

MANAGING TOOTH HYPERSENSITIVITY – A FACTOID

The cause, diagnosis & treatment of tooth hypersensitivity has bewildered clinicians & patients for decades. Written accounts of fluid hydrostatic pressure in the dentinal tubules began, beginning with John Neill's 1838 publication, reemerging with Alfred Gysi in 1900 & its final acceptance by Martin Brännström's 1967 hydrodynamic concept of fluid flow through the dentinal tubule complex—others may have thought or discussed this fluid concept earlier, but their names remain unwritten. All other myths, misconceptions & theories of nerve conduction & odontoblast process nociception are now left to history.

Today, we can easily treat dentine sensitivity at chairside by the topical application of the unique chelation chemistry of **Super Seal**[®]. Those clinicians who still believe the outmoded ideas that acids kill the dental pulp are referred to published studies of Kozlov & Massler (1960), Kakahashi et al (1969), Brännström (1966) & Cox et al (1987), to understand that acids **do not** cause inflammation or death of the dental pulp.

Research studies have shown that **Super Seal**[®] is an ideal clinical agent to stop the dentine fluid flow by forming a **calcium oxalate (CX) nano-crystal complex** with the **calcium hydroxyapatite (CH)** in enamel lamella & defects as well as the dense peritubular dentine of vital human tooth substrates. Clinical studies have shown that topical application of **Super Seal**[®] is the simplest & most cost effective means—in terms of clinical-patient time & costs—to stop patient sensitivity following bleaching or drinking of acidic colas or juices.

SEM observations have confirmed that the treatment of human dentine with **Super Seal**[®] produces a uniform precipitate of **CX nano-crystals** in the enamel lamella spaces & dentinal tubules to completely block the enamel defects & block the fluid flow in the dentinal tubules to depths of 7-12 μ m.

The following tables identify various commercial agents by their chemistry & type—the most common are generic **VARNISHES** each containing a resin—mostly synthetic—in an organic solvent. **GLUTARALDEHYDE** solutions are mutagenic & cytotoxic to eukaryotic cells. Some **POTASSIUM AGENTS** precipitate nano-crystals that block fluid flow & others may alter nerve function. Many **ADHESIVE SYSTEMS** contain immunogenic hydroxy ethyl methacrylate (**HEMA**), Bis-phenol-A (**BP-A**) & other agents that are known to be injurious & toxic to any human cell with a nucleus (called eukaryotic).

HISTORICAL PERSPECTIVE OF DENTINE DESENSITIZERS

Following centuries of tooth extraction some academics & clinicians in Europe realized that some carious teeth could be restored with *stopping agents*—most were mixtures of gum resins with powders or zinc & oxychloride cement. France in the 1850's was a hotbed of clinicians who restored teeth with crude porcelain teeth—sugar & other sweets were the main cause of tooth decay.

As the medical-dental profession was evolving in Europe, some clinicians in the American colonies began to devise instruments for the removal of the decayed tooth debris. Some realized that if they were conservative in scooping out the soft caries, they could avoid a mechanical exposure of the vital pulp. Some clinicians (M.H. Webb, G.V. Black) realized that if foils of tin, gold & platinum were to remain in the cavity; they should provide “holding” retention into the cavity. In doing so, they removed normal vital dentine, which exposed the dentinal tubules, patients complained of postoperative hypersensitivity. At that era, there was no scientific agreement on the dentine sensitivity mechanism. A few academics prepared tooth sections & demonstrated the presence of nerves in the dental pulp—some entering into the dentine tubule complex.

This observation led clinicians to think about how to prevent the patient's postoperative hypersensitivity. If the outer cavo-surface margin was completely in enamel, they realized that zinc-phosphate cement would provide a good seal without pain. But, when the organic substrate of vital dentine was opened along the restoration interface—the patient felt pain.

In the late 1800's, different chemicals e.g. ammonium bromide, asbestos, potassium bromide, chloroform, cocaine, creosote, ether, formalin, menthol, phenol, silver nitrate, sodium bromide, trichloro-acetic acid, zinc chloride & essential oils such as clove or eugenol were placed on the cavity surface—some with success, but many others caused toxic reactions that later caused loss of the tooth.

In the early 1900's Europe, some clinicians realized that burnishing a paste of calcium hydroxide to the cavity interface provided some short-term relief, but long-term relief was not always achieved. Hence the placement of Zn-eugenol cement that created the era of intermediate restoration for temporary relief & varnishes soon followed as a routine cavity-lining agent. Hence the era of copal-varnish & ZnOE.

I have attempted to identify juried studies & to list many of the popular products, however, there are literally hundreds of commercial agents & many are sold on a regional basis. Consequently, the following table is not definitive, but only an attempt to describe most of the more popular systems on today's worldwide marketplace.

PRODUCT TYPE	NA – Not available	CHEMISTRY	NH – Not Harmful	HEALTH INFORMATION	DOCUMENTED STUDIES
VARNISHES	Generally apply multiple coats	Must air dry to form a 2 – 3 um thick layer		Most contain organic solvents that damage cells	Most are copies of the original copalite formula from the early 1900's & sold as a cavity liner
Copalite® Varnish	Cooley & Cooley Ltd	varnish ----- anhydrous ether----- chloroform-----		--NA --moderate hazard --carcinogenic	Manders CA et al, Am J Dent, 1990. Sepetcioglu F et al, J Oral Rehab, 1998
Duraphat®	Colgate Oral Pharmaceuticals	Colophonium----- ethanol----- sodium fluoride-----		--NA --moderate hazard --toxic if swallowed	Gaffar A, Cont, Dent Ed, 1999. Corona SA et al, J Oral Rehab, 2003
Fluoline-CP	PD Dental	varnish-----		MSDS is NOT readily available	Duran & Sengun, J Oral Rehab, 2004. Orchardson P, JADA, 2006
FluorilaQ™ Na FI varnish	Pascal Company Inc.	Sodium fluoride----- Ethanol-----		--toxic if swallowed --moderate hazard	No juried studies are readily available on the www / internet
Varnal® Cavity Varnish	Cetylite Industries	varnish-----		MSDS is NOT readily available	Chan K et al, J Prosth Dent, 1976. Newman S, J Prosth Dent 1984.
CavityShield® Varnish	3M-ESPE OMNI	5% sodium fluoride--- resin varnish -----		toxic if swallowed --NA	Autio-Gold JT & Barrett, Oper Dent, 2004.
Copal Cavity Varnish	Sultan HealthCare Inc.	varnish----- ethyl alcohol----- methylene chloride----		--NA --moderate hazard --chemical hazard	Royse MC et al, Pediat Dent, 1996.. Newman S, J Prosth Dent 1984.
Copaliner Varnish	H. J. Bosworth Company	resin mixture----- ethyl ether oxide-----		--NA --slight hazard	Morrow LA et al, Am J Dent, 2002
Vella™	Preventive Technologies Inc.	sodium fluoride ----- ethanol ----- rosin ----- xylytol -----		--toxic if swallowed --moderate hazard --NA --NA	No juried studies are readily available on the www / internet

Pure copal varnish was developed in the 1700's, originally a mixture of gum copal, turpentine & linseed oil spirits for furniture. Supposedly, the most pure form of gum copal was originally found & collected in the sands of Zanzibar of Africa—some believe that copal gum is left from the continual oozing up through deeper sands to harden by exposure to the sun & elements. Others think that copal is a product of a gum-resin of bushes & trees. The remaining gum that is now found & collected for sale as *amber* was formed many centuries ago, making it a curious natural phenomenon. It is the hardest natural gum & cannot be cut or dissolved with alcohol or turpentine. To be reduced to a fluid state, the gum is put in large kettles over low heat & when melted, turpentine & linseed oil are added. When cooled, the varnish is painted on any surface & the turpentine & the volatile oil evaporate, leaving a solid pure gum deposit that resists the atmosphere, but may not resist bacteria. Due to the rarity of *amber* resin & expensive cost, gum-copal has been replaced by tree resins, artificial alkyds & cheap synthetics that are often readily available but unconfirmed & unacceptable by strict *in-vivo* ISO / FDA biocompatibility testing.

Medical & Dental Material textbooks & academic lectures (Harris 1855, Dunham 1868, Webb 1883, Gorgas 1891, Shoemaker 1889, Long 1905, Buckley 1909) have reported that concoctions of zinc oxychloride cements & other agents were formulated through the late 1800's & placed as a cavity liner onto prepared enamel & dentin surfaces to supposedly seal the tubules against sensitivity before the permanent restoration, e.g. gold foil or amalgam was placed. Today, most commercially available "dental varnishes" are likely to be made from synthetic chemistries like poly-cyclo-hexanone rather than the original gum-copal of the 1900's.

I located many of the identified resin or varnish agents in searching available MSDS information sites. If you wish more detailed MSDS information, I suggest you personally contact the company for their published data they generally report is "on file" in their research records. When a dental varnish is placed on a moist tooth surface, the solvents dry, leaving a hard covering. Varnishes are **not** adhesive nor do they chemically bond to tooth tissues. With time the hard varnish often cracks, breaks down due to the microleakage of oral fluids, which causes deterioration & eventually permits bacterial microleakage leading to recurrent caries.

PRODUCT TYPE		CHEMISTRY	HEALTH INFORMATION	DOCUMENTED STUDIES
OXALATES				
Super Seal®	Phoenix Dental Inc.	Purified Water----- K oxalate salt-----	----NH ----NH	Galloway SD et al Arch Oral Biol, 1985. Sandoval VA et al, J Prosth Dent 1989, Farmer JB et al, JDR, 1990. Hafez A et al, JDR, 2000, Kolker et al, JDR, 2002. Huh JB, et al, J Dent, 2008,
Protect	Sunstar Butler	water ----- K ⁺ -oxalate -----	----NH ----NH	Greenhill JD et al JDR, 1981,
D/Sense Crystal	Centrix Inc.	binoxalate ----- nitric acid-----	--avoid eye contact --harmful to skin --rinse with water	No juried studies are readily available on the www / internet
BisBLOCK	Bisco Co	phosphoric acid----- oxalic acid-----	--denatures collagen --harmful to skin --rinse with water	Yiu C et al, J of Dent, 2006, Qiang HX et al, Chinese language, 2008, Turkkahraman, Angle Ortho, 2007
K-NITRATES				Most KNO ₃ agents are copies of the original 1974 Hodosh formula
K-nitrate	Richardson-Vicks Inc.	KNO ₃ -----	--NH	Hodosh A et al, J Prosth Dent, 1993.
K-nitrate generic	Block Drug Company, Inc	KNO ₃ -----	--NH	Hodosh, JADA. 1974
UltraEZ	Ultradent Co	KNO ₃ ----- 0.11% sodium fluoride	--NH --toxic if swallowed	Duran I et al, Eur J Dent, 2008, Turkkahraman, Angle Ortho, 2007
Oxa-gel Non-steroid	Laboratorios Beta, S.A. Art-dent	5% Sodic diclofenca- a phenylacetic acid- other agents NA	--non-steriodal anti inflammatory agent others - NA	De Assis et al, Braz Oral Res, 2006, Pereira A et al, Dent Mats, 2005, Limited information from company

In nature, **potassium (K⁺)** is a silvery-white metallic alkali discovered in 1807 by Sir Davy, which he derived from potash. **Potassium** rapidly oxidizes (tarnish) in air to a dark gray & is very reactive in water that generates heat. In nature, **potassium** occurs only as an ionic salt—as found in seawater. The **K-ion (K⁺)** is necessary for normal functioning of all living plant & animal cells. **K⁺** is important to prevent muscle contraction & its shortage in the body causes muscle weakness, respiratory paralysis & cardiac failure.

There are several types of **potassium** agents that are employed to treat dentine hypersensitivity. One mechanism is that (**K⁺ nitrate**) deactivates dental pulpal-nerve conduction. After neuroscientists learned the importance of **K** in brain & nerve function, they reported the membrane of millions of normal human eukaryotic cells is a gelatin-like intercellular substance that contains inorganic & organic matter called cytoplasm. The substance in the central nerve axon is called axoplasm. For the typical inactive neuron, the axoplasm has an overall negative charge based on the presence of sodium & **potassium ions**.

Normally there are more **potassium ions** inside the cell than on the outside, whereas there are more sodium ions outside the cell. Normally, the biological pump works to move sodium out of the cell & **potassium** into the cell. Basically, when increased amounts of **potassium ions** are flooded into a certain area such as a nerve cell or its axons in the dental pulp, as axons are not able to “fire” due to the increased **potassium-sodium** imbalance.

Potassium nitrate has been incorporated in many types of oral care agents. It has been reported to act by its ability to diffuse down the open dentinal tubules into the dental pulp to “shut-down” the patients nerve impulse & feeling of pain. For the most part Poulsen et al, Cochran Database Syst Rev, 2001, Kinshore A et al, J Endod, 2005 have reported **no strong evidence** to support the efficacy of KNO₃ agents for reducing dentine hypersensitivity. Since then, no further studies have documented that **potassium nitrate** forms crystals to block the tubules as reported by Brännström’s hydrodynamic theory.

The first oxalate was a neutral pH system of EDTA, DDS-1 & DDS-2, reported by Greenhill et al 1984. Many have reported it is not effective. **Super Seal®** is a **unique potassium oxalate** chelating agent that gained immediate clinical popularity in the 1990’s. Phoenix Dental was first to recognize that a low pH promoted the rapid chelation with CH to rapidly form acid resistant nano-crystals with the tooth’s enamel & dentine, proved as an economical desensitizer with many added benefits. In addition, it is not irritating to the dental pulp or cells of the oral cavity.

PRODUCT TYPE		CHEMISTRY	HEALTH INFORMATION	DOCUMENTED STUDIES
ALDEHYDES	Generally apply with brush or sponge & allow to remain on the tissue & then air dry to evaporate	Clinician must avoid placing on gingival & vital pulp tissues	glutaraldehydes are toxic to oral tissues	Few GTA products are sold as they are realized by most clinicians to cause cell death & gingival tissue loss
Gluma	Heraeus Kulzer Inc	5% glutaraldehyde--- 35% HEMA-----	--toxic hazard --pungent odor --irritant hazard	Kobler A et al, Am J Dent, 2008, De Assis et al, Braz Oral Res, 2006, Van Dijken et al, Dent Mats, 1989.
Dentine Desensitizer	Pulpdent Corporation	5% glutaraldehyde--- Water----- Fluoride-----	--toxic hazard --NH --irritant	No juried studies are readily available on the www / internet
Glu/Sense	Centrix Inc.	35% HEMA ----- 5% glutaraldehyde – gelled agent ? -----	---antigenic --mutagenic-toxic --no information	No juried studies are readily available on the www / internet
Systemp	Ivoclar/Vivadent	10% glutaraldehyde--- maleic acid----- 35% poly-ethy-glycol- water-----	--toxic hazard --moderate hazard --NH --NH	No juried studies are readily available on the www / internet

Glutaraldehyde is a toxic agent that is stable only under very controlled conditions. The clinician must read the attached MSDS attachments & be aware of all of the health hazards that are associated with the specific agent they are using. Heating of any glutaraldehyde product will alter the color to yellow & initiate a polymeric reaction or deactivation (efficacy). When the water is evaporated, the glutaraldehyde rapidly polymerizes to produce a residue that is flammable. When handling any type of glutaraldehyde solution, avoid bodily contact & inhalation of the vapor—it **should not** be dispensed to patients for at home use. Protective goggles or safety glasses with side shields & protective clothing should be worn. Eye baths & shower facilities must be provided to all involved in its use. Inhalation of glutaraldehyde vapor above 0.3 parts per million will cause irritation to the respiratory tract, sometimes causing asthmatic-like symptoms. If the odor of the glutaraldehyde persists in the workplace, then industrial hygiene personnel should be used to develop increased protective measures. In a clinic, there must be adequate ventilation of the area in case of a spill onto furniture. Concentrated solutions of glutaraldehyde-based agents such as Gluma should be handled at or near room temperature to avoid excessive vapor production. If glutaraldehyde product is warmed to increase its effectiveness, then active ventilation is required. Glutaraldehyde can easily cause irritation if it comes in contact with the skin or oral tissues (pulp – gingiva) & in concentrations of 10% or less it is easily absorbed by the skin in harmful amounts. Low concentrations of glutaraldehyde solutions e.g., 2-4% may cause minor irritation with local redness. At 0.02% the glutaraldehyde may cause chronic itching sensations. Protective measures must be used & maintained when placing any glutaraldehyde near the eyes & nose. Regarding hand protection with gloves, nitrile & butyl rubber gloves are suitable for use up to 50% glutaraldehyde while polyethylene gloves are acceptable with low concentrations less than 3.4%. Neoprene & polyvinyl chloride gloves are not acceptable or recommended for use with glutaraldehyde as they absorb & retain the agent. All gloves should be long enough to cover-up the arm to protect the skin. Any use around the face & eyes of 2% & greater will produce severe & irreversible eye injury. Solutions of 1% glutaraldehyde in water can cause moderate-to-severe eye irritation. Even the vapor contact of glutaraldehyde with the eyes may easily cause discomfort with excessive blinking & tearing.

Glutaraldehyde-based solution should not be discharged into lakes, streams, ponds, estuaries, oceans or any other water holding stations, unless approved by the US National Pollution Discharge Elimination Systems permit. In addition, glutaraldehyde cannot be discharged into any local municipal sewer system without prior notification to the local sewage treatment authority. When there are large quantities of glutaraldehyde to dispose of greater than 5%—high-temperature incineration is acceptable as it burns to CO₂ & H₂O.

Primarily, glutaraldehyde is used as a rapid fixative in the biological TEM as it quickly kills both normal human eukaryotic & bacterial cells by cross linking their proteins & is usually employed alone or mixed with formalin as the first of two fixative processes to stabilize specimens such as bacteria, plant material & human cells. Glutaraldehyde supposedly works on the tooth tissues by denaturing of all vital tooth fluid proteins. However, its placement on or near any soft tissues must be avoided due to toxicity. Many informed colleagues & patients have asked how the FDA can continue to allow glutaraldehyde & other toxic agents, e.g. formocresol, to remain in the chemistries of certain dental restorative products? An obvious answer: they were in clinical use long before the FDA defined biological *in vivo* cell culture tests that became required for all new chemicals. They survive in dental products under the FDA “grandfather clause” of the 1940’s. If glutaraldehyde was brought to the research bench today, **they would NOT pass the first ISO / FDA *in vivo* cell culture test.**

PRODUCT TYPE		CHEMISTRY	HEALTH INFORMATION	DOCUMENTED STUDIES
ADHESIVE SYSTEMS	one to multiple bottle systems	May etch with H₃PO₄ acids & then apply to tooth surface & air dry	Many contain polymers that damage tooth proteins	Became popular in the mid 1990's & generally necessitates more than one clinical step
Fuji GI-Lining cement	GC Corporation	distilled water----- polyacrylic acid----- Aluminosilicate glass	--NH --avoid eye contact --carcinogenicity	Kimura et al, Int J Paed Dent, 1994, Irie et al, Dent Mats, 1999.
Vitrebond glass ionomer liner	3M-ESPE	water----- 20%HEMA----- polycarboxylic acid-- aluminosilicate glass diphenyliodonium chloride benzenesulfonamide	--NH --skin antigen --eye irritant --avoid eye contact --avoid skin contact --skin irritatant	Chailertvanitkul et al, Int Endo J, 2007, Kimura et al, Int J Paed Dent, 2009,
MS-Coat	Sun Medical Co. Ltd.	water----- 4% MS copolymer----- oxalic acid-----	--NH --eye & skin irritant --skin irritant	Huh JB, et al, J Dent, 2008,
Adper™ Singlebond™ 1-XT	3M ESPE	10% silica filler----- polyacrylic acid----- polyitaconic acid-----	--NA --avoid eye contact --eye irritant	Marquezan et al, J Clin Ped Dent, 2008, Moura et al, J Appl Oral Sci, 2009.
Optibond Solo™	Kerr Co	ethyl alcohol----- alkyl dimethacrylate- barium & fumed silica sodium hexafluoroSi	--moderate hazard --NA --NA --NA	Sadek et al, J Adh Dent, 2005, Blaes, Pearls for the Practice, 2001, Eckert et al, Dent Mats, 2007.
Prodigy Unidose	Kerr Co	methylethacrylate ester mineral fillers----- activators & stabilizers	--skin irritant --NA --NA	Tyas, Dent Mats, 1998, Bowen, JDR, 2009, Nairobi, UN Env Prog, 2008,

The adhesive-resin-glass ionomer systems of today are represented by a wide number of commercial chemistries & consequently, they vary in pretreatment, drying, priming & bonding.

Many of the dental manufacturing companies have reformulated their adhesive & glass ionomer chemistries so as to market them as a cavity liner in which they also refer to them as an effective desensitizer. It is true that many of the adhesive systems will hybridize into the intertubular dentine—between the tubules—but not all will develop & form “tags” that may plug the tubule orifice. By the way, the real “bond strength” of any system resides in the intertubular hybrid layer & not in the resin tags (Gwinnett 1994).

The first generation of commercial adhesive systems the 1980's & were defined as total-etch systems. They were usually sold as a 2 or 3 bottle system. The smear layer was first etched with an inorganic acid etchant such as H₃PO₄ for 10-15 secs & then rinsed & air-dried. Next, a hydrophilic primer (bottle 2) that contains an acetone or alcohol & HEMA was liberally placed onto the enamel & dentine surfaces, generally for several coats & then the entire restorative interface was aggressively air dried to drive off (evaporate) the various solvents. The next agent (bottle 3) that was placed was generally some sort of bifunctional bonding agent. Please remember that the tooth interface is hydrophilic due to the open tubules allowing fluid to flow outward & so the cavity surface is hydrophobic (wet). The reason for this dual bifunctional interface is that the following composite system is hydrophilic & will not permit ideal bonding if moisture contaminates the bonding interface—reason to use a rubber dam! Originally, most systems were auto-cured but companies soon added agents that would permit light curing with UV light of certain wavelengths.

The following generation(s) of bonding systems—to speed up the clinical procedure—simply lowered the pH of the primer system to pH 2 to 4, so the smear layer was reconstituted to become incorporated into the primer layer, leaving all of the smear layer debris incorporated into the modified primer zone. The next bottle (2) generally contained a bonding interface that was dried & light cured. The definitive composite was placed & light cured.

As the next commercial advance, several companies were conceived to incorporate the primer & bonding resin into one-bottle system. To that end a number of similar commercial systems with varying chemistries that have come to the clinical marketplace. It must be mentioned that each system has its own manner of treatment & consequently, the clinician is responsible to read & fully understand the nature of the chemistries of the system they are using.

The clinician must understand the regional enamel & dentin substrate morphology & biochemistry. Many unidose systems behave differently in dissimilar depths of the dentine. **There is more organic dentine substrate towards the vital pulp & conversely, there is more mineral substrate towards the enamel-dentine junction.**

A BUCKET LIST OF SEVERAL DIFFERENT COMMERCIAL LINER SYSTEMS

PRODUCT TYPE	CHEMISTRY	HEALTH INFORMATION	DOCUMENTED STUDIES
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PRODUCT TYPE	CHEMISTRY	HEALTH INFORMATION	DOCUMENTED STUDIES
VARIOUS LINERS	Generally apply with a brush to create a 10 – 25µm film	Generally air dry to spread & evaporate the solvent	Most contain organic solvents dehydrates cells
Calciject	Centrix Inc.	water----- calcium hydroxide---	--NH --NH Niels et al, European Drug Index, 1997,
Tubulitec Cavity liner	Dental Therapeutics AB	ethyl acetate----- ethanol----- calcium hydroxide--- zinc oxide----- diiodidtymol-----	--NA --slight hazard --NA --NA --NA Johansson et al, Scand J Dent Res, 1993. Eriksen Eur J Oral Scis, 1973, Tveit et al, J Prosth Dent, 1985, Grajower et al, J Prosth Dent, 1976,
Hydroxyline	George Taub Products & Fusion Co	methyl ethyl ketone--- calcium hydroxide--- titanium dioxide-----	--moderate hazard --NA --NA Hydroxyline et al, J Proth Dent, 1976,
Dentinbloc	Colgate Oral Pharmaceuticals	sodium fluoride ----- stannous fluoride----- phosphoric acid- ---- Hydrogen fluoride ----	--toxic if swallowed --toxic if swallowed --skin irritant --moderate hazard Schwartz et al, J Prosth Dent, 1998, Garcca-Godoy et al, Am J Dent, 1998, Thrash et al, 1992.
Hurriseal®	Beutlich LP Pharmaceuticals	HEMA----- benzalkonium chloride-- sodium fluoride----- hyamine -----	--skin antigen --NA --toxic if swallowed --NA Kolker et al, JDR, 2002, Qahtani et al, Oper Dent, 2003,
Pulpdent	Pulpdent Corp.	water----- calcium hydroxide--- PMGDM----- Mg NTG-GMA-----	--NH --NA --moderate hazard --NA Taylor et al, Endod Dent Traumat, 1989,
Embrace™ Sealant	Pulpdent Corp.	acrylic resin----- silica amorphous ---- sodium fluoride-----	--NA --slight hazard --toxic if swallowed Kane et al, Am J Dent, 2009, Castro & Galvao, J Clin Ped Dent, 2004,
HYPO-CAL	Ellman International Inc.	lime water----- calcium hydroxide--- calcium hydrate----- slaked lime-----	--NA --low allergin --NA --NA Ida et al, J Endo, 1989, Mackie et al, Endod Dent Traumat, 1994, Morrier et al, J Endodont, 2003.
Chembar		polystyrene resin---- chloroform----- calcium hydroxide--- zinc oxide----- fluoride----- di-thymol-12-----	-- --denatures proteins --NH --NH --toxic if swallowed --NA No juried studies are readily available on the www / internet
Vivasens	Ivoclar / Vivadent	alcohol----- hydroxypropylcellulose polyacrylic acid----- potassium fluoride--- polyethylene-glycol dimethacrylate----	--slight hazard --NA --avoid eye contact --do not swallow --NA Hajizadeh et al, J Contemp Dent Pract,, Betke et al, Oper Dent, , 2006,
Calm-It	Dentsply Int	glutaraldehyde----- HEMA-----	--toxic hazard --skin antigen No juried studies are readily available on the www / internet
Micro Prime™-B or Micro Prime™ G	Danville Materials	HEMA----- benzalaconium hloride glutaraldehyde----- sodium fluoride----- water-----	--skin antigen --0.2 ppm --0.2ppm --toxic if swallowed --NH Malkoc et al, Europ J Ortho, 2005, Duran et al, Europ J Dent, 2008, Sengun et al, Oper Dent, 2005.

Ca(OH)₂ paste is perhaps the oldest recorded clinical treatment for sensitivity. In 1967, I learned that a number of Prosthetic Departments of a few European dental schools had been burnishing a Ca(OH)₂ paste onto the dentine of prepared crown preps for over 5-decades. That procedure provided some short-term post treatment relief, but eventually washed-out due to microleakage of oral fluids that dissolved the Ca(OH)₂.

REFERENCES

- Abel I. Study of hypersensitive teeth & a new therapeutic aid. *Oral Surg.*1958;11: 491-495.
- Absi E et al. Dentine hypersensitivity: uptake of toothpaste into dentine and effects of brushing, washing and dietary acids-SEM *in vitro* study. *J Oral Rehab.*1995;22:175.
- Addy M. Dentine hypersensitivity: A review. *J Clin Perio.*1983;10:351-363.
- Addy M et al. Dentine hypersensitivity. II Effects produced by the uptake *in vitro* of toothpaste into dentin. *J Oral Rehab.*1989;16:35-48.
- Addy M. Clinical aspects of dentine hypersensitivity. *Proc Finn Dent Soc.*1992; 88:407-412.
- Anderson D. The sensitivity of the human dentine. *JDR.*1958; 37:669-677.
- Arkovy J. Investigations on the development of dentine. *Transac Odont Soc.*1876;7:103-129.
- Berggren et al. The rate of flow in dentinal tubules due to capillary attraction. *JDR.*1965;44:408-415.
- Beers W, Sensitive dentine. *Brit J Dent Sci.*1893;36:604-606.
- Bennett R, Monheim's Local Anesthesia & Pain Control in Dental Practice. St. Louis: The C.V. Mosby Co, 1984:5-6.
- Blunden H et al. The effects of compounds used clinically in the management of dentin hypersensitivity on some physical properties in dentin. *IADR Brit Div.*1981;Abs:130.
- Bolden T et al. The desensitizing effect of Na-mono fl-phos dentifrice. *Perio.*1968;6:112-114.
- Brännström M. Dentin & Pulp in Restorative Dentistry, Wolfe Med pubs Ltd.1982: 9-19.
- Brännström M. Sensitivity of dentine. *Oral Surg.*1966;21:517-526.
- Brännström M et al. Transmission & control of dentinal pain: resin impregnation for the desensitization of dentin. *JADA.*1979;99:612-618.
- Brännström M. The hydrodynamics of the dental tubule & the pulp fluid. *Caries Res.*1967;1:310-317.
- Brännström M. The hydrodynamic theory of dental pain: sensation in preparations, caries, & the dental-crack syndrome. 1986. *J Endo.*12:453-457.
- Brännström M. Reducing the risk of sensitivity & pulpal complications after placement of crowns & fixed partial dentures. *Quint Int.*1996; 27:673-678.
- Brännström M et al. Occlusion of dentinal tubules under superficial attrited dentine. *Swed Dent J.*1980;4:87-91.
- Cagidiaco M et al. Dentin contamination protection after mechanical preparation for veneering. *Am J Dent* 1996;9:57-60.
- Calamia J et al. Effect of AmalgamBond (4META) on cervical sensitivity. *J Dent Res.*1992;71:32.
- Chabanski M et al. Aetiology, prevalence & clinical features of cervical dentine sensitivity. *J Oral Rehab.*1997;24:15-19.

- Charvat J et al. Titanium-tetra-fluoride for the treatment hyper sensitivity of dentine. Swed Dent J.1995;19: 41-46.
- Chesters R et al. Use of multiple sensitivity measurements & logit statistical analysis to assist the effectiveness of a K-citrate containing dentifrice in reducing dental hypersensitivity. J Clin Perio.1992;19:256-261.
- Christensen G. Marginal fit of gold inlay castings. J Prost Dent.1966;16:297-305.
- Cobb D. Effect of HEMA-containing dentin desensitizers on shear bond strength of a resin cement. Am J Dent.1997;10: 62-65.
- Coleton S. Sensitivity &laser treatment. JADA.1998;129:1200-1204.
- Copeland J. Simplified remedy for tooth sensitivity. North Western Univ. Dent. J.1985;64:13.
- Cox C et al Reparative dentin: factors affecting its deposition. Quint Int.1992;23:257-269.
- Cox C. Etiology & treatment of root hypersensitivity. Am J Dent. 1994;7:266-269.
- Cox C. Making it stick: without making it hurt. Continuing Dental Ed. Univ of Wash. Dental Sch. May 8th 1998.
- Cox C. Biocompatibility of dental materials in the absence of bacterial infection. Oper Dent.1987;12:146-152.
- Davidson D et al. The Gluma bonding system: a clinical evaluation of its various components for the treatment of hypersensitive root dentin. J Can Dent Assoc.1997;63:38-41.
- Dayton R et al. Treatment of hypersensitive root surfaces with dental adhesive materials. J Perio.1974;45:873-878.
- Ehrlich J et al. Residual fluoride concentrations SEM examination of root surfaces of human teeth after topical application of fluoride *in vivo*. JDR.1975;54:897-900.
- Felton D et al. Long-term effect of crown placement on pulp vitality. JDR.1989;68:1009.
- Felton D et al. Inhibition of bacterial growth under composite restorations following Gluma pretreatment. JDR.1989;68: 491-495.
- Felton D et al. Evaluation of the desensitizing effect of Gluma dentin bond on teeth prepared for complete-coverage restorations. Int J Prost.1991;4:292-298.
- Felton D et al. Effect of *in-vivo* crown margin discrepancies on periodontal health. JDR.1989;68:1008.
- Fearnhead R. The neurohistology of human dentin. Proc Royal Soc Med. Sect of Odont.1961;54:31-38.
- Fischer C et al. Prevalence & distribution of cervical dentine hypersensitivity in a population in Rio de Janeiro. Braz Jr Dent.1992;20:272-276.
- Fish E. The circulation of lymph in dentin & enamel. JADA.1927;14:804-817.
- Fish E. Dead tracts in dentine. Proc Royal Soc Med. Sect of Odont.1928;22:7-16.
- Fish E. The reaction of the dental pulp to peripheral injury of the dentine. Proc Royal Soc.1931;108:96-208.

- Flynn J et al. The incidence of hypersensitive teeth in the west of Scotland. *J Dent.* 1985;13:230-236.
- Furseth R. A study of experimental exposed & fluoride treated dental cementum in pigs. *Acta Odont Scan.*1970; 28: 833-850.
- Fusayama T. A simple pain free adhesive restorative system by minimal reduction & total etching. Tokyo. Ishiyaku Euro Am Inc.1993;128.
- Garberoglio R et al. SEM investigation of human dentinal tubules. *Arch Oral Biol.*1976;26:893-897.
- Garret J. Root plaining: A perspective. *J Perio.*1977;48:155-163.
- Gelskey S et al. The effectiveness of the Nd:YAG laser in the treatment of dental hypersensitivity. *J Canad Dent Assoc.*1993;59:377-386.
- Graf H et al. Morbidity, prevalence & intraoral distribution of hypersensitive teeth. *JDR.*1977;53:162.
- Green B et al. Ca(OH)₂ & K-nitrate as desensitizing agents for hypersensitive root surfaces. *J Perio.*1977;48:667-672.
- Greenhill J et al. The effects of desensitizing agents on the hydraulic conductance of human dentin *in vitro*. *JDR.*1981;60:686-698.
- Grossman H. The treatment of hypersensitive dentin. *JADA.*1935;22:592-602.
- Guerra A. *Modern Anesthesia in Dentistry.* Phil. PA: The Franklin Institute Press.1977;163-165.
- Gunji T. Morphological research on the sensitivity of dentin. *Arch. Histol Jpn.*1982;45:45-67.
- Gysi A. An attempt to explain the sensitiveness of dentin. *Brit J Dent Sci.*1900;43:865-868.
- Hanazawa K. A study of the minute structure of dentin, especially of the relation between the dentinal tubules & fibrils. *Dent Cosmos.*1917;59:125-148,271-300.
- Hernandez et al. Clinical study evaluating the desensitizing effects & duration of 2-commercially available dentifrices. *J Perio.*1972;43:367-372.
- Hiatt W et al. Root preparation. I. Obturation of dentinal tubules in treatment of root hypersensitivity. *J Perio.*1972;43:373-380.
- Hirvonen T et al. The excitability of dog pulp nerves in relation to the condition of dentine surface. *J Endo.*1984;7:294-298.
- Hodge H et al. The adsorption of strontium at 40° by enamel, dentine, bone & hydroxyapatite as shown by the radioactive isotope. *J Bio Chem.*1946;163:1-6.
- Hodosh M. A superior desensitizer Potassium nitrate. *JADA.*1974; 88:831-832.
- Holland G. The incidence of dentinal tubules containing more than one process in the cuspal dentine of cat canine teeth. *Anat. Rec.*1981;200:437-442.
- Hotz P et al. Epidemiology of dental erosion & toothbrush abrasion. *JDR.*1988;67:388.
- Hoyt W et al. Use of NaFI for desensitizing dentine. *JADA.* 1943;30:1372-1376.

- Huber C. The innervation of the tooth pulp. *Dent Cosmos*.1898;40:797-811.
- Hume W. An analysis of the release & the diffusion through dentin of eugenol from ZnOE. *JDR*.1984;63:881-884.
- Jain P et al. Dentin desensitizing agents: SEM & X-ray microanalysis assessment. *Am JD*. 1997;1:21-26.
- Jensen A. Hypersensitivity controlled by iontophoresis. Double blind clinical investigation.1964;68:216-225.
- Johanson G et al. Crown retention with use of resin sealer on prepared dentin. *JDR* 996;1918:257.
- Jensen M et al. comparative study of 2-clinical techniques for the treatment of root surface sensitivity. *Gen Dent*.1987;35:128.
- Johanson G et al. Crown retention with use of a 5% glutaraldehyde sealer on prepared dentin. *J Prost Dent*.1998;79:671-676.
- Johnson G et al. Outward fluid flow in dentin under a physiologic pressure gradient: experiment *in vitro*. *Oral Surg*.1973;35:238-248.
- Kanouse M et al. The effectiveness of Na-mono fluorophosphate in dentifrice on dental hypersensitivity. *J Perio*.1969;40:38-30.
- Kerns D et al. Effectiveness of NaFI on tooth hypersensitivity with & without iontophoresis. *J Perio*.1989;60:386-389.
- Kerns D et al., Dentinal tubule occlusion & root hypersensitivity. *J Perio*.1991;62:421.
- Kramer I. The response of the human pulp to self polymerizing acrylic restorations. *Brit Dent J*.1952;93:311-315.
- Kramer I. Relationship between dentine sensitivity & movements in the contents of the dentinal tubules. *Brit Dent J*.1955;98:391.
- Kun L. Biophysical study of the modifications in dental tissue induced by the topical application of strontium. *Schweiz monats schrift fur zahn*. 1976;86:611-676.
- Lan W et al. Sealing of human dentinal tubules by Nd-YAG laser. *J Clin Laser Med Surg*.
- Lefkowitz W et al. Desensitization of dentin by bioelectric induction of secondary dentin. *J Prost Dent*.1963;13:940-949.
- Land M et al. disturbance of the dental smear layer by acidic haemostatic agents. *J Prost Dent*.1994;72:4-7.
- Lennart E. Sensory nerve recordings in human teeth. *J Endo*.1986; 12(10):462-464.
- Lilja J. Sensory differences between crown & root dentin in human teeth. *Acta Odont Scand*.1980;38:285-291.
- Lilja J et al. Dentin sensitivity, odontoblasts & nerves under desiccated or infected experimental cavities. *Swed Dent J*.1982;6:93-103.
- Ling T et al. An investigation of potential desensitizing agents in the dentine disc model: a SEM study. *J Oral Rehab*.1997;24:191-203.

- Litch W. Am system of Dent. 1886;Vol.I: 832-840.
- Liu H et al. Sealing depth of Nd:YAG laser on human dentinal tubules. J Endo.1997;23:691-693.
- Lukomsky E. Fluorine therapy for exposed dentine & alveolar atrophy. JDR.1941;20:649-659.
- Marshall G et al. The dentin substrate: structure & properties related to bonding. J Dent. 1997;25:441-458.
- Mausner et al. Effect of two dentinal desensitizing agents on retention of complete cast coping using 4-cements. J Prost Dent 1996;75:129-134.
- McBride M et al. The effectiveness of NaFI iontophoresis in patients with sensitive teeth. Quint int.1991;22:637-640.
- Mummery H. The nerve supply of the dentine. Procds Royal Soc Med. Sect of Odont.1924;17:35-47.
- Mummery H. The innervation of dentin. Dent Cosmos.1916;58:258-269.
- Mummery H. Correspondence on the innervation of dentin. Dent Cosmos.1916;58:803-804.
- Murthy K et al. A comparative evaluation of topical application & iontophoresis of NaFI for desensitization of hypersensitive dentin. Oral Surg.1973;36:448-458.
- Myers T. Laser in dentistry. JADA.1991;122: 47-50.
- Nakabayashi N et al. Hybridization of dental hard tissues. Tokyo. Quint Pub Co., Ltd.1998.100-102.
- Nordenvall K et al. Desensitization of dentin by resin impregnation a clinical & LM investigation. J Dent Child.1984;July-August: 274-276.
- Markowitz K et al. Hypersensitive teeth: experimental studies of dentinal desensitizing agents. Dent Clin N Am.1990;34:491-501.
- McCormack K et al. Review article the enigma of K-ion in the management of dentin hypersensitivity: is nitric oxide the elusive second messenger. Pain.1996;68:5-11.
- Miller J et al. Use of water-free stannous fluoride concentrating gel in the control of dental hypersensitivity. J Perio. 1969;40:490-491.
- Mukai Y et al. Effects of FI-Lanthanum treatment for dentin hypersensitivity *in vitro*. Dentin / Pulp Complex. Tokyo. Quint Pub Co., Ltd.1996:248-250.
- Mumford J et al. Pain & protopathic sensibility. A review with particular reference to teeth. Pain.97;2:223-243.
- Nagata T et al. Clinical evaluation of a K-nitrate dentifrice for the treatment of dentinal hypersensitivity. J Clin Perio.1994;21:217-221.
- Närhi M et al. Conduction velocities of single pulp nerve fiber units in the cat. Acta. Phys Scand.1982;116:209-213.
- Närhi M. The characteristics of intradental sensory units & their responses to stimulation. JDR.1985;64:564-571.

- Neill, J. The Hydrostatic Pressure Doctrine. Transactions of the College of Physicians of Philadelphia. 1838, Vol. 111 No. 2,
- Ngassapa D. Neurophysiologic basis, aetiology & clinical aspects of hypersensitive teeth. East Afr Med Jour.1996;73:775-778.
- Olgart L et al. Nerve-pulp interactions. Arch Oral Biol.1994;39:475-545.
- Orologio et al. desensitizing effects of Gluma & Gluma-2000 on hypersensitive dentin. Am J Dent.1993;6:283-285.
- Orban B. Odontoblasts in the dentinal tubuli. JDR.1941;20:553-557.
- Orchardson R et al. Clinical features of hypersensitive teeth. Brit Dent J.1987;162:253-256.
- Orchardson R. Strategies for the management of hypersensitive teeth. Dentin / Pulp Complex. Tokyo. Quint Pub Co.,Ltd.1996:85-89.
- Pashley D. Dentin permeability, dentin sensitivity & treatment through tubule occlusion. J Endo.1986;12:465-474.
- Pashley E et al. Dentin permeability: Sealing the dentin in crown preparations. Oper Dent.1992;17:113-20.
- Pawlowska J. Strontium chloride—its importance in dentistry & prophylaxis. Czas stom.1956;9:353-361.
- Peacock J et al. Action potential conduction block of nerves *in vitro* by K-citrate, K-tartrate & K-oxalate. J Clin Perio.1999;26:33-37.
- Powell G. Laser in the limelight: What will the future bring? JADA.1992;123:71-74.
- Reinhardt J et al. Effect of Gluma desensitization on dentin bond strength. AJD.1995;8:170-172.
- Saleeb F et al. Surface properties of alkaline earth apatite's. J Electro-anal Chem.1972;37:49-53.
- Salvato A et al. Clinical effectiveness of a dentifrice containing potassium chloride as a desensitizing agent. Am J Dent.1992;5:303-306.
- Scherman A et al. Managing dentin hypersensitivity: what treatment to recommend to patients? JADA.1992;123:57-61.
- Schubach P et al. Closing of dentinal tubules by Gluma desensitizer. Euro J Oral Sci.1997;105:414-421.
- Sena F. Dentinal permeability in assessing therapeutic agents. Dent Clin N Am.1990;34:475.
- Silverman G et al. Desensitizing effect of a K-chloride dentifrice. AJD.1994;7:9-12.
- Skartveit L et al. *In vivo* uptake & retention of FI after a brief application of TiF₄ to dentine. Acta Odont Scand.1989b;47:65-68.
- Skartveit L et al. Root surface reactions to TiF₄ & SnF₂ solutions *in vitro*. An ultra structural study. Acta Odont Scand.1991;49:83-90.
- Smith H. Some observations on the cellular elements of the dental pulp. Brit J Dent Sci.1893;Vol.36:1104-1117.

- Smith H. The so-called "innervation" of dentin: An epicriticism. *Dent Cosmos*.1916;58:421-427.
- Smith H. Some observations on the histology, physiology & pathology of the dental pulp. *Proc Royal Soc Med. Sect of Odont*.1923;6:58-71.
- Spiro S. *Pain & Anxiety Control in Dentistry*. New Jersey: (ed) J K Burgess, Inc.1981:7-11.
- Stead W et al. A mathematical model for K-diffusion in dentinal tubules. *Arch Oral Biol*.1994;39:145.
- Smith H. The non-innervation of dentine. *Proc Royal Soc Med. Sect of Odont*.1924;17:63-79.
- Suggs A et al. Colloidal MSE for differential diagnosis & treatment of dentin hypersensitivity. *Dentin / Pulp Complex*. Tokyo. Quint pub. Co. Ltd.1996:245-247.
- Tarbet W et al. Clinical evaluation of a new treatment for dental hypersensitivity. *J Perio*.1980;51:535-540.
- TenCate A. *Oral histology development, structure & function*. St. Louis. Mosby Year Book Inc.1994. 204-209.
- Teranaka T et al. Effect on root surface demineralization treated with FI-Lanthanum. *JDR*.991;70:308.
- Trowbridge H et al. Effects of eugenol on nerve excitability. *JDR*.1977;57:115.
- Trowbridge H. Interdental sensory units: physiological & clinical aspects. *J Endo*.1985;11:489-498.
- Trowbridge H. Mechanisms & control of pain in dentin & pulp. *Univ of Mich. Symp*:Nov.1985.
- Trowbridge H. Review of dental pain- Histology & physiology. *J Endo*.1986;12:445-452.
- Trowbridge H et al. Effect of ZnOE & Ca(OH)₂ on interdental nerve activity. *J Endo*.1982;8(9):403-406.
- Tomes J. On the presence of fibrils of soft tissue in the dentinal tubes. *Philos Trans Royal Soc London*.1856;Vol. 146:515-523.
- Tomes J. *A system of dental surgery*. London.1859:322-335.
- Weber D. Human dentine sclerosis: a microradiographic survey. *Arch Oral Biol*.1974;19:163-169.
- White C. On some points on the minute anatomy of the pulps of the teeth. *Trans Odont Soc*.1870;5-93.
- White J et al. Effect of Nd: YAG laser treatment on hydraulic conductance of dentin. *JDR*.1990; 69:169.
- Wichgers T et al. Dentin hypersensitivity. *Gen Dent*.1996;May-June:225-230.
- Yamamoto Y et al. Precipitation of oxalates in dog dentinal *tubules in vivo*. *Dentin / Pulp Complex*. Tokyo. Quint Pub Co.,Ltd. 1996:278-279.
- Yoshiyama M et al. Adhesion to wedge-shaped defects & treatment of dentin hypersensitivity. *Modern Trends in Adh Dent*. Sapporo Japan Feb 21,1998.