TEETH HYPERSENSITIVITY

Introduction:

Teeth hypersensitivity is an exaggerated response to a sensory stimulus that usually causes no response in normal healthy teeth. As a source of chronic irritation, teeth hypersensitivity affects eating, drinking, and breathing. Hypersensitive teeth are characterized by transient pain in response to evaporative, tactile, thermal, electrical or chemo-osmotic stimulation of exposed dentin in teeth where no other defects or pathology exist.

Aetiology of teeth hypersensitivity

I) Preoperative etiological factors:
   a) Bacterial
   b) Chemical - Osmotic
   c) Mechanical
   d) Thermal
   e) Idiopathic

II) Post operative (Iatrogenic):
   a) Factors related to cavity preparation
   b) Factors related to restorative phase and restorations
   c) Factors related to vital teeth bleaching.

I) Preoperative etiological factors:
   a) Bacterial:
      Dental caries produces different levels of teeth hypersensitivity that is mainly related to
      1- The depth of decay
      2- Dentin conductance
      3- Pain threshold of the patient himself.
1- **The depth of decay:**
A- Caries in enamel involve no or little hypersensitivity,
B- In dentin is characteristic to be short and sharp pain arising from exposed dentin in response to stimuli,
C- Dull ache pain if pulpal changes occur which might represent an irreversible pulpitis.

2- **Dentin conductance:**
Greater degree of sensitivity happens when dental caries passes the DEJ. as caries penetrates further into the tooth, sensitivity lessens until pulp becomes involved.
Deeper in dentin and near the pulp, the number of dentinal tubules is higher, the bigger the diameter of the dentinal tubules, the shorter their length, the higher the permeability of the dentinal fluids and consequently the higher the degree of hypersensitivity.

b) **Chemical-Osmotic:**
Erosion is defined as the dissolution of teeth by acids which are not of bacterial origin. When an acid or an osmotic agent like sugar adhere to the margins of leaky restoration or exposed dentin that will affect the flow of dentinal fluid and result in hypersensitivity. Erosion can be of Extrinsic or Intrinsic origin.
Extrinsic erosion results of exposure to extrinsic food, fluid or agents, such as citrus fruits, pickled food, fruit juice, carbonated drinks, wines, ciders, vitamin C, some mouth rinses with low PH and bleaching agents especially those delivered in a vacuum formed trays for In home applications.

Intrinsic erosion may result from gastric reflux as in Hiatus hernia, alcoholism, eating disorders like bulimia nervosa \(^{(60)}\). In case of intrinsic erosion the palatal aspect of the upper anterior teeth and the occlusal and buccal surfaces of the lower posterior teeth are primarily affected.

c) Mechanical

i) Attrition:
Is defined as wear of teeth at sites of direct contact between teeth. Attrition is associated with occlusal function and can be aggravated by habits or parafunctional activities which is known as bruxism.

ii) Bruxism:
The aetiology of bruxism is unknown but it could be associated with:

- Sleep disorders as obstructive sleep apnea and Snoring.
- Malocclusion
- High consumption of alcohol and heavy smoking
- Stress, digestive problems.
- Disorders as Huntington and parkinson’s diseases
- Drugs as: MDMA, cocaine

The bruxism results in hypersensitivity to heat and cold, fractured teeth and fillings, musculofacial pain and headache, stiffness and pain in the joints and earache.
iii) **Abrasion:**
It is defined as the wear of teeth caused by objects other than other teeth such as tooth brush/toothpaste abrasion, scaling and root planning and pipe smoking.

iv) **Abfraction:**
It is defined as the wear of teeth at the cervical portion as a result of occlusal loading that leads to cuspal flexure, this in turn results in compressive and tensile stresses at the cervical fulcrum area of the teeth with the resultant weakness and gradual loss of the cervical portion.

v) **Gingival recession:**
It may result from tooth brush abrasion, malocclusion, excessive brushing and flossing. Gingival recession results in exposure of cementum which less resistant to abrasion and acids than enamel, that consequently will lead to exposure of dentin and hypersensitivity.

vi) **Cracked tooth syndrome:**
It is defined as incomplete fracture of the vital teeth, can be involved in dentin only or extending to the pulp. Cracked tooth syndrome resulted in Teeth hypersensitivity with biting relieved with releasing the bite. It might involve severe spontaneous pain in case of pulp involvement, also can happen in sound teeth especially upper premolars, or most commonly in teeth that is restored with big restoration or direct gold.

vii) **Trauma**
Can involve fracture of enamel only with little or no sensitivity, enamel and dentin with moderate to sharp pain with stimuli typically evaporative, thermal, mechanical (tactile) or osmotic, pulp involvement with dull ache spontaneous pain, or fracture of the root which will result in tenderness to touch or percussion. The trauma
might result in no damage at all but tenderness to touch as a result of trauma to the periodontal ligament that can subside later.

d) **Thermal and idiopathic:**
Which can result in reversible hypersensitivity that subside by treating the exposed dentin and preventing the cause, and irreversible pulpal damage.

**II) Post operative (Iatrogenic):**

a) **Factors related to cavity preparation:**

i) **Type of cutting instruments:**
Rotary instruments produce more heat generation than the hand instruments. Dull instruments might require higher pressure for cutting which will result in more heat generation. Heat can destruct pulpal tissue, coagulate protoplasm and even burn dentin. Proper cooling is mandatory with all rotary instruments.

ii) **Instrumentation pressure:**
Cause heat generation and might cause actual aspiration of odontoblastic nuclei into the tubules.

iii) **Vibration:**
Cause a rebound response as a result of using eccentric burs, which can result in necrotizing effects on dentin. Rebound response appears microscopically as a limited area of necrosis at an area remote from the cut dentinal tubules.

iv) **Dentin Desiccation:**
Can result from heating of dentin during cutting, use of chemicals to sterile the cavity or use of air as a coolant for final cavity toilet.

v) **Actual cutting in dentin:**
Every square millimetre of dentin cut exposes 30,000 to 45,000 dentinal tubules with resultant fluid movement in each one of them stimulating nerve damage.
b) Factors related to restorative phase and restorations

i) Polymerization shrinkage

The polymerization shrinkage results in stresses at the composite/tooth interface resulting in microleakage, micro cracks or deformation of tooth structure. Microleakage can result in secondary caries formation and the consequent teeth hypersensitivity. Stresses are greatest in cavities with high ratio of C factor (ratio of bonded surfaces to unbonded surfaces), decreasing C factor will result in decreasing stresses from polymerization shrinkage.

ii) Undercured resin

Can result from a light source of inadequate intensity, or not close enough to the resin, or a light which is attenuated by passage through tooth structure or restoration. That results in a well cured surface covering incompletely cured layer, which will result in marginal fracture, open margin and chemical toxicity from the monomers or the bonding agent.

iii) Microleakage

Any restoration though exhibits clinical satisfactory adaptation, shows some leakage. The ingress of fluids and micro organisms can be the cause of dentinal hypersensitivity in addition to the fluid movements within the dentinal tubules.

iv) Inadequate liner and/or base

Any metallic restoration conducts thermal changes to the underlying dentin and pulp which can cause hypersensitivity specially in the first few days postoperatively. The pain is elicited after heat or cold application and
relieved with removal of the stimulus. The greater the temperature gradient, the more painful and lasting the stimulus. The dentin effectiveness as a thermal insulator depends on its thickness.

\[ v) \text{ Fractured restoration} \]
Exposes dentin, admits oral fluids and microbes which will cause recurrent caries and dentin hypersensitivity.

\[ vi) \text{ Cracked tooth} \]
Pain on biting and eating citrus fruits, this sharp pain will disappear when pressure is released. Commonly happens in teeth with large restoration and cast restoration without proper consideration for cusp protection.

\[ vii) \text{ Galvanism} \]
When two dissimilar metallic restorations brought into contact the current will pass between them and a galvanic stimuli will be generated. Hypersensitivity is usually felt in the tooth containing the restorative with the lower potential, i.e: Amalgam.

The degree of hypersensitivity will depend on some factors as:
The difference in the electrical potential between the dissimilar metals, the electrical resistance of dentin and soft tissues, presence of base and its thickness, the current intensity, the pulpal condition and the patient threshold.

\[ viii) \text{ Faulty occlusal and/or proximal relationship} \]
Occlusal contact if high will affect the periodontal ligament and lead to sensitivity with bite and/or mobility.
Proximal contact if tight will result in excessive pressure that might result in pain and interproximal bone resorption and damage of the supporting structure.
Proximal contact if light will result in food impaction interproximally and gingival inflammation, which if neglected will result in damage of the supporting structure.

**ix) Finishing Procedures**

Over finishing of restoration might result in heat generation which will lead to hypersensitivity.

Finishing of the restoration cervically might lead to scratch or total loss of the cemetum in this area.

Over carving of Amalgam restoration or uncovering the exposed dentin by cement in case of indirect restoration will result in teeth hypersensitivity.

**x) Barodontology**

It is the teeth hypersensitivity that occurs with reduced pressure, which occur during Aviation (in aeroplane). This could be due to expansion of air voids under a restoration, gases in a non-vital or due to expansion of air voids incorporated into the restoration during its application. Voids may be due to: inadequate condensation.

**xi) Gingival reaction to the restorative procedure and restoration**

- Retraction cord and chemical tissue packs used before the elastic impression taking, can result in soft tissue irritation.
- Improperly contoured or overhanging temporary dressing can result in gingival irritation.
- Overcontoured permanent restorations will contribute to poor gingival health by preventing through cleaning of the area.
- Undercontoured restorations will contribute to lack of protection of the gingival crevice and mechanical trauma of the free gingiva.
• Cementing media left in the crevice may cause gingival irritation that ranges from mild to severe inflammation.

xii) Pulpal reaction to the restorative procedure and restoration.

The pulpal reaction to a restorative procedure is difficult to determine, sometimes even with a conservative cavity preparation, the pulp experiences a degree of degeneration. Deep restoration may cause the pulp to devitalize many years later. A pulp exposure followed by a direct pulp capping might initiate an immediate hyperemia which can lead to root canal treatment. Generally if the hypersensitivity remains for several weeks the root canal treatment is recommended. A direct pulp capping is considered successful if the tooth is a symptomatic and gives a positive vitality test from 3-6 months later.

xiii) Local anesthesia

Sometimes soreness occurs at the site of injection as a result of haematoma and or infection. Beside discoloration the area is usually tender.

c) Factors related to vital teeth bleaching.

Both office and home bleaching procedures may induce discomfort in some patients. The principal complaints are mild tooth sensitivity to temperature changes and local oral mucosal irritation. Bleaching sensitivity is commonly associated with Carbamide peroxide vital teeth bleaching \(^\text{32}\) due to the by products of 10% carbamide peroxide readily pass through the enamel and dentin into the pulp in a matter of minutes. Sensitivity takes the form of reversible pulpitis caused from the dentin fluid flow and pulpal contact of the material. Sensitivity
can happen in any form of the bleaching agents, it depends mainly on the peroxide concentration and the patient threshold.

**Natural dentin desensitization**

Some natural processes can improve hypersensitivity overtime, even without treatment intervention. These include sclerosis of dentin, deposition of secondary and tertiary dentin, creation of a smear layer and calculus formation on the surface of the dentin. Sclerosis of dentin involves deposition of minerals within tubules that results in a thicker layer of peritubular dentin, this process eventually results in the tubule becoming smaller in diameter, making it less permeable and less able to transmit stimuli. Secondary dentin develops after the tooth root is formed, it is secreted slowly on the floor and roof of the pulp results in decrease in the size of the pulp chamber. Tertiary dentin or the reparative dentin is formed after the exposed dentin has been traumatized by a stimulus, this natural process decrease the permeability of dentin.
The smear layer is described as a combination of organic and inorganic debris, the smear layer plugs the dentinal tubule orifices with debris that consists of dental shavings, tissue debris, odontoblastic processes and microbial elements.

Calculus formation provides a protective coating to cover dentin from stimuli. Usually sensitivity occurs immediately after removal of heavy calculus which subside naturally within 2 weeks.

**Management of postoperative dentin Hypersensitivity**

**Diagnosis:** Which includes:

History taking, clinical examination and differential diagnosis, assessment of the etiological factors and management of the etiological factor after accurate diagnosis

a) **History:**

The patient is asked to describe the characteristics of pain as: onset, duration, stimuli, spontaneity, intensity and factors that relieve the pain.

After careful listening to the pt, the dentist need to gather the information related to Subjective information (symptoms) and the objective information (Signs) through clinical examination.

Subjective evaluation could be carried out by a *verbal rating scale*, which is a 4 scale grading pain as slight, moderate, severe and agonizing, by *Visual analogue scale* is a line of 10cm in length the extremes of the line represent the limits of pain experienced by the patient from no pain and till the most severe pain and the *McGill word descriptors* by answering a pre prepared questionnaire.

b) **Clinical and Radiographic examination:**

it is the *objective evaluation* of clinical signs using:
- Mechanical stimulus: Probe, scaler, constant pressure probe and Yeaple pressure stimulators.
- Chemical (osmotic) stimulus: Sodium chloride, sucrose, glucose and calcium chloride.
- Electric stimulus: Electrical pulp tester and dental pulp stethoscope.
- Evaporative test: air blast, Air jet stimulator
- Thermal stimulus: Ethyl chloride, ice stick and heat thermoelectric devices.

It involves clinical examination of teeth by percussion, probing, transillumination and other methods to detect the cause of the problem as caries, fractured restoration, cracked tooth or others.

c) Radiographic examination to examine the bone, root and surrounding structure to confirm the diagnosis.

d) Differential diagnosis:
Dentin hypersensitivity is diagnosed by exclusion (33), so all the other factors should be ruled out. From the diagnosis we can point the aetiological factor and treat the source of the problem.

**Techniques to Reduce Preoperative Hypersensitivity**

- Minimize preparation trauma
- Maximize dentin seal using dentin bonding agents
- Avoid:
  - inadequate isolation
  - overetching, dessicating demineralized dentin
  - inadequate priming, primer solvent not evaporated
  - inadequate adhesive placement, adhesive not photopolymerized
- Ensure temporary crown is well-fitting
- Ensure correct occlusal contacts
Techniques to Reduce Preoperative Hypersensitivity

I) Home care:

Desensitizing toothpastes / dentifrices

Potassium salts

Toothbrush and toothpaste application: Practitioners should educate patients on how to use dentifrices and monitor their toothbrushing techniques. Use of a soft or ultrasoft manual toothbrush with soft end rounded bristles lowers the risk of gingival recession and abrasion of exposed cementum and dentin. With powered toothbrush less pressure is required on the teeth, they require a light grasp to remove plaque. Dentifrices should be applied by toothbrushing. There is no evidence to suggest that finger application of the paste increases effectiveness. Many patients habitually rinse their mouths with water after toothbrushing. Rinsing with water may cause the active agent to be diluted and cleared from the mouth and, thus, reduce the efficacy of the caries-reducing effect of fluoride toothpastes.

Mouthwashes and chewing gums: Studies have found that mouthwashes containing potassium nitrate and sodium fluoride, potassium citrate or sodium fluoride or a mixture of fluorides can reduce Dentine hypersensitivity.

Dietary Modifications: Controlling the consumption of acidic food and drinks such as citrus fruits, wine, pickled foods and carbonated beverages. Avoiding brushing immediately after ingestion of acidic food, as it may accelerate the combined effect of abrasion and erosion, rinsing with water is recommended before brushing. Additional recommendations includes drinking something neutral or alkaline such as milk or water after consuming an acidic diet, Sipping acidic drinks through a straw and reducing the quality and frequency of acid intake.
Management of parafunctional habits:

Parafunctional habits can result in bruxism, teeth flexure or abfractions that lead to hypersensitivity, treating the cause as malocclusion, occlusal prematuraty or stress is recommended first, then preventing further tooth structure damage by conservative measures as night guard and fluoride. If home care fails to reduce Dentine hypersensitivity compared with baseline levels, the next level of treatment, an in-office method, should be started.

ii) In office treatment:

Fluoride: Fluorides such as sodium fluoride and stannous fluoride can reduce dentin sensitivity. Fluorides decrease the permeability of dentin in vitro, possibly by precipitation of insoluble calcium fluoride within the tubules.

Iontophoresis: This procedure uses electricity to enhance diffusion of ions into the tissues. Dental iontophoresis is used most often in conjunction with fluoride pastes or solutions and reportedly reduces Dentin hypersensitivity.

Potassium nitrate: Potassium nitrate, which usually is applied via a desensitizing toothpaste, also can reduce dentin sensitivity when applied topically in an aqueous solution or an adhesive gel. Potassium ions do reduce nerve excitability in animal models.

Oxalates: Oxalate products reduce dentin permeability and occlude tubules more consistently in laboratory studies than they do in clinical trials.

Calcium phosphates: Calcium phosphates may reduce dentin sensitivity effectively. Calcium phosphates occlude dentinal tubules and decrease dentin permeability.
**Orajel Tooth Desensitizer**: treats pain from sensitive teeth by blocking dentinal tubules preventing excitation of the tooth nerve.

**NovaMin**: is the brand name of a particulate bioactive glass that is used in dental care products. It consists of 45% SiO2, 24.5% Na2O, 24.5% CaO and 6% P2O5. It delivers an ionic form of calcium, phosphorus, silica, and sodium which are necessary for bone and tooth mineralization. NovaMin can be used as an effective, non-toxic alternative to fluoride.

**Casein Phosphopeptides**: It is a water-based topical cream, sugar-free with bioavailable calcium and phosphate, in the form of CPP-ACP (casein phosphopeptides-amorphous calcium phosphates). Recent studies reported that it provides extra teeth protection and neutralizes acids from acidogenic bacteria and from other external and internal acid sources.

**Adhesives and resins**: Because many topical desensitizing agents do not adhere to the dentin surface, their effects are temporary. Stronger and more adhesive materials offer improved and longer-lasting desensitization. In the 1970s,

**Lasers**: The effectiveness of lasers for treating dentine hypersensitivity varies from 5 to 100 percent, depending on the type of laser and the treatment parameters.