Shiraz University Of Medical Science

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TITLE:

PULP CAP AND PULP RESPONSE

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Thesis For The Degree Of D.M.D

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October: 1999

Pulp Cap and Pulp Response

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Content *page*

Intruduction

Repair is surprising power which is present in living creature compensate injury. All medical procedure in fact are in direction to this power to function perfectly as much as possible and without this nothing can save health of living tissue.

In the field of dentistry, progress to a conservative approach is sensible. In former times extraction was the only way to get ride of dental pain.

Sometimes later, It became clear that earlier removal and filling the tooth can usually stop the disease and return the tooth to normal function and since then filling method and matrial began to improve. However deep caries which injured The pulp was a problem becuse they didn't know a way to preserve vitality of injured pulp and return it to normal position method to coverd pulp wound like an open wound on another part of body faild becused pulp can not tolerate more than a limited inflammation this procedure made nerve ending very sensetive and result to an awful pain. there for destraction or removal of this "troublesome nervetissue" appeared to be the solution.

Pulp is the formative organ of the tooth it build primary dentine during the development of the tooth. Secondary dentine after tooth eruption and reprative dentine in response to stimulation as long as odonotoblast remain intact.

Exposure of the pulp cused most commonly by caries but may also cused by trauma from blow or during cavity prepration. Pulp exposure by caries occure more frequently in primary teeth. Exposure of the pulp by caries is

invariability accompanied by infection of the pulp and trumatic exposure is fallow by infection if the expose pulp become contaminated by saliva.

The aim of pulp capping is to maintain the vitality of the pulp.Becuse even a tooth with perectly performed and successful root cannal treatment possesses certain disadvantag when compared to a tooth with a vital pulp.

There are 3 acceptable procedure for maintaing vital pulp.

1) Natural pulp cap.

2)Direct pulp cap.

3) indirect pulp cap.

By the way there are many skeptics who condemn pulp capping but like to keep an eye on research progress being made considerable literature emphasize the negative aspect of vital pulp therapy and discourages it's practice. Some clinicians and investigators continue to condemn pulp capping therapy for the same reason reported in the literature 80 years ago despite the advances made in pulp biology. Clinicians are well aware of immediate and long term success race after root canal therapy but are less certain of success of pulp cappingy a number of negative question plague clinicians when confronted with the choice of treatment. The research data on pulp capping is at times inadequate, confusing, mis-leading or even incorrect and diminishes the confidence of the practitioner in performing pulp capping. (1)

How ever studies have shown that success rate is vary form 50% to 70% depending on method used to evaluate the out come.

I.Histology

Odontoblasts

odontoblast, the second most prominent cell in the pulp, reside adjuecent to the predentin with cell bodies in the pulp and cell processes in the Dentinal tubales. They are approximately 5 to 7 μm in diameter and 25 to 40μm in length.

They have constant location adjucent to predentin in what is termed the odontogenic zone of pulp. These cell arise from preipheral [(2)] Mesenchymal cell of dental papilla during tooth development and differentiate by acquiring the characteristic morphology of glycoprotein synthesis and secretion.

The cell bodies of the odontoblast columnar in appearance [(2)] the cell body of active adontoblast has a large nucleus that may contain up to four nuclei. The nucleus is situated at the basal end of the cell and is contained within a nuclear envelop, a well developing golgi complex centrally located in the supranuclear cytoplam, consist of an assembly of smooth walled vesicles and cisternae. Numerous mitochondria are evenly distributed throughout the cell body. Reris particulary promineat consisting of closely stacked cisternae form in parallel arrays that are dispersed diffusely within the cytoplasm. Numerous ribosomes closely associated with the membranes of cisternae mark the sites of protein synthesis within the lumen of the cisternae filamentous material probably representing newly synthesized protein can be observed. [(3)]

The Mature odontoblast does not well tolerate change in it's surrounding. If odontoblast stimulate it may activate to form dentin in highly rate. This may occure under normal physiological circumstance . Odontoblasts are capable of mitosis therefore when these cells injured it dies and is replaced by underlying cell that mature from dividing undifferentiated precursors or by re differentation of fibroblast. Any damage to dentine is passed to the odontoblast that is intimatly related to dentin through the communicating tuble.

The cell actually increase as it's process leghes during dentin formation when the procces become 2 mm long. It is then many times greater in volume than the cell body. (2)

In the coronal portion of the pulp where odontoblast are more columnar they elaborate regular dentin with regular dentinal tuble.

The odontoblast in apical portion appear less differentiated and elaborate less tubular more amorphous dentin. (4)

Reparative Dentin.

The term most commonly applied to irregularly formed dentin is reprative dentin, because it so frequently formes in response to extensive abrasion, errosion, caries, or operative procedure. And appears to be a component of the reparative process. It must be recognized how ever that this type of dentin has aslo been observed in the pulp of normal un erupted teeth without any obvious injury. [3] It will be recalled that secondary dentin is deposited cir cumpulpally at very slow rate through out the life of the Vital tooth. Incontrast formation of reprative dentin occure at the pulpal surface of primary or secondary dentin at sites corresponding to areas of irritation. [3]

The majority of odontoblasts in this situation degenerate but a few may continue to form dentin. Some of the odontoblasts that are killed are Replaced by the migration undifferentiated cells arising in deeper regione of the pulp to the dentin interface it is belived that the origion of the new odontoblast is From undifferentiated perivascular cell both of the damaged and the newly differentiated odontoblasts then begin deposition of reparative dentin this action to seal off the zone of injury occurs as a healing process initiated by the pulp, resulting in resolutions of the inflammatory process and removal of dead cells. [2]

Reprative dentin is less tubular and the tubules tend to be more irregular with larger lumina in some cases no tubules are formed the cells that form reparative dentin are not as the primary odontoblasts of the coronal pulp and are often cuboidal [3]

The newly form dentin bridge is composed first of thin layer of a tubular dentin on which a relatively thick layer of tubular Dentin is deposited. The fibro dentin was lined by cell resembling mesenchymal cells where as the tubular dentin was associated with cell closely resembling odonotblast.

It's seems that this layer protect the pulp or in other way it serves a protective function. The junction between developmental and reprative dentin has been studied using a dye diffusion technique first noted the presence of an a tubular zone situated between primary dentin and reprative dentin. Scott and Weber found in addition to a dramatic reducation in a number of the tubles that the wall of the tubles along the junction were thickened and often occluded with matrial similar to peritubular Matrix. These observation would indicated that the junctional zone between developmental and reprative dentin is an a tubular zone of low permeability. (3)

There is investigation was desinged to study the formation of secondary dentin in permanent teeth of young cat ofter denervation the result was that formation regular secondary dentin appear to be enhanced on the denervated side at 30 days, and 90 days post operatively. Where as at 180 days there was no differance between sides. The result indicate that intradenal Nerve influence secondary dentin formation in feline permanent teeth. (6)

Histopathology

The dental pulp is delicated connective tissue liberally interspersed with Tiny blood vessels. Lymphatics myelinated and unmyelinated nerves and undifferentinted connective tissue cells [5]

The enclosure of the pulp tissue within the calcified walls of the dentin precludes the excessive swelling of tissue that occure in the hyperemic and edematous phase of inflammation. In other tissues the fact that the blood vessels supplying the pulp tissue must enter the tooth through the tiny apical formina precludes the development of an extensive collateral blood supply to the inflammed part. [5]

The inflammation can range from Acute to chronic and it may be precipited by chemical, bacterial, or radiation type injuries or by a combination of these.

Initialy either injury to cells or enzymatic by product metabolism may cuse release or activation of chemical mediators which in turn cause vasodialation. An icrease in blood vessel permeability and leukotaxis these chemicals plus change in metabolism contribute to the cardinal sings of inflammation evedent in any connective tissue. [7]

There are several types of chemical mediators which play a role in pulp inflammation.

Most of the cause of pulpitis are primarily a result of the dental caries in which bacterial invasion of dentin and pulp tissue occurs. Dental caries does not result an acute inflamatory.

The pulp defends itself against the lesion of caries quite efficiently.In response to oncoming process of decay sclerosis of dentin an increase in peritubular dentin constitues the initial defense of pulp against dental caries. Tending to slow down the decay. In response to further irritation as a caries progresses, the pulp will intiate reprative dentin formtion. (4)

Frankle 1972 said that the reaction of pulp dentinal system to an irritant will be proportional to the duration and intensity of the offending agent.

The cellular elements in choronic inflammation are lymphocyte, plasma cell, and macrophge. An increase in number of mast cell has been reported in association with plasma cell and lymphocytes, and. In chronic inflammation have the function of producting of antibodies which Neutralize antigen. Thus the reaction to dental caries apparently involve. (4) both cell mediated and hormonal immunologic phenomena. Theoretically the local immune response should retrad and eventually eliminate the inflammatory process by Neutralizing and eliminating the ethiological factors how ever the immunologic mechanisms may contribute to the destructive phase of inflamation and there is possibility of an Arthus type reaction may also occur in the later stage of pulpitis with infiltration of polymorphonuelear leukocyts. (4)

Degranulation of these cell in the presence of complement serves pulp destruction and pulp death or pulp Neccrosis.In most cases there may be an Acute exacerbation of a chornic inflammation with appearance of leukocytes rapidly migrate through the endothelium lined structures in increasing numbers. (5) ,and there is vascular dilation which lead to escape of microganism in to the pulp tissue proteolytic and other enzmes present in

lysosomes. Activate and the break down material and microorganism phagocytized by macrophage, and there is caries reduction in pulp size by secondary or reprative dentin formation and reduction in vascularity by pulp stones, (5) Dystrophic calcification and scar tissue in the infected pulp chambers there are severl species of bacteria more than 90% of the strain are anaerobic. (5)

If there is specific micro organism cause pulpal and periapical infection, therefore there will be swelling and exudatine.

Continious marginal leakage is one of the common cause of the pulp degeneration under restoration.

The function of the pulp are nutritive becuse it Nourishes the dentin throug odontoblast and their processes and by mean of the blood Vascular system of the pulp. Protective becuse the sensory Nerves in the tooth respond with pain to all stimuli, the Nerves also initiate reflex that control circulation in the pulp. Other function of the pulp are fomative and inductive. Formative because the pulp organ cell produce the dentin that surrounds and protect the pulp and inductive because it induce oral epithelial differentiation into dental lamina and enamel organ formation. (2)

components against harmful physical ,chemical and microbial irritants.

After mechanical exposure an acute inflammation occure in pulp at the site of the exposure but the remainder of the pulp is usually unaffected the underlying blood vessels dilate,edema occurs and polymorphonuclear leukocytes accumalate at the site of injury within a few days an acute abecess may develop in the region of the exposure. (4) The severity of the reaction

depend on the amount of the initial tissue damage. How ever in humans, repair can occure ofter Pulp exposure the prognosis for healing is much better for mechanical exposure of the pulp than it is for carious exposure, becuse the pulpitis which develop after machanical exposure usually is not complicated by precvious inflammation and infection. (4)

Repair depends on the amount of tissue destraction the amount of hemorrhage, the patient age(and hence the blood supply of tissurs), the resistance of host and other factors which influence the ability of injured pulp, and connective tissue to repair itself. (4)

There is study that, expression of transforming growth factor-Beta "tGf-beta" is forms odontoblast, Lead to their sequestration within dentin matrix."tGf beta1" and "beta3" stimulate matrix secreation and aslo initiate odontoblast cytodifferentiation in vitro and in vivo. The result of this study that "TGF-Beta1" and "TGF Beta3" can stimulate secreation of extracellular Matrix by odonotblast, are Mitogenic to pulp cell and that "TGF-Beta3" may have inductive effects on pulpal cell. Such activats might be important during reparative processes in dentin-Pulp complex after tissue injury. (9)

There is another study which revels cell monolayers from human pulpal explants were passaged 3 to 4 times befor characteriztion of the response of the cell to calcitonin gene- related peptide "CGRP".

These results indicate that pulp cell possess the cellular machinery to respond to "CGRP" and that stimulation of the production of Bone morphogenetic protein 2 "BMP-2" a factor to be associated with induction of dentin formation is component of the response. (10)

Using of human dental pulp fibroblast or cell in experimental data can be strongly influenced by the pulp cell strain used and the culture technique employed, indeed study of human pulp cell proliferation using pulp cell which are not of the same transfer number may not be relevant. (11)

another studies show that recombinant human osteogenic protein-1 in collagen carrier matrix appear to be suitable as a bio-active capping agent for surgicaly exposed dental pulp. (12)

adminestration of micro dose calcium into small pulpal exposure in rat maxillary insisor tooth in the absence of hydroxyion shows that in vitro complete release of all calcium salt occurred within 12-15 hours except for the very water insoluble calcium stearate. It was anticipate that the relase of clciun would be significantly more prolonged in vivo becuse of the physical constraints of prepared cavity as well as resricted access to fluid flow (13)

Reaction aslo occure around the dentin chips which have been pushed into the pulp as a result of exposure fibroblasts or undifferentiated mesenchymal cell are attracted to these chips and being elaborating dentin matrix as more matrix is elaborated many chips may be welded togather forming a dentin bridg. The formation of reparative dentin is part of repair process but this not the final proof of successful pulp capping (4)

after amount of large exposure the remainds of the pulp may be replaced by granulation tissue. 3 months later the pulp in most instance is still chronically inflamed or become necrotic and granuloma developed apically.

Fig 1:

Acute inflammation fallowing mechanical pulp exposure (top) under the region of the exposure (exp) an acute abcess (Abs) has developed (bottom). Higher magnification of inflammatory cells in the pulp. (P) They are mainly polymorphonuclear leukocytes (D) dentin.

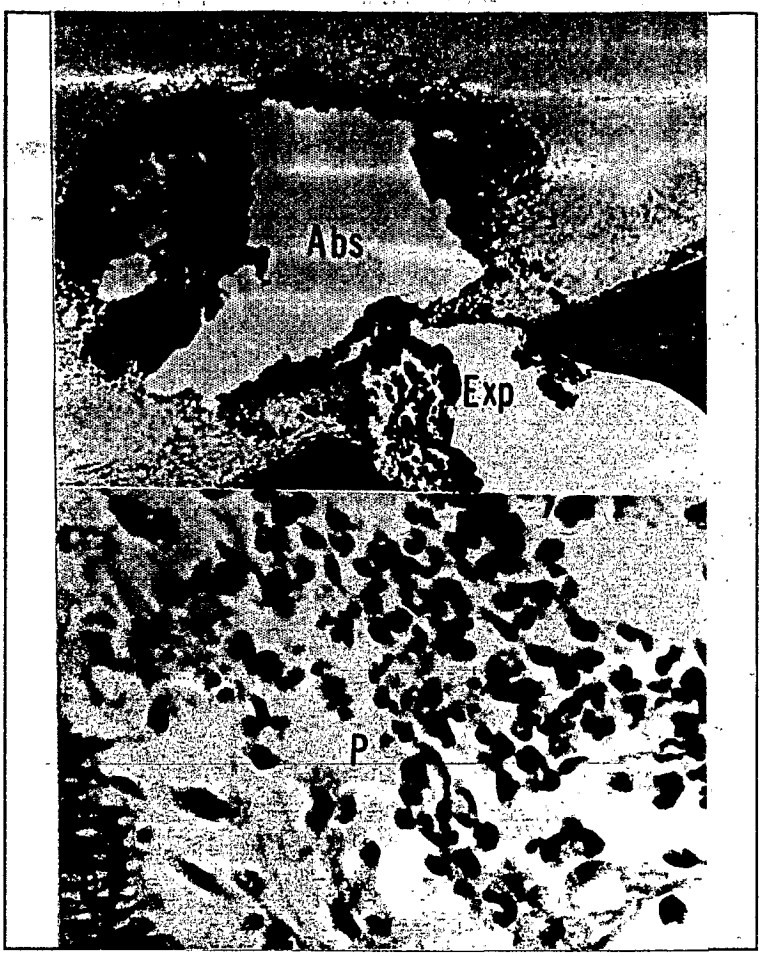

Fig 2:

The pulp (P) recovered after exposure A bridg (B) composed of reparatiue dentin (Rd) has formed over the region of exposure(D) dentin

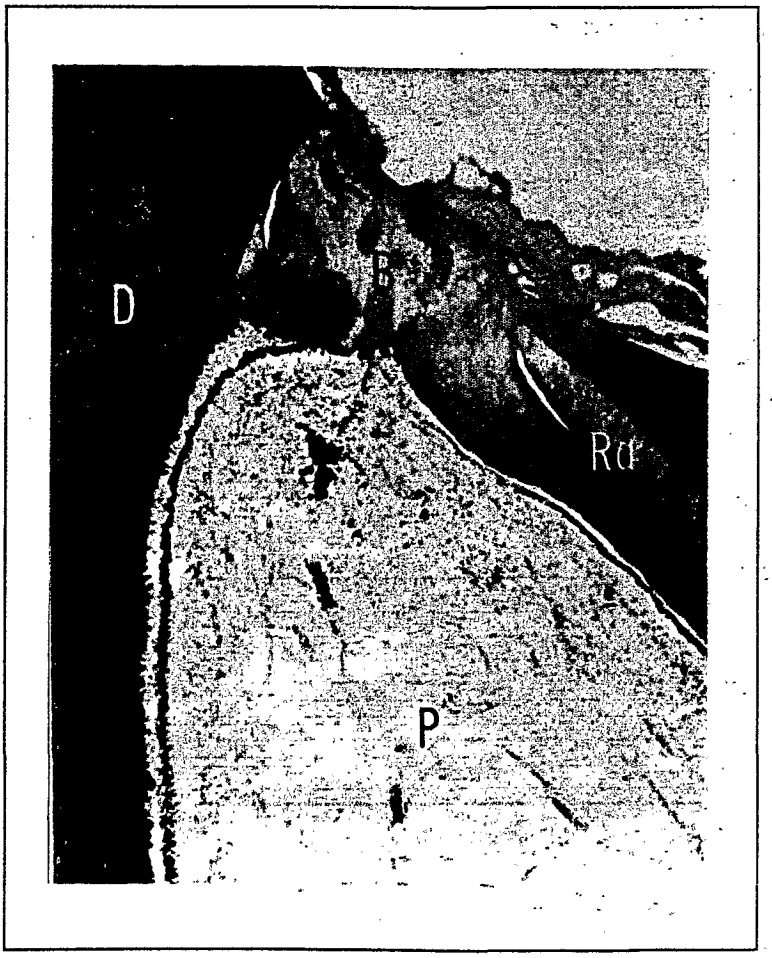

Fig 3:

Inflammation of pulp under bridge in spite of (bridge) formation (B) The pulp (P), is chronically inflammed (INF). (DC) dentin chips

Fig 4:

Calcification under calcium hydroxide eight months after the application of calcium hydroxide to an exposed pulp (P), Calcification (Ca), has continued almost obliterating the pulp (D) dentin, (Rd), reprative dentin

Pulp studies have shown. The presence of immunocompetent cell and cells that recognize foreign antigens as a result of intraction of micro or rganism and their by products various mediators of inflammation such as neuropeptids, vasoactive, amines, kinins, complement compoents, and arachi doniac acid metabolites are released.

Neuropeptid are protein generated from somatosensory and autonomic nerve fiber fallowing injury. These substance participate in process of inflammation and pain transmission.

The importance of vasoactive amines such as Histamin pathophysiology of pulpal inflammation by the presence of histamin like substance in wall of blood vessels in experimentaly incluced pulpities.

Kinins are considered the main mediators of the pain associated with inflammatory responses the complement system when activated causes enhanced phagocytosis and increased vascular permeabiltiy .

Arachidonic acide is released from the phospholipids of cell membrance as a result of cell damage. When metabolized various prostoglandins thromboxanes and leukotrienes are produced these metabolites have been identified in exprimentaly induced pulpities an their concentration have been significantly reduced by the use of non steroidal anti inflammatorry medication. (3)