Dentinal hypersensitivity

Lecture by: Dr. Anitha. S

Dept of Periodontology

JSSDCH

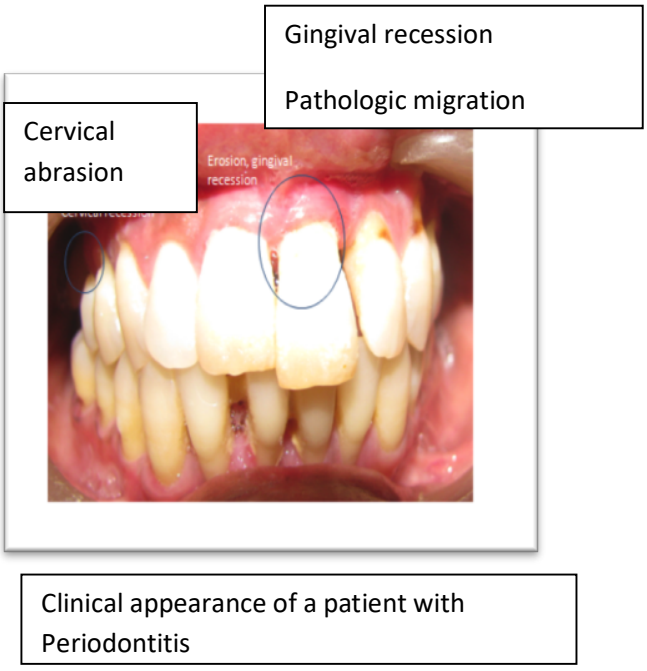
MYSORE

Definition:

Dentinal sensitivity/dentinal hypersensitivity are terms DS/DH) used interchangeably. It is defined as "an exaggerated response to application of a stimulus to exposed dentin, regardless of its location. DH is characterized by short, sharp pain arising from exposed dentin in response to stimuli, typically thermal, evaporative, tactile, osmotic, and chemical & which cannot be ascribed to any other dental defect / pathology."

Etiology:

Gingival recession	Cervical abrasion
erosions	Extensive instrumentation-subgingival scaling Root planing
Post surgical periodontitis	Cemento-enamel junction-developmental anomalies



Clinical appearance of a patient with Periodontitis

Origin of pulp & dentin are derived embryologically from the odontoblastic processes. These processes may extend through the entire thickness of dentin from pulp to DEJ. These processes may have extensions of odontoblasts, which are the major cells of pulp-dentin complex. The odontoblastic processes are actually the extensions of odontoblasts, which are major cells of pulp-dentin complex. The odontoblastic processes are surrounded by dentinal fluid inside the tubules. The dentinal fluid forms around 22% of total volume of dentin. It is an ultrafilterate of blood from the pulp via dentinal tubules & forms a communication medium between pulp via the odontoblastic layer & outer regions of the dentin

Pathogenesis

DH is supposed to develop in 2 phases:

Lesion localization:

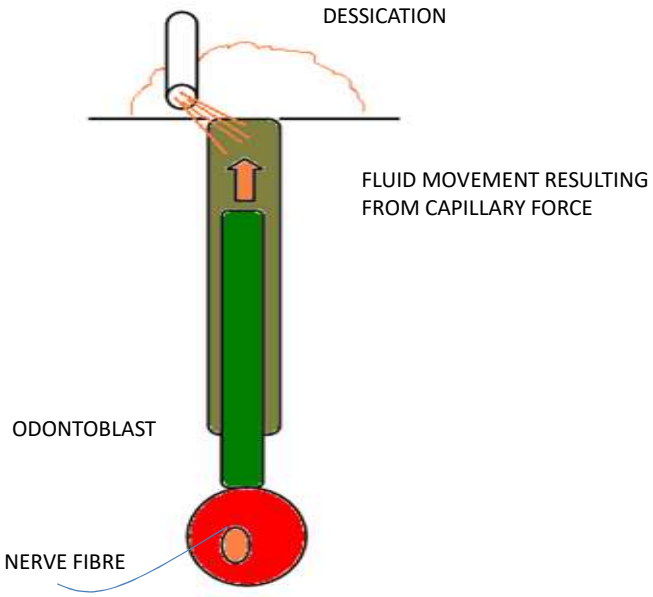
occurs by loss of protective layer covering on the dentin, thereby exposing it to the external environment.

Includes loss of enamel via attrition, abrasion, erosion, abfraction.

Recession can be due to tooth brush abrasion, pocket, post surgical, tooth preparation, excessive flossing, following tooth preparation.

Sensitization occurs after protective covering of smear layer is removed, leading to exposure & opening of dentinal tubules.

Mechanisms:



3 theories/ mechanisms

1. Direct innervation
2. Odontoblastic receptors
3. **Fluid movement/ hydrodynamic theory-MOST ACCEPTED**

Direct innervations theory:

- According to this theory the nerve endings penetrate dentin & extend to DEJ
- Lack of evidence
- Developmental studies have shown that plexus of Rashkow & intratubular nerves do not establish themselves until tooth has erupted but however newly erupted teeth are sensitive
- Pain inducers such as bradykinin fail to induce pain when applied to dentin
- Local anaesthetics do not prevent pain.

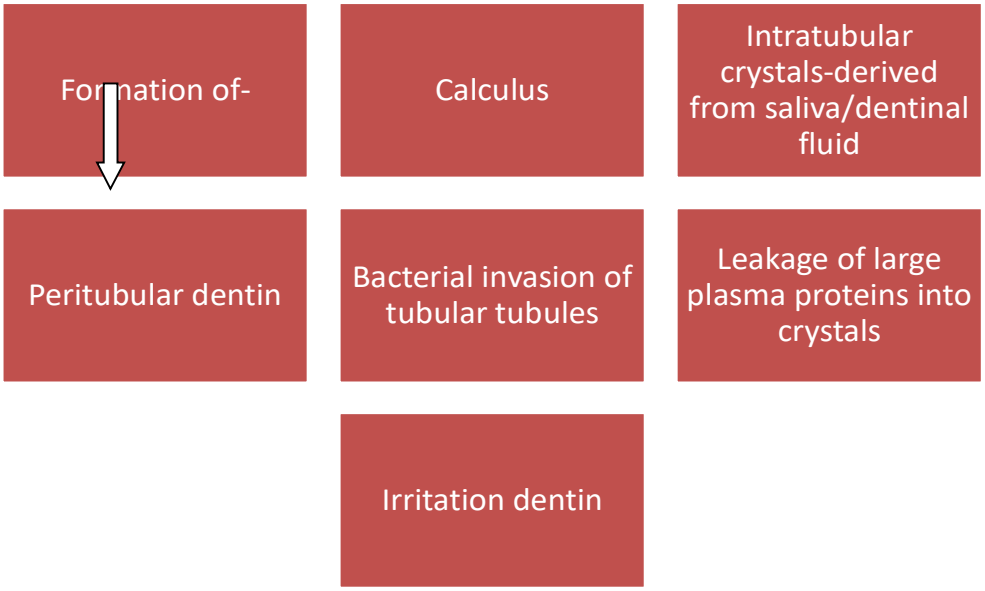
Odontoblastic receptor theory:

- This states that odontoblasts themselves & relay signal to a nerve terminal.
- Odontoblasts are actually matrix forming cells & hence they are not considered to be excitable cells & no synapses have been demonstrated between odontoblasts & nerve terminals.

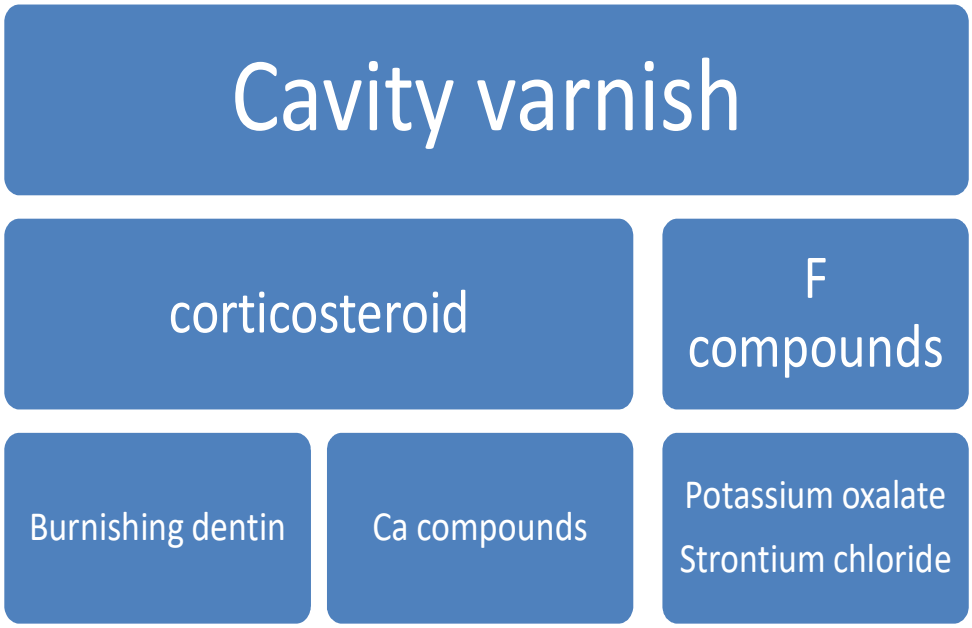
Fluid movement/ hydrodynamic theory:

- Proposed by Brannstrom 1961
- According to this theory, dentinal pain is due to hydrodynamic mechanism i.e., fluid force
- SEM analysis of hypersensitive dentin shows the presence of widely open dentinal tubules
- This theory is based on presence /movement of fluid inside dentinal tubules
- This centrifugal fluid movement in turn activates nerve endings at the end of dentinal tubules/ at pulp dentine complex.
- This is similar to activation fibers surrounding the hair by touching/ applying pressure.
- Response of pulpal nerves mainly δ delta), interdentinal afferent fibers, which depends upon the pressure applied i.e., intensity of stimuli
- It has been noted that stimuli which tend to move the fluid away from pulp- dentin complex produce greater pain. These stimuli include drying, evaporation & application of hypertonic chemical substances
- 75% of patients with DH complain of pain on application of cold stimuli
- Wider the tubules greater is the fluid movement

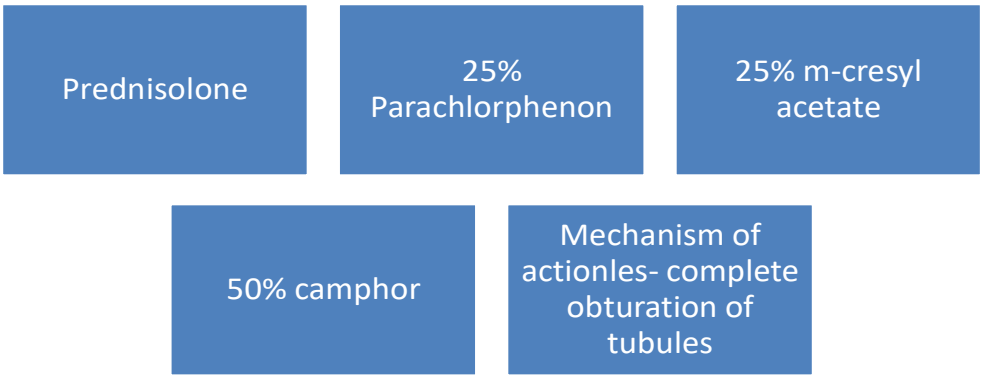
Physiological/pathological desensitization



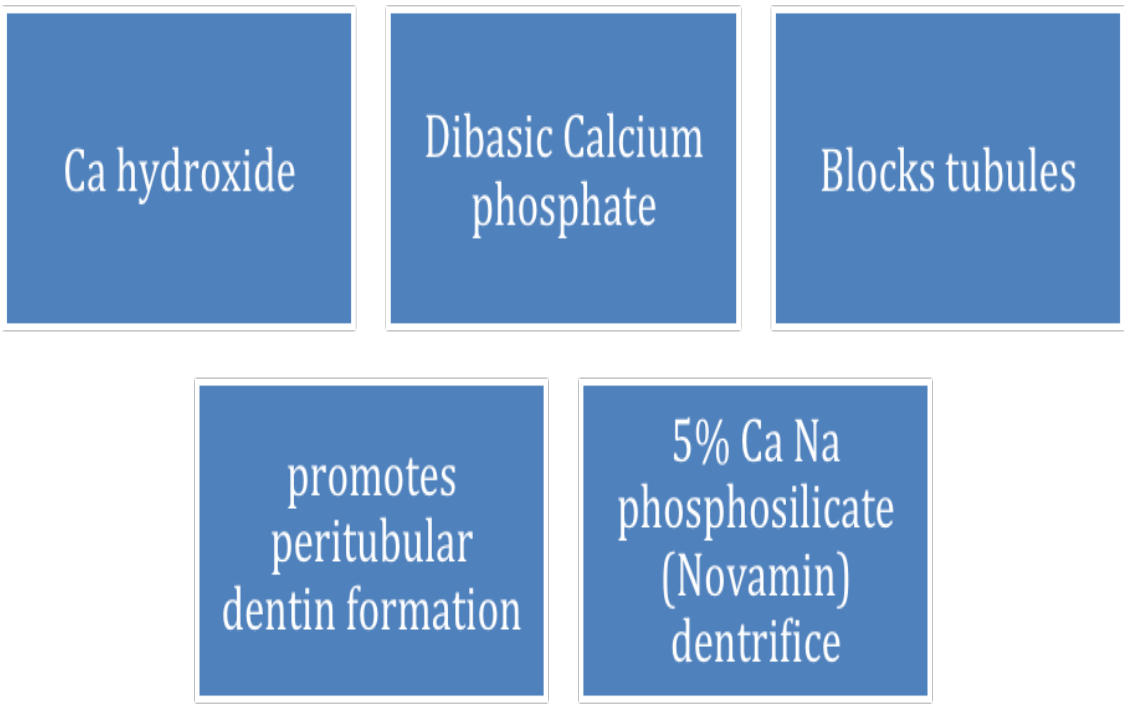
Hypersensitivity treatment



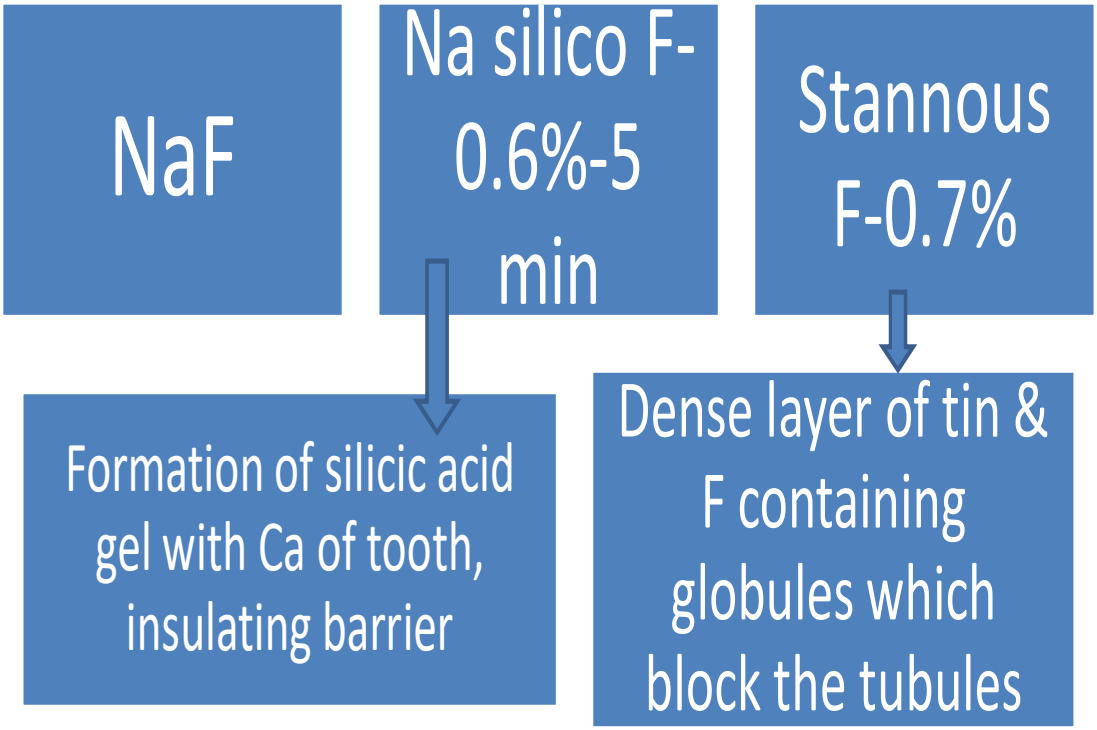
Corticosteroids



Calcium compounds



F compounds


NaF
Na silico F-0.6%-5 min
Stannous F-0.7%
Formation of silicic acid gel with Ca of tooth, insulating barrier
Dense layer of tin & F containing globules which block the tubules

F compounds-F iantophoresis

Fast & effective

Not effective in pulpal inflammation

Apical complications

Occlusal trauma

Exposed dentin

Mechanism of actions

Use of electric current to drive high concentration of ions into hard & soft tissues

Principle-similar to electromagnetic charges repel each other

Not effective in-
Pulpal inflammation
Apical complications
Occlusal trauma
Exposed cementum

Na F dissolved in solution

It ionizes with F becoming –vely charged

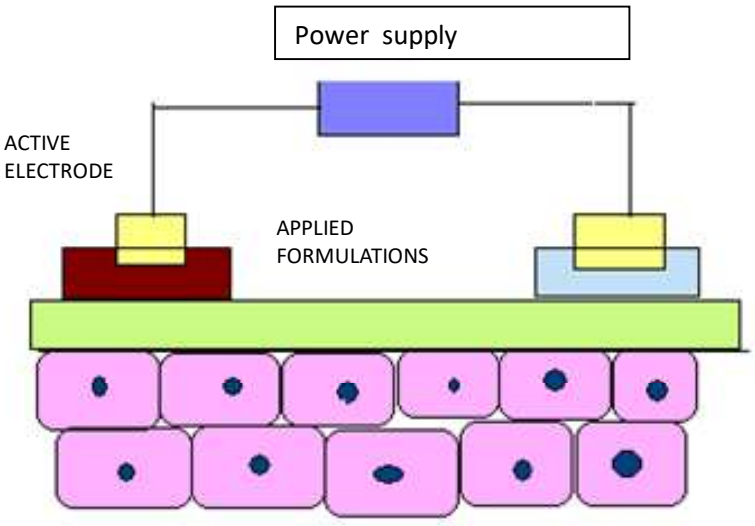
When placed on tooth with cotton tipped rod which is negatively charged electrode

An electric current is then passed through the tooth to other electrode which is held by the patient completing the circuit

F ions are pushed into dentinal tubules where they reach with ions in HA to form fluoroapatite

Which is insoluble compound that plugs the tubules

CONCEPT OF IANTOPHORESIS

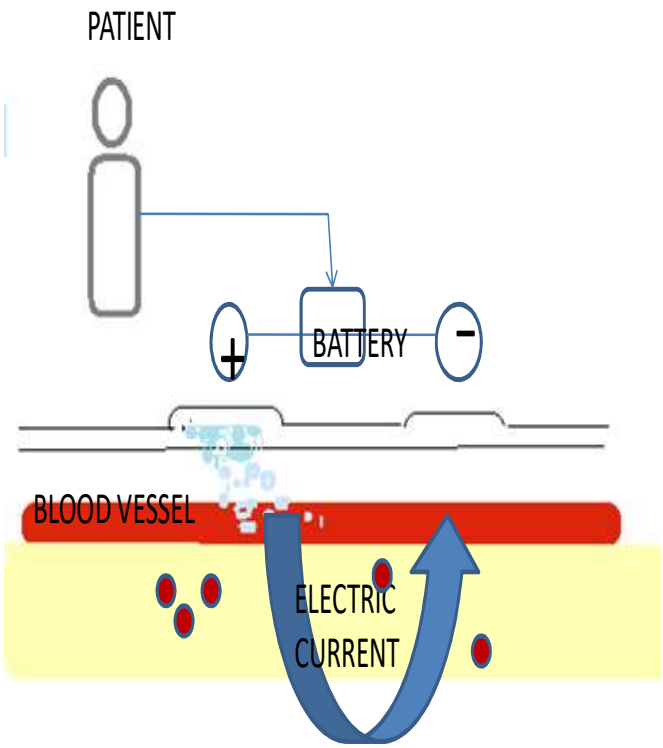


IANTOPHORESIS

COMPLETE KIT

ACTIVATED

CONCEPT OF IANTOPHORESIS



TREATMENT TECHNIQUE

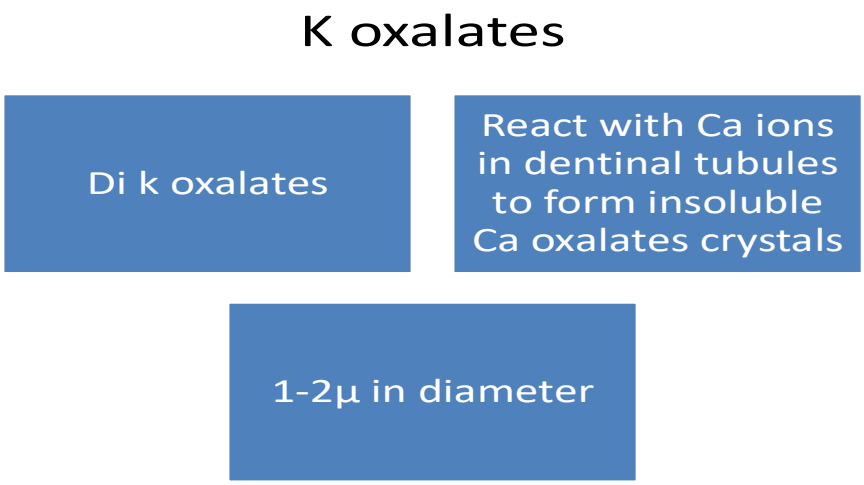
- REMOVE PLAQUE & PERFORM REGULAR PROPHYLAXIS
- ANY FILMS ON TOOTH INHIBIT F PENETRATION
- DRY THE TOOTH WITH GUAZE.
- USE COTTON ROLLS TO PREVENT MOISTURE CONTAMINATION OF TOOTH SURFACE
- IF TIP TOUCHES THE GINGIVA, EFFECTIVENESS OF TREATMENT GETS REDUCED

TREATMENT TECHNIQUE

- **PLACE A NEW APPLICATOR, SATURATE IT WITH NaF**
- **TO OPERATE A CIRCUIT NEEDS TO BE ESTABLISHED**
- **GREEN LIGHT ON THE DESENSITRON WILL INDICATE THAT IT IS SWITCHED ON**
- **A KNOB ON THE DESENSITRON CAN BE ADJUSTED FOR THE INTENSITY, WITH A MAX OF 0.5mA, A SETTING IDEAL FOR MOST OF THE SENSITIVE PATIENTS**
- **IF PATIENT C/O DISCOMFORT, THE INTENSITY CAN BE REDUCED**
- **IF PAIN IS NOT REDUCED\, THE TREATMENT CAN BE REPEATED 3 TIMES/WEEK**

CONTRAINDICATIONS-IANTOPHORESIS

- ALLERGIES TO Na & F
- PACE MAKERS/CARDIAC ARRYTHMIAS
- OPEN SORES


K oxalates
Di k oxalates
React with Ca ions in dentinal tubules to form insoluble Ca oxalates crystals
1-2µ in diameter

Strontium chloride

Strontium deposits

By exchange inCa ions in dentin resulting

Recrystallization in the formation of St apatite complex

Concept of Nerve hperpolarization

Hyperpolaraization intradental nerve by > extracellular k ions concentration

Therefore interfering with N transmission

Eg-k nitrate

RESTORATIVES-

Dental resins & adhesives

Dentin bonding agents

4-M6TA adhesive system

Strongest among others of the glycoprotein

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Adhesive materials

- Are considered dentinal materials & dentinal adhesives are considered tubules sealers
- Dentinal adhesives in the form of bonding agents & varnishes can be indicated
- Produce an immediate effect, but they are easily removed
- HEMA, benzalkonium chloride, glutaraldehyde, fluoride,

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Dentrifices

5% kNO3	Na mono flourophosphate
Arginine & calcium carbonate-pro-arg technology-1450 ppm F	Baking soda TOOTHPASTE delivering Ca, phosphate, F to the tooth surface (Ghassemi et al. J clin Dent 2009)

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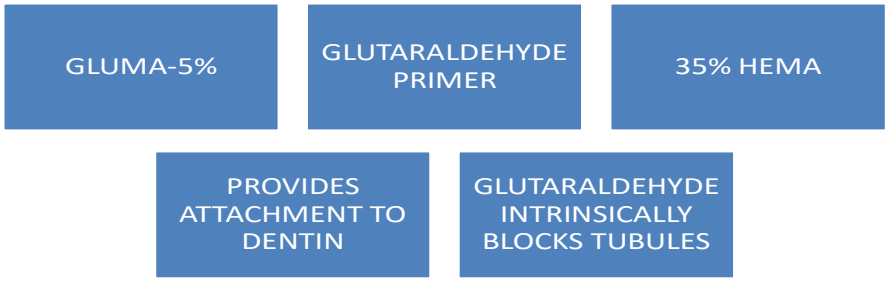
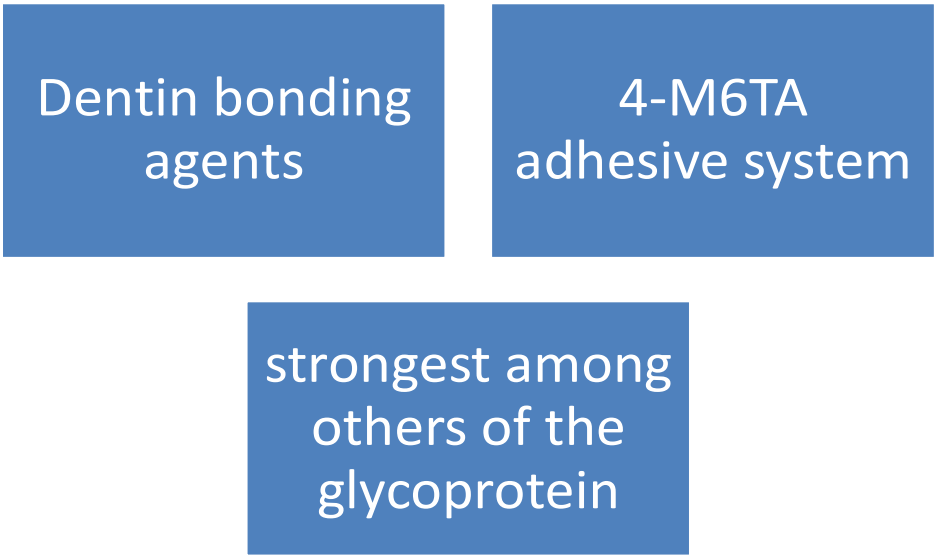
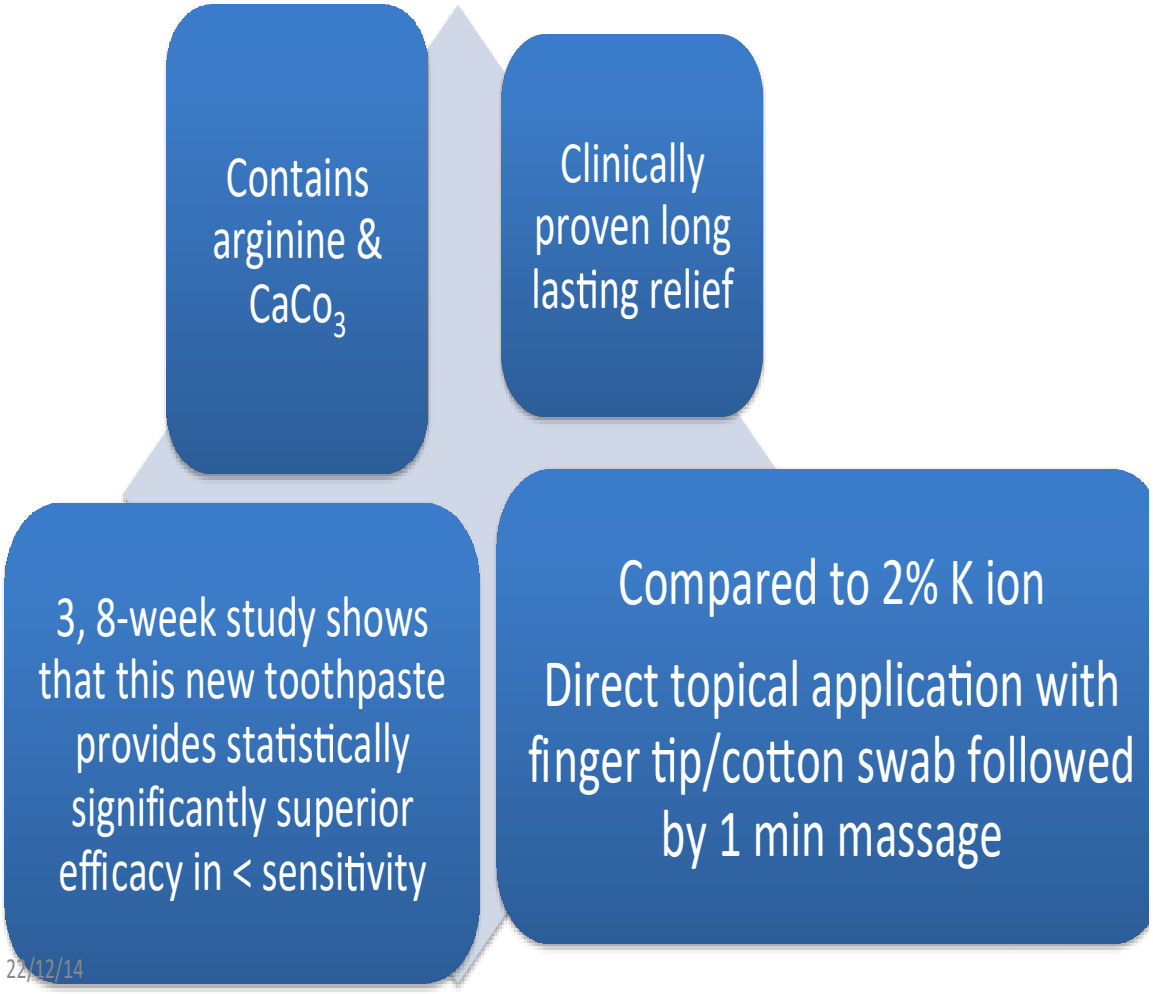
DBA

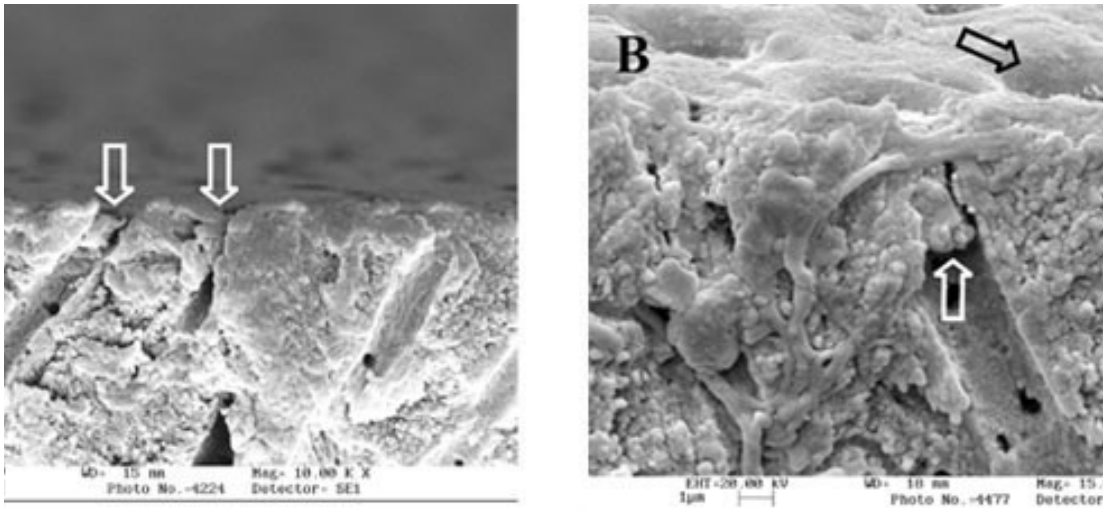


Dental resins & adhesives



Pro-argin technology tooth paste- (Cummins D- Am D J 2010)





micrograph of acidulated phosphate fluoride-treated dentin. Note the precipitates within
e (white arrow), and the funnel shape dentinal tubules opening (black arrows). (A: x 10,00

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Restorations:

- Glass ionomer Restorations
- Composite restorations
- Sandwich-technique: glass ionomer+ composite

- Er:YAG
- XeCl excimer 308 nm LASER

LASERS

QUICK & SIMPLE

< SENSITIVITY DRASTICALLY

ND:YAG

Without adverse reactions

Lased dentin harder than non-lased

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Surgical procedures

- GTR
- CORONALLY REPOSITIONED FLAPS
- SEMILUNAR FLAPS
- COMBINATIONS
- FREE GINGIVAL GRAFT
- CONNECTIVE TISSUE GRAFTS

MUCOGINGIVAL SURGERIES- CORONALLY ADVANCED FLAPS

SEMILUNAR FLAPS

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MUCOGINGIVAL SURGERY-

PRE-OP & POST-OP: GRADE 3 GINGIVAL RECESSION, POST-OP FGG

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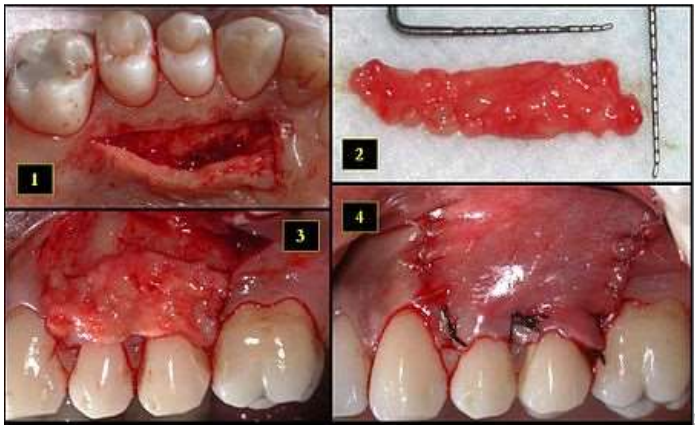
CONNECTIVE TISSUE GRAFT



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Summary of treatment protocols

1. Nerve desensitization
   - Potassium nitrate

2. Cover or plugging dentinal tubules
   a. Plugging dentinal tubules
      - Ions/salts
        - Aluminum
        - Ammonium hexafluorosilicate
        - Calcium hydroxide
        - Calcium carbonate
        - Calcium phosphate
        - Calcium silicate
        - Dibasic sodium citrate
        - Fluorosilicate
        - Potassium oxalate
        - Silicate
        - Sodium monofluorophosphate
        - Sodium fluoride
        - Sodium fluoride/stannous fluoride combination
        - Stannous fluoride
        - Strontium acetate with fluoride
        - Strontium chloride

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At home desensitizing therapy:

- Requirements for an ideal dentine desensitizing agent as:
- rapidly acting with long-term effects
- non-irritant to pulp
- painless and easy to apply
- should not stain the tooth
- therapy for management of DH is primarily aimed at occluding the dentinal tubules or making coagulates inside the tubules.
- Patients are often prescribed over-the-counter desensitizing agents
- These "at home" desensitizing agents include toothpastes, mouthwashes and chewing gums
- Majority of the toothpastes contain potassium salts (potassium nitrate, potassium chloride or potassium citrate), sodium fluoride, strontium chloride, dibasic sodium citrate, formaldehyde, sodium monofluorphosphate and stannous fluoride. Potassium salts act by diffusion along the dentinal tubules and decreasing the excitability of the intradental nerve fibers by blocking the axonic action.

- Various clinical studies have shown the efficacy of potassium salts in controlling the DH. It has been shown that toothpastes containing 5% potassium nitrate and 0.454% stannous significantly reduced the DH. Also, toothpastes containing potassium nitrate and fluorides have been shown to reduce post-bleaching sensitivity. The desensitizing toothpastes should be used with the help of a toothbrush with soft bristles. Patients should be advised to

use minimal amount of water to prevent the dilution of the active agent. Along with the desensitizing toothpastes, mouthwashes and chewing gums containing potassium nitrate, sodium fluoride or potassium citrate are also recommended. The results of "at-home" desensitizing therapy should be reviewed after every 3–4 weeks. If there is no relief in DH, "in-office" therapy should be initiated.

In-office desensitizing agents:

Theoretically, the in-office desensitizing therapy should provide an immediate relief from the symptoms of DH. The in-office desensitizing agents can be classified as the materials which undergo a setting reaction (glass ionomer cement, composites) and which do not undergo a setting reaction (varnishes, oxalates).

Summary

- Permeability & fluid movement in open, exposed dentinal tubules has provided a favored theory for stimulus transmission, through dentine. Occlusion of dentinal tubules has been identified as a potential method of reducing pain associated with sensitive teeth
- However these treatments can be expensive & their effects are transient. Current treatments work to occlude dentinal tubules

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Clinical situation-1

A male patient with cervical abrasions irt 34,35, periodontal abscess 27-with gingival enlargement irt same with H/O dentinal hypersensitivity-treatment options Include GI restorations for cervical abrasions, pocket therapy for 26, 27

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Clinical situation 2

Patient with class 3 gingival recession irt 23 with h/o gen. hypersensitivity

- **34- cervical caries**

Treatment options-

Mucogingival surgery irt 23

34-restorations

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