

Proximal Contact Areas: A Forsaken Facet of Restorative Dentistry

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The integrity of dental arch is maintained through proximal contact areas between the teeth in the same arch and through the occlusal contact areas with the antagonist arch. A vital amount of care is taken to adjust occlusal high points in restorative dentistry. On contrary proximal contact area disharmony is always an abandoned factor in clinical practice. In fact, a little information is available in literature for managing proximal contact disharmony. Glossary of Prosthodontic Term defines Interproximal Contact Area as the region on the proximal surface of a tooth that touches an adjacent tooth [1].

The size, location and shape of these proximal contact areas depend on the anatomical surface contours of the two adjacent proximal surfaces, and whether they are on the mesial or distal aspects of the teeth [2]. Proximal Contact Strength is defined as the resistance against separation of contact area during function [2].

Significance of Proximal Contact Area

The tooth is stabilized by contact with the adjacent teeth as well as by occlusal contact with the opposite tooth [3].

1. Masticatory forces are exerted on the teeth from the antagonist and through the food bolus. These forces are exerted in various directions and are transmitted to adjacent teeth and periodontal ligaments. The redistribution of these forces is carried out through interproximal contact areas. It provides an efficient mechanism for protecting the teeth and the periodontium against trauma [2]. Hence correctly located proximal contact areas maintain integrity of dental arch.
2. The morphology of the proximal area is such that the adjacent marginal ridges on the occlusal aspect of adjacent contacting teeth are analogous to the transverse fissure of a molar, deflecting the food to the buccal and/or lingual aspects of the teeth during chewing. Hence the proximal

contact also plays an important role in protecting the periodontium against damage due to food impaction.

- Excessive proximal contact pressure between teeth can cause wedging of teeth and undesirable tooth movement consequent crowding and/or repositioning of teeth that can change the occlusion, leading to strain on the musculature, causing myofascial and temporomandibular joint dysfunction. Conversely, a weak or slightly open contact would permit food impaction and cause subsequent dental caries, halitosis, periodontal disease, or drifting of teeth.
3. During parafunctional masticatory movements, teeth and supporting tissues are guarded from excessive occlusal loading by distribution of occlusal forces through appropriate proximal contacts. Hence restoring proper proximal contact is an important aspect of treatment TMJ disorders and occlusal rehabilitation.
 4. The contact point defines dimensions of the gingival embrasure and the height of the interdental papilla, as well as the incisal embrasure, which widens coronally from the area of contact. Maintenance of proper incisal embrasure dimensions is important for efficient mastication and give individuality to the anterior dentition [4].

Important Guidelines for clinicians

- Dental laboratory technician should be requested to avoid adjustment of the proximal surfaces of the adjacent teeth on the final cast, in order to maintain the natural contour and space between the teeth.
- The fit, margin, occlusion, and proximal contacts of the completed restoration should first be evaluated on the articulated model.

- The marginal fit of the restoration should be evaluated clinically with an explorer. If margins are properly adapted the proximal contacts should be checked with ultra-thin abrasive dental strips to find the tight proximal contact on either side. If the restoration is not adapted properly, the side with the less marginal discrepancy is the side with the tight proximal contact.
- The clinical adjustment and refining of proximal surfaces of final restoration should preferably be performed using abrasive diamond strips. These are available in various coarseness designs. Conventional method of refining proximal surfaces of restoration by using rotary instruments may damage the restoration. Also, small restorations such as crowns, inlays, or onlays are not only difficult to hold but they are also difficult to see. Furthermore, incremental adjustments with this method require multiple trials. This adds to the appointment time.
- The final seating of the restoration should be completed by passing the serrated diamond strip gently bucco-lingually to cut and clean out the remaining cement in the distal and mesial interproximal spaces.

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Clinical examination and samples processing

The rats were sedated by halothane and the supporting tissues of the teeth were clinically evaluated for possible tissue alterations, such as redness, swelling, and suppuration. After the respective monitoring periods, rats were chosen from each treatment group and the rats were anesthetized then euthanized with over dosage of i.p. injection of thiopental (5 ml/100g, Triopental, Biochemie GmbH, Vienna, Austria). The mandibular segment containing incisors teeth of the rats were placed in 10% buffered formalin for 24h at 4°C and then decalcified for 4 weeks in ethylenediaminetetraacetic acid. The specimens were embedded in paraffin and 5 µm thickness serial sections were prepared through the exposure site. Light microscopic examination was performed in haematoxylin and eosin stained sections for histological changes.

Histologic assessment

The following criteria [14] were used to assess the specimens:

1. Inflammatory cell response: inflammatory cell infiltration of the pulp tissue was scored as (0), absence of inflammatory cells; (1), a few scattered inflammatory cells; (2), moderate inflammatory cell infiltration around the exposure site; and (3), heavy inflammatory cell infiltration of the coronal pulp or abscess formation.
2. Tissue necrosis: Pulp tissue organization was scored as (0), no necrosis or presence of complete tissue organization in coronal and radicular pulp; (1), partial necrosis to the area under capping material (2), partial necrosis to the half or more of the coronal pulp tissue and (3), complete tissue necrosis of the coronal pulp.
3. Hard tissue formation: The presence of discontinuous or continuous hard tissue around the exposure site in all the sections examined was scored as (0), no dentin bridge (1), partial calcified bridge formation and (2), complete calcified bridge formation.

Statistical analysis

The obtained data were analyzed using Mann-Whitney U test for pairwise group comparison. Wilcoxon signed rank test was used for the group comparison at different time periods.

Results

Histological results

All procedures were performed according to the institutional standards for the care and use of experimental animals at the Medical Experimental Research Center (MERC), Mansoura University, Egypt. A total of 52 male Wistar albino rats (150 - 200g) were used for the study. Before the experiment, rats were fed standard rat chow and water ad libitum and housed in cages with controlled temperature 22°C and 12h light/dark cycle for 1 week. Rats were randomly allocated into three groups. Group I (negative control, n

= 3), group II (positive control, n = 24) and group III (tested one, n = 24). Group I rats received no treatment. The lower incisor pulps of group II and group III rats were directly capped with 3Mix-MP and calcium hydroxide, respectively. One-third numbers from each group were euthanized according to experimental periods of 2 days, 15 days and 30 days.

Figure 1: Representative decalcified section of group I showing (A) rat dental pulp (P) with highly cellular connective tissue. Inset, higher magnification showing tubular dentin (D), predentin (Pd), odontoblastic layer (Od). Group II after 2 days (B) the exposed area is obstructed by fibrous tissue (Ft) and the whole pulp infiltrated with inflammatory cells (Ic). Inset, cytoplasmic vacuolization (Vac) of the pulpal cells and partial collagen hyalinization (Hy). Group II after 15 days (C) the pulpal exposure site completely obstructed with more fibrous tissue (Ft), greater area of collagen hyalinization (Hy) with diffuse pulp disorganization (Dis). Inset, higher magnification for pulpal tissue disorganization (Dis) and hyalinization (Hy). Group II after 30 days (D) little amounts of fibrous tissue and collagen hyalinization (Hy) with discrete pulp disorganization (Dis) close to the pulp exposure site. Group III after 2 days (E) a small superficial areas of clot necrosis (Ne) as well as small zone of fibrous tissue (Ft) beneath necrotic tissue. Inset, higher magnification for the zone of fibrous tissue (Ft). Group III after 15 days (F) formation of a reparative dentin bridge (Db) at the site of the exposure. Inset, a cellular dentin bridge (Db). Group III after 30 days (G) complete dentin bridge (Db) formation and normal highly cellularized pulpal connective tissue (p) (H and E x100 and inset x200).

Statistical results

Mann-Whitney U test revealed a significant difference between group II and group III regarding amount of inflammatory cell infiltrate and the areas of tissue necrosis at all examination periods. In addition, a significant difference was found between the two groups regarding amount of hard tissue formation at 15 and 30 days examination periods. No significant difference

was found between the two groups at 2 day examination period (P = 1.000) (Table 1). Wilcoxon signed rank test demonstrated significant differences between 2 and 15 days, 2 and 30 days and 15 and 30 days examination periods for group II and III for the parameters tested except the amount of tissue necrosis at the period of 15 days compared to 30 days for group III (P = 0.109) (Table 2).

Periods	2 day					15 day					30 day				
	Rank		Mann-Whitney	Z	Sig	Rank		Mann-Whitney	Z	Sig	Rank		Mann-Whitney	Z	Sig
	II	III				II	III				II	III			
Inflammatory cell response	28.77	20.23	185.5	-2.645	0.008	32.31	16.69	100.5	-4.223	0.000	30.50	18.50	144.0	-3.338	0.001
Tissue necrosis	29.67	19.33	164.0	-2.973	0.003	32.50	16.50	96.0	-4.529	0.000	28.50	20.50	192.0	-2.514	0.012
Hard tissue formation	24.50	24.50	288.0	.000	1.000	17.63	31.38	123.0	-3.968	0.000	17.50	31.50	120.0	-4.399	0.000

Table 1: Mann-Whitney U test for pairwise group comparison and its statistical significance.

Variable Groups	Inflammatory cell response ranks						Tissue necrosis ranks						Hard tissue formation ranks						
	Group II			Group III			Group II			Group III			Group II			Group III			
	-ve	+ve	Sig	-ve	+ve	Sig	-ve	+ve	Sig	-ve	+ve	Sig	-ve	+ve	Sig	-ve	+ve	Sig	
Wilcoxon test																			
2day*15 day	5.94	0.00	0.005	10.50	1.50	0.000	7.15	6.50	0.046	7.08	6.00	0.003	0.00	11.50	0.000	0.00	12.50	0.000	
2 day*30 day	10.00	0.00	0.000	11.50	0.00	0.000	8.00	0.00	0.000	10.00	0.00	0.000	0.00	12.50	0.000	0.00	12.50	0.000	
15 day*30 day	7.92	5.00	0.006	6.00	6.00	0.007	8.00	0.00	0.000	6.19	5.50	0.109	5.50	6.05	0.008	0.00	5.00	0.003	

Table 2: Wilcoxon signed rank test for group comparison between 2, 15 and 30 days and its statistical significance.

Discussion

Despite the progress made in the field of pulp biology, there is no single therapeutic regimen for direct pulp capping that can achieve, predictably and reliably, the goals of preserving tooth vitality and tooth function [15]. Calcium hydroxide being considered the gold standard for vital pulp therapy, it has been shown that calcium hydroxide seriously impeded the healing process [16]. With the advent of non-instrumentation endodontic treatment and lesion sterilization and tissue repair, local application of antibiotics has been investigated.

3Mix-MP TAP has been used for decontamination of young permanent teeth aiming the regeneration of dental pulp tissue [17]. *In vivo* researches, have shown that this specific combination of antibiotics is the most successful; but no study to date has confirmed that it is the most efficient one

available [10,12,18,19]. Revascularization/Revitalization of open-apex primary incisors has become a viable treatment option, particularly in cases of traumatic pulp injuries [20]. In these cases, direct pulp capping might be indicated for maintaining pulp vitality and function [21].

Primary teeth shows close similarity to those is the rat incisors which called open rooted teeth that, refers solely to the large apical opening [22]. Regeneration in teeth with open apices presents multiple challenges for successful treatment. Depending upon the above concepts, the goal of the present study was to evaluate the response of the exposed incisor rats' dental pulp to 3Mix-MP TAP, qualitatively and quantitatively. Limited data available regarding histological reaction to the use of 3Mix-MP, thus the findings of this study can only be compared to other medicament used as a pulp capping.

In the present study, pulp response in group III after 2 days showed small superficial areas of clot necrosis as well as small zone of fibrous tissue beneath necrotic tissue. A pattern which was already described by several studies [23-25]. Most interesting was that during this period a significant difference was found between groups II and III. This might be attributed to the bactericidal activity provided by TAP in group II and the lower pH provided by calcium hydroxide in group III. The necrotic layer can serve as a surface on which pulp cells absorb, polarize, and transform to odontoblast like cells [26].

The production of fibrous tissue was a prominent feature in both groups, such character were also evident in a study of direct capping with platelet-rich plasma and enamel matrix derivative that showed dense reticular fiber deposition where odontoblast-like cells differentiate and organize to produce dentin [27]. A significant difference between groups II and III was found regarding amount of inflammatory cell infiltrate and the areas of tissue necrosis at all examination periods. The observed presence of a moderate amount of inflammatory cell infiltration within the pulp tissue is consistent with Browne, *et al.* [28] and Cox [29] as they stated that favorable pulpal responses accompanied by the presence of some inflammatory cells indicate a bacterial-tight seal preventing microleakage.

Regarding amount of hard tissue formation no significant difference was found between the two groups at 2 day examination period. According to Moazzami, *et al.* [30], the dentine bridge was histologically detectable after two weeks of pulp capping with mineral trioxide aggregate. In a recent study [31], one day of pulp capping dispersed dentin particles was visible as signs of mechanical trauma due to the pulp exposure procedure.

Two weeks after direct capping with 3Mix-MP TAP, the pulpal exposure sites was completely obstructed with more fibrous tissue formation underlined by greater area of collagen hyalinization with diffuse pulp disorganization which decreased after 30 days. The intense fibroblastic proliferation tends to isolate the exposure area [25]. Group treated with calcium hydroxide showed, formation of reparative bridge with a tubular dentin at the exposure site after 15 days. Later on, complete dentin bridge formation and normal highly cellularized pulpal connective tissue were seen after 30 days.

Formation of mineralized barrier was concluded to the formation of dentin bridge [32]. The density of odontoblast-like cells was suggested to be the most important factor influencing dentin bridge formation [33], whose potential depends on several pre- and post-operative factors such as pulp status, the prevention of bacterial infection, and the exposed pulpal size [34].

A significant difference was found between the two groups regarding amount of hard tissue formation at 15 and 30 days examination periods. Also, significant differences at different time periods for group II and III for the parameters tested except for the amount of tissue necrosis for group III at the periods 15 days compared to 30 days.

With respect to the hard tissue formation on the exposed pulp tissue, the average amount of generated hard tissue in the calcium hydroxide treated group was significantly better compared to the experimental group used 3Mix-MP TAP. It is well known that calcium hydroxide have a defect in the continuity of dentine bridge-like structures, allowing the penetration of microorganisms into the pulp tissue via microleakage from the cavity margin [35], and also its antimicrobial properties are lost [36]. Probably if evaluation period were longer, signs of complete dentinal bridge formation would be seen.

On the other hand TAP can maintain pulp tissue integrity as it contains both bactericidal (metronidazole, ciprofloxacin) and bacteriostatic (minocycline) agents [19]. Tetracycline causes inhibition of collagenases and matrix metalloproteinases also increase the level of interleukin-10, which is an anti-inflammatory cytokine. In addition, metronidazole and ciprofloxacin can generate fibroblasts. These fibroblast cells play a significant role in tissue regeneration by involving pulp progenitors via complement activation, which throws light on the probable reparative procedure of targeting pulp fibroblasts in dentin-pulp regeneration [37-40].

Also in this study, It was observed that 3Mix-MP seem to preserve pulp tissue and promote the regeneration of soft tissue. The nearly normal pulpal architecture indicates that the TAP has good biocompatibility and regeneration ability. The criteria that characterize a successful direct pulp capping vary among authors. Clinically successful pulp capping is considered if the tooth is free of symptoms, reacts adequately to sensibility tests and has a normal

radiographic appearance. However, the clinical criterion is inadequate for an evaluation of the long-term prognosis for teeth treated by pulp capping. Therefore, a critical evaluation of the results of pulp capping can only be made histologically [41].

Conclusion

Based on the present study, 3Mix-MP tri-antibiotic paste in pediatric dentistry seems to be optimistic. It is biologically compatible maintaining pulp integrity, but it hasn't the potentiality to induce dentin bridge formation. Further studies with a larger sample size and longer follow-up periods both clinically and histologically are recommended.

Conflict of Interest

All authors deny any conflict of interest.

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