems that produce repeated exposure of teeth to gastric acids. In these cases, the tooth lesions generally present a rounded, cupped-out defect initially confined to the enamel, if left untreated, the loss of tooth structure due to the chemical attack will accelerate once dentin has been reached, and deeper pattern of destruction will be seen.



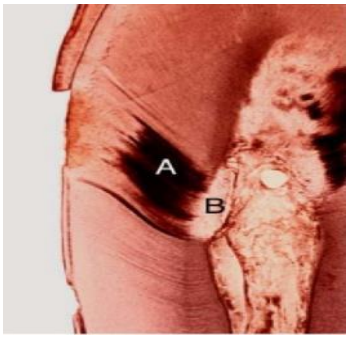
2-Chemical irritation of various restorative materials.

Defense Mechanisms of the dentino-Pulp complex

The dentine and the pulp should be considered as one vital organ (the dentine-pulp complex) because they are similar in development, structure and function. The reaction of the pulp and dentin to injury is mainly related to the activity of the odontoblast cells. A variety of reactions to the injury (caries, operative procedures and physiologic conditions such as attrition…etc.) could be seen **which include:**

1- Reduced dentin permeability: A reduction in the dentine permeability because of the diffusion of plasma proteins into the dentinal tubules. The coagulation of plasma proteins (fibrin) from pulpal blood vessels in the tubules beneath a recently cut cavity. Pathological precipitation of intratubular materials i.e. mineral deposits, collagen fibrils, proteoglycan linings and bacteria and the formation of a smear layer of dentine debris on its cut surface during cavity preparation. All these factors can contribute to intratubular occlusion that reduces permeability.

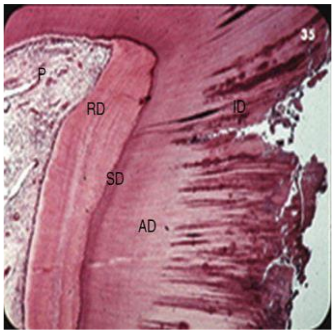
2- Dead Tracts: These are regions of empty tubules in primary dentin that result from degeneration of the odontoblastic process found under most carious cavities. At the proximal end of the tubules (near the pulp), the dead tract has been sealed off by a layer of impermeable calcified tissue protecting the pulp.



3- Sclerotic Dentin: It is a mineral deposition (sclerosis) in the dentinal tubules, because of aging or slowly progressing caries, which result in the gradual occlusion of dentinal tubules and decrease in dentine permeability, and reduce sensitivity to cold and rapid air movements. The peritubular dentin become wider and thicker as the tubules being filled and obliterated with calcifying minerals. Continued intratubular mineralization of dentin may result in complete obturation of the tubules. These areas of dentin are harder, denser, less sensitive, and more protective of the pulp against subsequent irritations.
Sclerosis resulting from aging is called **physiologic** dentin sclerosis, while sclerosis resulting from irritation is called **reactive** dentin sclerosis. Clinically,

sclerotic dentin is shiny, darkly colored, and feels hard to explorer tip, while the freshly cut normal dentin lacks the shiny reflective surface and allows some penetration by the sharp explorer tip (softer than sclerotic dentin). It is not uncommon to find sclerotic dentin under old restoration which usually shows a great amount of discoloration.
The apparent function of sclerotic dentin is to wall off the lesion by blocking (sealing) the tubules. The permeability of this type of dentin is greatly reduced in comparison to normal dentin because of the decrease in the tubule lumen diameter.
Sclerotic dentine appears translucent under the light microscope due to a reduction in light scattering through the affected tissue, therefore, it is referred to as the translucent or transparent dentine or zone.

4- Tertiary Dentin (Reparative D., Reactionary D., or irregular secondary D.): The dentine can be classified as primary, secondary and tertiary. Secondary dentine produced after completion of tooth formation because of physiological conditions subsequent to the formation of primary dentine during tooth formation.
Tertiary dentin (tertiary dentinogenesis) can be divided into reactionary dentin and reparative dentin: reactionary dentin is a tertiary dentin formed by surviving post-mitotic odontoblast cells as a reaction to stimuli such as caries, while reparative dentin is formed by odontoblast-like cells.
Reactionary dentine is the outcome of odontoblastic response to irritation caused by dental abrasion, attrition, cavity preparation, erosion or dental caries. Acidic by-products of the caries process will initiate a cascade reaction that stimulates odontoblasts to secret modified atubular dentinal matrix with altered biochemical properties.
In case of **reparative dentine**, caries advancing at a high rate with high acid production results in degeneration and death of the odontoblasts and their processes in the tubules, as well as mild inflammation of the pulp. In about 15 days, new odontoblasts are differentiated from stem cells of the pulp, and these replacement odontoblasts lay down the reparative dentin that is confined to the localized irritated area of the pulp cavity wall.
The structure of reparative dentin is more often irregular, atubular dentin depending on the severity of the stimulus. Reparative dentin is a defense reaction localized to the area of injury.
(**reactionary** dentin is the result of irritation of postmitotic odontoblasts, whereas **reparative** dentin is formed by odontoblast-like cells which differentiate from stem pulp cells after the cell death of primary odontoblasts).



5- Infected Dentin: It is very softened and contaminated with bacteria. It includes the superficial necrotic dentin tissue or zone. Clinically, necrotic dentin is wet mushy, easily removable mass. Histologically, this dentin is structureless with granular appearance and contain mass of bacteria. Remnant of distorted dentinal tubules filled with bacteria may be seen. It is easily removed by hand instrument without excessive force and flake off in layers parallel to the DEJ.
If the lesion is progressing slowly, there will be a zone of sclerotic dentin subjacent to the demineralized affected dentin.

6- Affected Dentin: It is the softened (demineralized) dentin because of the acidic products of the bacteria present in the superficial infected layer of carious lesion. Affected dentin still not invaded by bacteria and has intact tubules containing odontoblastic processes that have porous surface and contain crystalline material. This dentin is capable of remineralization, provided the pulp remains vital.
In slowly advancing lesion, we should remove all softened infected dentin down to the identified zone of affected dentin. In rapidly advancing lesion, there is little clinical evidence by texture and color change to indicate the limit of the infected dentin. Caries indicator could be helpful to distinguish between infected and affected dentin.

7- Inflammation of the pulp:

Inflammatory Conditions of the Pulp

In order to select the proper choice of treatment, the status of the pulp must be determined with accuracy. Sufficient irritation induces injury; incites inflammation. Histologically and physiologically, the inflammation is similar to that occurring in other connective tissues. However, the long term response of the pulp to severe irritation is different than in other tissues. In contrast to most soft tissues, the pulp has no room in which to swell. This inability to swell may well lead to increasing cell death in an ever widening area. It is generally the coronal pulp that is injured and it lacks collateral blood supply; extra nutrition and defenses cannot be marshaled quickly to the area. Another factor working to the detriment of the injured pulp is inflammation itself. Inflammation is a protective response. The immune response may destroy normal cells as well as foreign substances. Again, the pulp, with its compromised blood supply is unable to cope with this increasingly severe damage. The most severe reaction occurs at the time of actual pulp exposure.

- **In progressive order the status of the pulp in the response to caries are as follows:**
  1- Healthy pulp.
  2- Hyperemia.
  3- Acute pulpitis.
  4- Chronic partial pulpitis (without necrosis).
  5- Chronic partial pulpitis (with necrosis).
  6- Chronic total pulpitis with partial necrosis.
  7- Total necrosis of the pulp.
  8- Acute pulpitis superimposed on chronic pulpitis.

1-Healthy Pulp

Normal pulp, free of disease, or healthy, may show a wide variation in its histological structure according to its age and function. There are no inflammatory cells.

2- Hyperemia

Hyperemia is a physiologic term meaning an increase in blood flow through tissue. In histology, dilated and congested vessels were seen. Pulp could not be inflamed. About 41% of the carious teeth did have hyperemia, suggesting that frequently this may be an early sign of inflammation. Because the early vascular events of hyperemia precede or are an early component of inflammation, the removal of the precipitating cause of hyperemia should revert the microcirculation to its normal state.

3- Acute Pulpitis

This occurs as a sequent to various operative procedures including mechanical pulp exposure also following deep scaling and curettage. Always an acute reaction develops beneath the affected dentinal tubules. Acute infection could superimpose itself on an existing chronic inflammatory reaction e.g. the operative manipulation will induce an acute reaction on an already existing chronic reaction due to the previous restoration.

Histologic changes associated with inflammation: The odontoblast cells may be destroyed or ruptured by edema. Often evidenced by increased eosinophilia of the connective tissue. Marked dilatation of lymphatics and of blood vessels is accompanied by packing of erythrocytes and pavement of leukocytes along the vessel walls, and within capillaries. Infiltrate of leukocytes, is soon evident around the dilated vessels. This reaction lasts 3 days and it either disappears and resolves into a repair process or it changes into a chronic inflammatory reaction depending on the irritant "if it persists".

The clinical manifestation is mild pain during hot and/ or cold application, the pain remain as long as the stimulus remains. This condition is categorized as reversible pulpitis, as the pulp is able to reverse to healthy condition due to the absence of partial necrosis and if the stimulus is removed

4- Chronic Partial Pulpitis (without Necrosis)

This develops from deep dental caries, pulp exposure, operative procedures, deep periodontal lesion, and orthodontic tooth movement. The inflammation is confined to the coronal portion of the pulp. Histologically the chronic form usually dominated- small Lymphocytes, Monocytes, Macrophages, and plasma cells are abundant.

In children and young adults, the hyperplastic tissue reaction occurs because the young dental pulp has a rich blood supply and favorable immune response that is more resistant to bacterial infection. This is known as pulp polyp, also known as chronic hyperplastic, is an uncommon and specific type of inflammatory hyperplasia that is easy to bleed by touch. The pulp polyp is usually an incidental finding that occasionally mimics reactive and neoplastic diseases of the gingiva and adjacent periodontium, it's easy to be differentiated from gum polyp by follow the origin of each by using a probe. The pulp polyp is the result of both mechanical irritation and bacterial invasion into the pulp of a tooth that exhibits significant crown destruction due to trauma or caries. Usually, the entire dentinal roof is exposed with the crown of a carious tooth. The large exposure of pulpal tissue to the oral environment and bacterial invasion results in a chronic inflammatory response that stimulates granulation tissue reaction. Treatment of a pulp polyp includes: The more conservative vital pulpotomy treatment has been successful in selected cases when only the coronal pulp is affected (there's

no necrosis in the pulp). Or either root canal therapy or extraction of the tooth when there is necrosis in the pulp. In that case, this condition is categorized as irreversible pulpitis, as the pulp is not able to reverse to healthy condition due to the presence of partial necrosis. Or it can be categorized as reversible pulpitis if there was no partial necrosis in the pulp.

5- Chronic Partial Pulpitis with Partial Necrosis

It develops from the same irritant as above but with the persistence of such irritant. This causing area of liquefaction or coagulation necrosis with the inflammatory lesion has extended to deeper pulp tissue.

Painful symptoms may occur, these areas of liquefaction necrosis are called "pulp abscess" since they are surrounded by disintegrated polymorphonuclear leukocytes, collagen fibers and chronic inflammatory cells. The pain is spontaneous and is due to the pressure of the abscess on the nerve surrounding it. Sometimes the pain may last for ½ hr, and it doesn't disappear with analgesics.

Radiograph should be taken when the tooth in such condition. It could be exposed (the caries has gone to the pulp- chronic open pulpitis) or it could be closed (the effected caused by bacterial toxins or sever irritant that the pulp can't recover).

The available treatment for this type of pulpitis is either endodontic (root canal treatment) or tooth extraction. This condition is categorized as irreversible pulpitis, as the pulp is not able to reverse to healthy condition due to the presence of partial necrosis.

6- Chronic Total Pulpitis with Partial Necrosis

This develops with the extension of the inflammation to involve the entire pulp tissue (coronal and radicular) due to the persistence of the irritant and the development of liquefaction necrosis within the inflammatory area or coagulation necrosis. We get severe pain, sometimes lasting for many hours or when the patient sleeps this will increase the pressure inside the pulp and cause throbbing pain. The available treatment for this type of pulpitis is either endodontic (root canal treatment) or tooth extraction. This condition is categorized as irreversible pulpitis, as the pulp is not able to reverse to healthy condition due to the presence of partial necrosis.

7- Total Necrosis of the Pulp

Pulp in which the cells have died as a result of coagulation or liquefaction. histologically, coagulation cell means is still recognizable but the intracellular details have disappeared. While liquefaction necrosis, the entire outline of the cell has disappeared, and liquefied area. There is a dense zone of

polymorphonuclear leukocytes with cells of chronic inflammation. Treatment of choice is root canal treatment or tooth extraction.

8- Acute Pulpitis Superimposed on Chronic Pulpitis

There's severe pain especially at night till the abscess is formed then there's slight relief but not complete relief unless we do drainage through the tooth (access opening) or surgical incision with antibiotic cover, we should see the patient within few days and then treatment is continue by root canal treatment or extraction.