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The International Journal of Oral and Dental Sciences (IJODS) is the official journal of the Lebanese University School of Dentistry. It is a semiannual journal that will be published every May and November. IJODS is a peer-reviewed journal aiming at providing its readers of ideas, opinions, developments and key issues in dentistry - clinical, practical and scientific research advancements spanning over all the dental disciplines.

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Editorial

Knowledge is a deadly friend

As we embark on a new issue of IJODS I would hope that you have already discarded your comfort zone and shaken yourself out of the "numb state". Helping you get out of that state we have added as promised the focus on research section that you will find at the end of each issue.

Gaining knowledge is an important weapon, but having it abused and misused turns it to a weapon of mass destruction. A British rock band; King Crimson; said it best "Knowledge is a deadly friend, if no one sets the rules. The fate of all mankind I see is in the hands of fools." It is essential to combine our knowledge of the subject matter with the wisdom of where, how and when to apply this knowledge. One of the outlets to gain knowledge is through journal manuscripts, please learn how to navigate and extrapolate the right information from said manuscripts.

Dentistry is in constant state of change and we have to be able to move with the fast evolving fields of dental science and industry. Always redefine your standards of care to fit the demands, safety and expectations of patients. Dental educators also have the responsibility of waking up their student's minds turning them into thinkers and eventually wise doers and not just followers.

I would like to remind you that the main mission of the journal is to become an indexed journal. I urge you to strictly follow the author guidelines provided to you at the end of this issue. In addition, we hope to increase the flow of submission, promising that the rate of manuscript acceptance will not be more than 3 months from the date of submission. Please give us your feedback; this input is extremely valuable to the editorial board.

Please read responsibly

Rima Abdallah, BDS, CAGS, DSc
Editor-in-Chief

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Influence of resin cement thickness on the regional push-out bond strength of fiber post to root canal dentin

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ABSTRACT

Objective: The purpose of this study was to evaluate the effect of a self-adhesive resin cement thickness on the regional bond strength of a fiber post to root canal dentin.

Materials and methods: Thirty extracted human maxillary incisors and canines were prepared and divided into 3 groups as follows: Group 1 was restored using a fiber post size identical to the drill size (size 1, Rely X Fiber post, 3M ESPE); groups 2 and 3 were both prepared with drill size 3, and restored using the size 2 and 1 fiber post (3M ESPE) respectively. Fiber post were cemented using a self-adhesive resin cement (Rely X Unicem). Samples were subjected to push out bond strength test and cement thickness analyzed under microscopy. Data were analyzed with a one-way analysis of variance (ANOVA) (p<0.05).

Results: the push-out bond strengths and cement thickenesses were significantly different between groups, and group 2 yielded the highest bond strength (11.7 ± 0.4 MPa) correlated to a mean cement thickness of 157.4 b ± 0.9μm. For all groups, the apical third had the lowest bond strength value (P < .05).

Conclusion: The highest bond strength was obtained with one incremental oversized post, an cement thickness of 150 μm is required for an optimum cementation.

Keywords: Fiber post, push out, bond strength, resin cement

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INTRODUCTION

Endodontically treated teeth with loss of coronal tooth structure generally require a radicular post for restoration of the tooth function. The introduction of fiber posts helped to improve stress distribution, as their elastic modulus is similar to that of dentin as demonstrated by several in vivo[1-3] and in vitro studies.[4,5] Glass fiber posts were reported to contribute to the reinforcement and strengthening of endodontically treated teeth under full coverage crowns.[6-8]

Retention of fiber posts in roots depends on the bond strength between post material and a resin luting agent, bond strength between post space dentin and resin luting agent.[9] In push-out tests major portion of retention will be created not only by adhesive bonding agent, but also by micro retention from the frictional fit between two surfaces.[9]

In order to improve bond strength between pre-fabricated posts and resin cement, surface treatment procedures have been suggested by using mechanical or chemical agents. The chemical treatment is aimed at roughening post surface enhancing

mechanical interlocking between post and resin cements. Recent studies have shown that post surface pre-treatment increases bond strength between fiber and materials used for core build-up.[10,11]

Adhesive failures associated with fiber posts are common and usually happens along the resin cement that fills the post-dentin interface.[12] Although the ideal thickness of the resin cement needed to improve retentive bond strength is under debate; a recent study reported that highest push-out bond strength values were obtained when one incremental oversized post space was used. Clinically, fiber post space has to provide an optimum cement thickness (around 120 μm) for adequate cementation.[13]

An excessively thick layer of luting around a fiber post is an unfavorable factor for the long-term success of post-retained restorations, owing to the high frequency of decementation.[14,15] Passive retention of the post is improved if it fits snugly into the prepared space and if the luting layer is fine and even.[14,16] Post decementation generally occurs at the dentine/cement interface[17,18] due to the bubbles and pores that form as a result of curing stress.[16] The bond between the luting and the radicular dentine is sometimes unable to withstand curing shrinkage.[19]

The aim of the present study was to evaluate the effect of thicknesses of self-adhesive resin cement at different root levels, on the retention of fiber posts to root dentin.

MATERIALS AND METHODS

Thirty recently extracted human maxillary canine and central incisors were selected for this study (mean length 23 ± 2 mm). External debris was removed from the teeth using an ultrasonic scaler (Parkell Electronics Division, Farmingdale, NY, USA). Radiographs were used to eliminate specimens with irregularity shaped canals, with pronounced canal curvatures, or evidence of internal resorption. Teeth with root shorter than 10 mm or with defects or cracks were excluded. The selected specimens were stored in an aqueous solution of 0.525% Chloramine T at 4°C and used within 2 weeks after extraction. Teeth were cut off at 2 mm coronal to the most incisal point of the cemento-enamel junction (CEJ) using a low speed diamond saw (Isomet 2000, Buehler Ltd, Lake Bluff, NY, USA) under copious water cooling.

Root canal preparation was performed at a working length of -0.5 mm from the apical foramen using rotary instruments (FlexMaster, VDW, Munich, Germany) with a step-back technique. Canals were prepared to ISO-file size of 035, 6-degree taper. The root canals were irrigated between instrumentation and canal spaces were filled with irrigation solution (NaClO 5%) during instrumentation phase. Root canals were dried and obturated with thermoplasticized injectable gutta-percha (Obtura II, Model 823-600, Obtura Corp., 1663 Fenton Business Park, Fenton, MO, USA.). After endodontic treatment completion, teeth were stored in water for 24 h. Gutta percha was removed with a warm endodontic plugger. The three experimental groups are listed in table 1 and were as follows:

* Group 1 (n=10): Canals were prepared with low-speed post drill-size 1 provided by the manufacturer (Rely X Fiber Post, 3M ESPE). To ensure parallelism of the paths of post insertion and removal, all post space preparations were performed by the same operator using a dental surveyor (Fedi 18 Mariotti & Co., Italy). The roots were transferred into the acrylic mount using autopolymerizing resin (L.D. Caulk Division, Dentsply Int. Inc. city and country), the acrylic resin extended to a level 1 mm below the buccal aspect of CEJ. Immediately after the acrylic resin reached its doughy stage, specimens were detached from the surveyor and placed into a cool water bath. Subsequently the mounted specimens were secured in the dental surveyor and the coronal 9 mm of each root canal were prepared with the same dowel space drills size 1, (apical=0.7, and coronal=1.3 mm diameter) leaving a minimum apical seal of 4 mm of gutta-percha. Following dowel space preparation, the canals were rinsed with water using a 22-gauge needle-irrigating tip (Ultra dent Products, South Jordan, UT, USA) and dried with sterile paper points (Coltene, Whaledent GmbH, Langenau, Germany). Size 1 (0.7 - 1.3 mm diameter) fiber posts were used for this group. Rely X UniCem Aplicap cement (3M

ESPE) dispensed from auto mixing syringe was placed into the canal spaces with an elongation tip according to the manufacturer instructions. The 1.3 mm dowel space drill was detached from the dental surveyor, replaced with the same diameter of fiber posts and immediately inserted as close to the center of the dowel space as possible to maintain an even thickness of cement.

* Group 2 (n=10): Same procedure as group 1 but the drill size 3 (0.9 - 1.9mm diameter) was used in combination with size 2 (0.8 - 1.6 mm diameter) fiber posts.
* Group 3 (n=10): same procedure as for group 1 but the drill size 3 (0.9 - 1.9mm diameter) was used in combination with size 1 (0.7 - 1.3 mm diameter) fiber posts.

Preparation of specimens for the push-out bond strength test

After 24 hours of water storage at 37°C, the roots were cut into three 2 mm thick slices perpendicular to the long axis of the tooth by using the Isomet saw under water-cooling. The first cut started 2 mm below the CEJ; thus, the slices represented a coronal, middle, and an apical location of the post space preparation. Hereafter, a 1 mm thick slice was cut off each sample using the same saw. The slices were fixed onto slides and polished up to 600 grit. The specimens were then subjected to a push out test in a universal testing machine (Accuforce Elite Test Stand, Ametek, Mansfield & Green Division, Florida, USA) at a cross-head speed of 0.5 mm/minute. With regard to the tapered design of the posts, three different sizes of punch pins, as well as three different openings, were used for the push out testing.[20]

The maximum stress was calculated from the recorded peak load divided by the interfacial area (SL) of the post fragment. In order to calculate the exact bonding surface, the tapered design of the posts with regard to the respective part of the post was considered. Therefore, each specimen was measured using a micrometer screw (Ultra Cal Mark, City, Switzerland), and the SL was calculated using the following formula:

$SL = \pi (R + r) [h2 + (R- r) 2] 0.5$

where $\pi = 3.14$, R=coronal post radius, r= apical post radius and h=specimen slice thickness.

Failure mode Analysis

After push-out test, specimens were assessed initially under a stereomicroscope (Swift Stereo Eighty Microscope, Swift Instruments International SA, Tokyo JAPAN) and then with higher magnification using scanning electron microscopy (SEM) to determine the mode of failure classified as follows: mode I: adhesive failures between post and cement with no cement visible around the post; mode II: adhesive failures between cement and root dentin; mode III: mixed failures with cement covering the post surface; and mode IV: Cohesive failures inside the dentin. For the SEM evaluation the samples were gold sputtered (JEOL, Fine Coat, Ion Sputter JFC-1100, Tokyo, Japan) mounted on a metallic stub, and then observed under a scanning electron microscope (XL 20; Philips).

Cement thickness evaluation

Before push-out bond strength testing, a digital image of each specimen was taken to measure the resin cement thicknesses using a metallographic optical microscope (Leica, Germany) with ×5 magnification. Eight points were determined in each digital image and resin cement thicknesses were measured between the canal wall and the fiber-post perimeter at these points using a specific image processing software (Photoshop CS5, Adobe, CA, USA).

Two specimens from the second apical slices of each group were evaluated by SEM (500× magnification) to compare the cement thicknesses with the optical microscopy results.

Statistical analysis

One–way ANOVA was performed to examine the effect of cement thickness on interfacial strength for each group. In case of significant differences, Post hoc multiple comparisons were performed using the Tukey test ($P<0.05$).

For the variable regional bond strength, same statistical tests were performed; all statistical analysis was performed using statistical software (SPSS Window Standard Version 10.0; SPSS Inc. USA).

RESULTS

The regional push-out bond strength data is summarized in Table 1. The results showed that there was a significant difference ($P<.05$) in mean push-out bond strengths between the 3 groups with different cement thicknesses (Table 2); while group 2 showed the highest mean bond strength (11.7 ± 0.4 MPa). Regarding the variable root regions, the coronal and middle thirds from each groups demonstrated significantly higher bond strengths than the apical third ($P < .05$)

Regarding cement thicknesses (Table 2 and Table 3) there was a significant difference between all groups, while highest thickness was reported on the coronal level of group 3 ($310.91 \pm 1.93\ \mu m$).

Most of the failures (approximately 59% to 62%) were found to occur between the post and the resin cement without any visible cement around the post (mode I). Group 2 was the exception, as this group demonstrated 45% mode I failure and 39% mode III failure (Table 4).

SEM micrograph of middle section of specimen in group 2 showed a good adhesion between the post and the cement. (Fig. 1); while specimen in group 3 reported the presence of wide air bubbles within the core of the thick layer of the resin cement (Fig. 2).

Table 1: Mean and standard deviation of push-out bond strength (MPa) by root section. Similar superscript indicates no statistical significant difference ($p< 0.05$)

Group	Root Section	Mean	SD	N
Group 1	Coronal	10.266[a]	0.931	20
	Middle	9.179[a]	1.265	20
	Apical	7.962[b]	0.927	13
Group 2	Coronal	13.201[a]	0.673	20
	Middle	12.577[b]	0.601	20
Group 3	Coronal	10.639[a]	0.547	20
	Middle	9.832[b]	0.490	20
	Apical	8.056[c]	0.839	13

DISCUSSION

To assess the bond strength between fiber posts and the root canal dentin, conventional shear and tensile tests, microshear, microtensile, and pull-out and push-out tests have been used.[21-23] It is suggested that the bond strength is better obtained by the push-out test than with the conventional shear test because the fracture occurs parallel to the dentin bonding interface in the push-out test, which makes it a true shear test.[21] The shear stress achieved with the push-out tests is comparable to the stress, under clinical conditions, at the interface between the dentin and the luting cement, as well as between the post and the luting cement.[24]

Although the main disadvantage of the use of human teeth is the relatively large variation in size and mechanical properties of the specimen, previous studies have reported that intact natural central incisors and canines are the best option to clinically simulate the treatment of endodontically treated teeth with endodontic post system.[12] The post system Rely X Fiber post used in this study (3M ESPE, Seefeld Germany) is a parallel, tapered-end glass fiber post which offers the potential advantages of reduced stress concentration at the apex, preservation of tooth structure, and allows uniform progressive increase in cement thickness and post diameter when using a standardized incremental intervals.[12,25]

It has been shown that light-activation of the dual-cured resin sealer from the canal orifice offers the advantage of establishing an immediate coronal seal, however, it blocks the pathway for stress relief.[25] Therefore, in this study, Rely X Unicem was not light cured.

The highest bond strength reported in this study was obtained when oversized post space was used (group 2), these results are in accordance with previous published studies;[16,26] this can be explained by the fact that the intimate fit between the post and root dentin in group 1 created a narrow space that didn't allow the cement to develop its maximum strength and preventing the penetration of the resin into the microporosities of the post surface.

The cement thickness values reported were significantly different between the 3 groups and between the different levels of each group; the highest push-out bond strength was obtained in group 2 at coronal level (192.22 ± 0.87 μm) and the lowest bond strength in group 1 at the apical level (27.19 ± 0.88 μm); this can be explained by the cavity configuration or C-factor that is estimated by dividing the free surface area by the total bonded area.[25,27] The thin cement layer in group 1 created an insufficient stress relief by flow and a high probability that one or more bonded areas will debond; whereas in group 2 the space provided by the oversized drill allowed the resin cement to flow resulting in stress relaxation within the resin.[12] In group 3, despite the decreased C factor leading to a more efficient stress relaxation, the bond strength was lowest than in group 2; this may be due to the incorporation of air bubbles in the bigger cement thickness weakening the resin cement by inhibiting the resin polymerization.[27,28]

Possible suggestions that the retention of thick smear layers, smear plugs and other debris on root canal walls, the residual gutta percha in areas that the preparation drill would not reach, the conical post shape at its apical end, the fastened setting of the material due to lack of oxygen in the deep narrow ends of the canal space and the adverse cavity configuration, may possibly have accounted for the lower bond strength values reported at the apical thirds of the roots.[23-28]

Table 2: Mean and standard deviation of resin cement thickness (µm) by root section. Similar superscript indicates no statistical significant difference (p< 0.05)

Group	Root Section	Mean	SD	N
Group 1	Coronal	67.26[a]	1.21	20
	Middle	39.11[b]	0.45	20
	Apical	27.19[c]	0.88	13
Group 2	Coronal	192.22[a]	0.87	20
	Middle	152.66[b]	1.51	20
	Apical	127.42[c]	0.34	12
Group 3	Coronal	310.91[a]	1.93	20
	Middle	262.27[b]	1.59	20
	Apical	220.03[c]	0.52	13

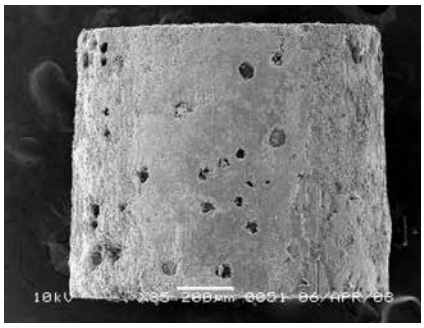
Table 3: Mean and standard deviation of resin cement thickness (µm) and push-out bond strength (MPa). Similar superscript indicates no statistical significant difference (p< 0.05)

	Group 1	Group 2	Group 3
Push-out bond strength (MPa)	9.13 [a] ± 0.6	11.7 [b] ± 0.4	9.6 [a] ± 0.7
Cement thickness (μm)	44.5 [a] ± 0.8	157.4 [b] ± 0.9	264.4 [c] ±1.1

Table 4: Distribution and percentage of failure modes in each group.

Failure mode	Groups 1	Group 2	Group 3
I	33(62%)	24(45%)	32(61%)
II	6(11%)	5(10%)	7(13%)
III	10(19%)	21(39%)	10(19%)
IV	4(8%)	3(6%)	3(6%)
(n)	53(100%)	53(100%)	52(100%)

The mode of failure evaluation showed that around 60% of the specimens in groups 1 and 3 exhibited a mode I failure at the post cement interface, these results are in agreement with other studies.[17,29] The epoxy resin matrix of Rely X fiber posts may explain the, lower interfacial strength due to the absence of chemical union between the methacrylate-based Rely X Unicem luting agent and the epoxy resin, a highly cross-linked matrix of fiber posts, which do not have any functional group available for reaction.[29] Furthermore, during the setting reaction of Rely X Unicem, the negatively charged phosphoric acid groups of the methacrylate monomers bond to calcium ions in the tooth structure. Simultaneously,



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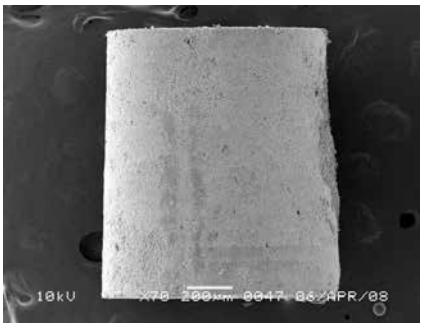

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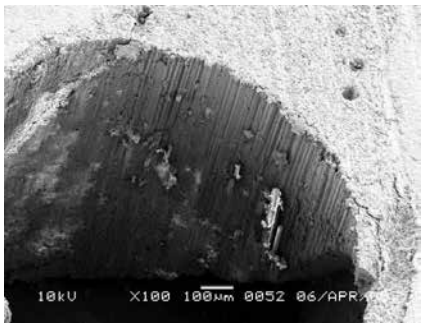

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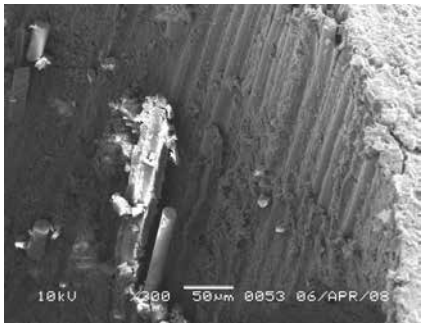

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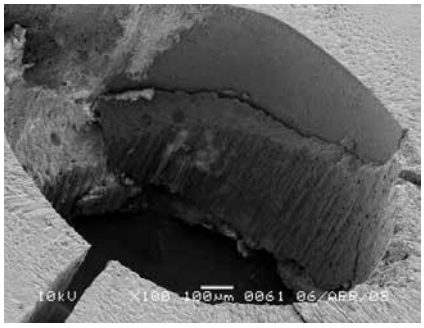

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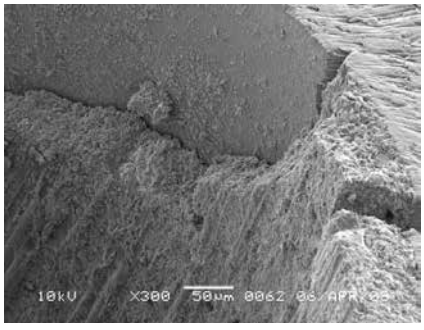

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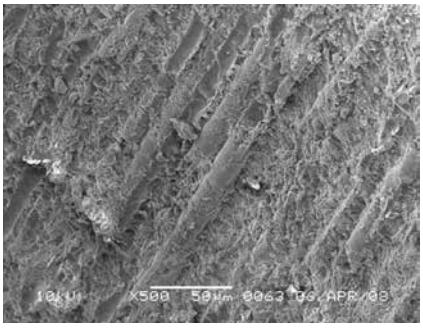
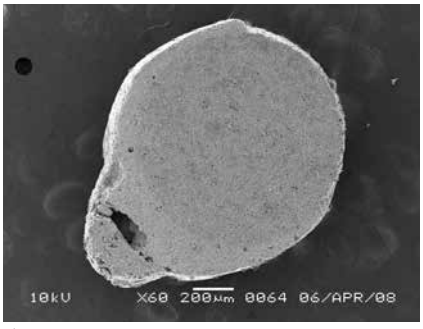


H

Fig. 1. SEM microphotographs of specimens in group 2. A. Post at the middle section. Despite the presence of air bubbles, this group yielded higher bond strength than all groups tested. Note the adhesion between the post and the cement. B. Post at the coronal section, at this level, the adhesion was too strong that fracture occured within the fiber post. C- Same coronal section as in (B) x100 magnification at the dentin interface. Some fibers were detached from the post. D. Same specimen (x300). E. Close up view (x500) some fibers that were completely detached from the post still retained in the post space. F. Another area of the resin-dentin interface (x100). G. Same microphotograph as in (F) x 300. areas of separation between the resin and dentin are shown. Note the thickness of the cement and the impression of the longitudinal fibers corresponding to the resin/post interface. H. Same micrograph as in (G) but x500. Note the surface texture of the cement corresponding to the longitudinal fibers of the post.

Fig. 2. Micrographs of specimens in group 3: A. Note the wide air bubbles within the core of the 300µm thick layer of the resin cement. Cohesive fracture in the luting agent at the proximity of the air bubble is also shown. B. Areas of separation between the cement and the post (right). Cohesive fracture within the thick layer of the cement (left).



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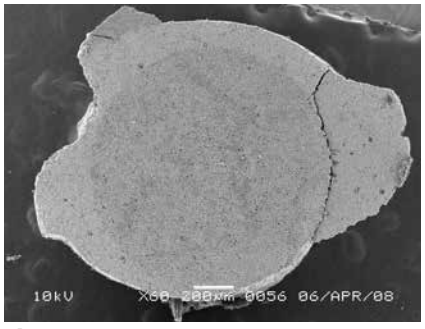

B

setting of the cement takes place through the radical polymerization reaction of the methacrylate monomers initiated by the initiator system. Successively a highly cross-linked three-dimensional network is formed, leaving no functional groups to react with the resin of the fiber post.

In Group 2, 45% of the specimens showed type I failure mode whereas 39% type III mixed fracture, with the cement covering more than 50% of the post surface. Therefore, only Group 2 exhibited higher bond strength at the post-cement-dentin interfaces. It seems that only the 190μm cement thickness obtained in Group 2 during post space preparation provided maximal strength and stress relaxation to the Rely X Unicem for the development of higher bond strengths at the post/cement/dentin interfaces.

CONCLUSIONS

Within the limitations of this in-vitro study the following conclusions can be drawn:

- The highest bond strength was obtained when one incremental oversized post drill was used
- The lowest bond strength was reported at the apical third of each group
- The average 150 μm of cement thickness is required for optimum cementation of fiber post with self adhesive resin cement

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Changes in keratinized tissue width and tissue thickness following subepithelial connective tissue grafts. A clinical and histological short-term study

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ABSTRACT

Background: There is a general consensus that keratinized gingiva is necessary for the maintenance of periodontal health in patients with suboptimal levels of plaque control although the exact amount remains controversial. While bilaminar subepithelial connective tissue grafts (SCTGs) result in augmentation of tissue thickness at recipient sites, controversial findings have been reported as to the capacity of this procedure to increase the apico-coronal height of keratinized gingival at the grafted area. The objective of this study is to, clinically and histologically, evaluate the capacity of palatal connective tissue to induce differentiation of non keratinized mucosal epithelium when SCTGs are combined with repositioned flaps in areas lacking keratinized gingiva.

Methods: SCTGs were performed in bilateral sites in 10 patients (30 mandibular teeth). The clinical parameters (width of keratinized gingiva (KGW), mucogingival junction and tissue thickness) were evaluated and compared between baseline and two months postsurgically using customized acrylic stents. Punch biopsies were obtained from the recipient sites at 2 months and processed for histological evaluation.

Results: SCTGs resulted in a mean change in KGW of 0.64±0.78 mm. Mean tissue thickness increase averaged 1.48±0.23 mm. No significant displacement of the mucogingival junction was observed. The epithelium covering the biopsies was of the non keratinized type and had mostly a flat epithelium-connective tissue interface typical of alveolar mucosa.

Conclusions: SCTGs affect the thickness rather than KGW. Histologically, palatal connective tissue grafts tended to maintain their intrinsic histologic characteristics of dense connective tissue in the short healing period following transplantation into areas of alveolar mucosa. This was not however associated with the phenotypic expression of keratinization by the overlying epithelium.

Key Words: subepithelial connective tissue graft, keratinized gingiva, keratinization, histology, mucogingival junction, tissue thickness.

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Subepithelial connective tissue (SCTG) graft-based procedures are currently considered the gold standard in the treatment of gingival recessions as they provide the best outcomes relative to the percentages of mean and complete root coverage.[1] In addition, the clinical outcome of SCTG procedures include an increase in gingival tissue thickness (GT) that is approximately 0.5 mm regardless of the harvested graft thickness.[2-7]

Ample documentation is currently available as to the increase in apico-coronal width of KG (KGW) at recession sites treated with SCTG.[1] Greater postsurgical increase of KGW has been demonstrated in recession sites treated with the envelope variant of SCTG when compared to SCTG combined with coronally positioned flap (CPF).[8-11] When SCTG is combined with CPF, the palatal connective tissue grafted beneath alveolar mucosa does not seem to induce the differentiation of alveolar mucosa surface epithelium into keratinized epithelium.[8,10,12] Several hypotheses have been proposed to justify the increase in KGW achieved clinically

following SCTG combined with coronal flap positioning: 1) reversion of the mucogingival junction to its preoperative "genetically determined" position and corresponding increase in keratinized gingival width has been proposed in several clinical studies;[13-16] 2) tendency for flap retraction/shrinkage during wound healing leaving the most coronal part of the graft uncovered;[9] 3) granulation tissues deriving from the periodontal ligament during healing of the combined procedure resulting in a connective tissue similar to the gingival connective tissue with the potential to induce keratinization of the covering epithelium;[17-19] and 4) postoperative creeping attachment20-22 with coronal migration of the gingival margin might occur over the denuded portion of the root when complete root coverage is not achieved following SCTG.

The amount of KGW increase following SCTG combined with coronally advanced flap is highly variable among published clinical studies and varies between less than 1 mm[10,11,23] to more than 3mm.[15,24] Subsequently, SCTG do not seem to represent a viable therapeutic alternative[25] when periodontal soft tissue non-root coverage augmentation procedures are considered. No documentation is currently available as to the application of SCTGs at teeth without gingival recession to increase KG width. The objectives of the present 2-month prospective clinical and histological study are to evaluate the capacity of palatal connective tissue to induce differentiation of non keratinized mucosal epithelium when SCTGs are combined with repositioned flaps in areas lacking KG. More specifically, the study compares periodontal parameters including KGW, tissue thickness, and position of the MGJ prior to and following SCTG and assesses the short-term histological healing of SCTG.

MATERIALS AND METHODS

Patient and site selection

Ten systemically healthy adult patients ranging in age between 20 and 45 years (mean age 24.7±7.2 years) planning on undergoing orthodontic therapy and requiring a non-root coverage mucogingival periodontal procedure for gingival thickness increase were selected from a patient population attending the Department of Periodontology, School of Dentistry at the Lebanese University of Beirut based on the following inclusion criteria: 1) presence of thin buccal gingival tissue in bilateral sites with less than 2 mm of keratinized gingiva; 2) absence of buccal gingival recession ≥1 mm; 3) absence of interproximal attachment and bone loss with a buccal sulcular depth ≤3 mm; 4) no intake of medications known to alter the gingival components both clinically and histologically, such as cyclosporine A, calcium channel blockers, phenytoin, etc.; and 5) smoking less than 5 cigarettes per day.

All patients were given detailed information about the objectives of the study and the procedures involved and their written informed consent obtained. The protocol was approved by the Ethical Committee of the Lebanese University of Beirut, Faculty of Dental Medicine.

All patients were subjected to initial therapy when necessary and instructed in adequate oral hygiene measures prior to surgery. At the end of the hygienic phase, all patients had to demonstrate non traumatic brushing technique and display full mouth plaque scores (FMPS) inferior to 10%.[26]

Surgical procedures and postoperative care

Before the surgical procedures were performed, a customized acrylic stent was fabricated to allow reliable and reproducible measurements at the selected sites. The stent had a groove on the mid-buccal aspect of the selected teeth in order to consistently reproduce alignment of a periodontal probe (PCP/UNC 15 probe, Nordent Manufacturing, Inc., IL, USA) serving as the measurement instrument.

Immediately prior to surgery, the thickness of the buccal gingival tissue (TT1) was recorded transgingivally by inserting an endodontic reamer fitted with a rubber disk stop through the mid-buccal gingiva until bone contact, perpendicularly to tissue surface. The rubber stop was then placed in tight contact with the gingival surface and the penetration depth measured with a caliper (Mitutoyo, UK). The measurement

was obtained just coronally to the MGJ and rounded to the nearest 0.1 mm.

The SCTG surgery was performed as follows. Horizontal right angle incisions were made into the adjacent interdental papillaes mesially and distally to the selected sites, at the level of the cemento-enamel junction of the teeth in question. A sulcular incision was made to connect the horizontal incisions and a partial-thickness dissection was then carried out extending apically at least 3 mm beyond the mucogingival junction and mesiodistally to the line angles of the adjacent teeth. Care was taken to avoid any instrumentation of the root surface.

A connective tissue graft consisting of deep connective tissue and periosteum was harvested from the canine-premolar area of the palate using a trap door approach. Graft mesio-distal dimensions were such to parallel those of the recipient site. Care was taken to remove visible adipose and glandular tissues and to create a uniform graft thickness of about 1-1.5 mm. The graft was secured in position at the level of the CEJ and sutured with the flap to the interproximal papillaes using sling or interrupted 5-0 or 6-0 monofilament sutures (Ethicon®, Johnson & Johnson Intl., St-Stevens-Woluwe, Belgium). Care was taken to completely cover the donor tissue at all sites.

Two months following healing of the SCTGs, a small 2-mm diameter biopsy was obtained interproximally within the perimeter of the grafted area, apically to the mucogingival junction and along the central papillae longitudinal axis (Fig. 1). The 3 layered cylindrical biopsy including the grafted tissue sandwiched between the periosteum/connective tissue and overlying alveolar mucosa of the recipient site was obtained using customized circular punch mounted on a slow-speed contrangle.

Following all surgical procedures, patients were prescribed analgesics (as needed) and 0.12% chlorhexidine mouthrinse 3 times daily for 3 weeks following surgery. Sutures were removed 10-15 days postoperatively. Patients were asked to avoid mechanical plaque control until healing had progressed sufficiently to allow resuming normal oral hygiene measures. Patients were recalled for oral hygiene reinforcement and professional supragingival plaque control on monthly basis.

Clinical parameters evaluated

The following clinical measurements were evaluated at all sites using the customized stents:

KGW: measured from the gingival margin to the mucogingival junction. A Schiller iodine solution was used to stain the alveolar mucosa and facilitate identification of the mucogingival junction. KGW was recorded at baseline (KGW1) prior to surgery) and 2 months postoperatively (KGW2);

location of the mucogingival junction (MGJ) measured from a reference point on the stent. MGJ location was assessed at baseline (MGJ1) and 2 months post-surgery (MGJ2):

location of the gingival margin (GM) measured for the stent at baseline (GM1) and at 2 months postsurgically (GM2);

Thickness of the buccal gingival tissue was recorded 2 months postsurgically (TT2) as described above.

All baseline and postoperative measurements were carried out by 2 calibrated experienced periodontists using a periodontal probe (PCP/UNC 15 probe, Nordent Manufacturing, Inc., IL, USA) and rounded to the nearest 0.5 mm (except for TT which was rounded to the nearest 0.1 mm).

Histological evaluation

All soft tissue specimens were immediately rinsed with sterile saline and fixed in 10% neutral buffered formalin solution for at least 24 hours. The samples were dehydrated in 5 subsequent alcohol baths (1 hour each) of ascending concentrations, cleared in 4 successive xylol baths (2 hours each), and finally impregnated in 2 consecutive paraffin baths. The tissues were paraffin embedded and sectioned step serially at levels 50 μm apart, along a plane parallel to the long axis of the biopsy core for the punch biopsies. For the histological analysis, 2 sections, 4-5 μm in thickness, representing the most central portion of the biopsy, were selected and stained with hematoxylin-eosin. The sections were evaluated for general

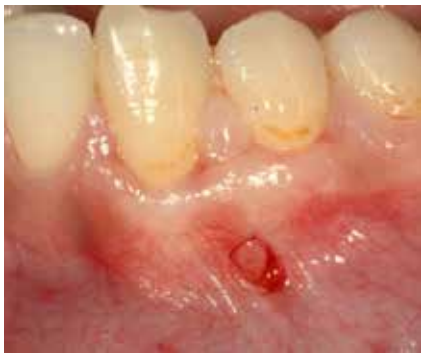
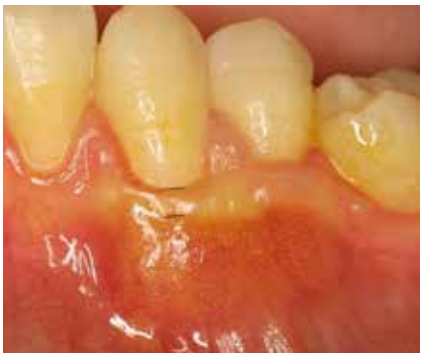


Fig. 1. Healed connective tissue graft with the circular profile of the punch biopsy.



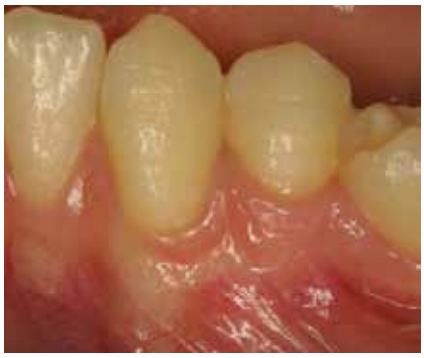


Fig. 2. Note the minimal clinical gain in keratinized tissue width between baseline (a) and post-grafting (b) at teeth 33 (+0.5 mm) and 34 (+0.5 mm) and the significant increase in tissue thickness.

Table 1. Summary of periodontal variables at baseline and 2 months post-grafting.

	Baseline Mean ± SD (mm)		2-Month Follow-up Mean ± SD (mm)	P value
KGW1	1.42±0.60	KGW2	2.05±0.85	0.007*
MGJ1	6.48±1.03	MGJ2	6.82±0.87	0.203
GM1	4.92±1.50	GM2	4.51±1.27	0.012*
TT1	0.71±0.12	TT2	2.19±0.24	0.0001*

KGW= apico-coronal width of keratinized gingiva; MGJ= mucogingival junction; GM= gingival margin; TT= gingival tissue thickness
** indicates statistical significance*

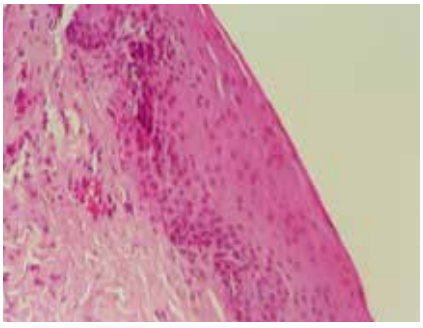


Fig. 3. Photomicrograph showing non-keratinized epithelium covering the biopsy surface (original magnification 20x).

histological tissue characteristics, orthokeratinization, parakeratinization or absence of keratinization of the covering epithelium in the core biopsies. In addition, the presence, location and amount of adipose tissue were recorded.

Statistical analysis

The mean values of all clinical parameters (KGW1, KGW2, MGJ1, MGJ2, TT1, TT2) were calculated for all patients and compared between the 2 evaluation periods (baseline and 2 months post-grafting) using the t-test. The significance level was set at $P<0.05$.

RESULTS

Clinical findings

In total, 10 patients represented by 8 females and 2 males contributed with 30 mandibular teeth requiring gingival augmentation. The selected sites included 12 mandibular canines, 14 mandibular first premolars, 4 mandibular second premolars, 1 central incisor and 1 lateral incisor. All patients healed uneventfully and were available for the follow-up evaluation. Most patients maintained good levels of oral hygiene with no clinically visible signs of gingival inflammation throughout the study. Only 1 patient in demonstrated high plaque levels at 1 month following surgery but reverted to high standards of self homecare at the 2-month postsurgical evaluation.

Baseline and 2-month follow-up data of all the clinical outcome variables are summarized in Table 1. A mean KGW increase of 0.64±0.78 mm was obtained following SCTG (KG2>KG1; P=0.007) (Fig. 2). When evaluating the outcome variable MGJ, connective tissue grafts did not result in any significant displacement of the mucogingival junction (P=0.203). In contrast, a statistically significant coronal displacement of the gingival margin averaging 0.41 mm was noted at the 2-month follow-up (P=0.012). A significant increase

in gingival thickness of 1.39 mm was observed at all sites. Tissue thickness (TT1) which was less than 1 mm at baseline (0.71±0.12 mm) increased to 2.19±0.24 mm at 2 months (P=0.0003).

Histologic Findings

Eighteen 2-month post-grafting biopsy cores were available for histological analysis. In most specimens (12 over 18), the connective tissue immediately underlying the gingival epithelium was characterized by low collagen density. The intermediate area of the recipient sites which hypothetically includes the graft tissue demonstrated a higher content in collagen fibers in 15 out of 18 biopsies, indicating that the graft tended to have a denser collagen network than that of the overlying mucosal tissue. The deepest portion of the post-grafting cores- corresponding to the residual connective tissue-periosteum remaining over the host's bony surface at recipient site preparation appeared as a dense connective tissue in 9 specimens and loose in 9 biopsies. The epithelium covering the 18 biopsies was of the non keratinized type and had mostly a flat epithelium-connective tissue interface with no rete pegs typical of alveolar mucosa (Fig. 3).

DISCUSSION

Connective tissue grafts placed under alveolar mucosa should not theoretically yield a differentiation of the mucosa epithelium into keratinized epithelium as demonstrated in histological studies[17,27] and confirmed by clinical data from various investigations.[8-10,28] In the present study, the mean baseline KGW increased from 1.42±0.60 mm to 2.05±0.85 at 2 months. These results parallel the findings of Maurer et al. (2000) who demonstrated an increase in KG averaging 1.2±1.1 mm two months following the connective tissue graft procedure. The increase in KGW in the present report can be attributed to a possible repositioning of the covering flap in an apical direction during wound healing, thus exposing the connective tissue graft.[9] Although an attempt was made to completely cover the connective tissue grafts with the gingival flap at completion of the surgical procedure in the present investigation, trauma to the flap during recipient site preparation and manipulation, and/or non-passive flap positioning may have resulted in marginal recession/ shrinkage of the gingival flap or loss of the epithelial/connective tissue integrity at the flap margin. In this case, the inductive potential of the graft on epithelial phenotype is restricted to the exposed portion of the SCTG in the short-term perspective.[8,10,28]

Overall, the results of the present study confirmed a significant coronal displacement of the gingival margin close to a half millimeter at all sites 2 months postsurgically. These observations could be related to the tendency of the clinician to position the graft and/or the recipient site flap slightly coronal to the CEJ to minimize potential gingival recession postsurgery. Creeping attachment is another mechanism that can result in postoperative coronal migration of the gingival margin in root coverage procedures.[20,29] In the present study, the SCTG was performed to increase gingival thickness in areas without recessions. It is more likely that the coronal displacement of the gingival margin had occurred as an immediate result of the surgical procedure and not as a postoperative gradual creeping.

SCTGs are routinely associated with a significant increase in gingival thickness.[2,3,6,7] From these clinical studies, it could be concluded that 50% to 70% of the applied SCTG thickness is lost during tissue maturation and remodeling. Despite the reduction in tissue thickness in the course of healing, the use of a SCTG results in significant increase in TT amounting to about half millimeter when compared to preoperative dimensions. When considering together the temporal changes in TT,2,7,30,31 it seems that the majority of soft-tissue dimensional changes that occur after initial healing occur from 3 weeks to 6 months after a SCTG procedure. Although documentation of dimensional changes of SCTG between 3 weeks and 6 months postoperatively is not currently available, data on TT changes of other soft-tissue augmentation procedures[30] suggest that significant graft thickness changes occur by 3 months postoperatively following free gingival grafts. The results of the present study

indicate that SCTG used for non-root coverage indications yield an increase in tissue thickness greater than 1 mm at all sites at 2 months postoperatively. It is likely that further dimensional changes are to be expected, mainly during the third month[30] resulting in some decrease of the TT achieved at 2 months post-grafting.

At 2 months post-grafting, the core biopsies were lined by a non keratinized epithelium. This finding suggests that the palatal tissue embedded underneath the alveolar mucosa of the recipient sites maintained its histological characteristics of dense connective tissue without affecting the differentiation of the overlying epithelium into a keratinized epithelium. This implies that transplantation of dense connective tissue into alveolar mucosa sites with loosely arranged connective tissue does not alter the original characteristics of the transplanted tissue, at least in the early healing stages. It could be speculated that the presence of a layer of connective tissue with low collagen density interposed between the surface epithelium and the grafted tissue could block the capacity of a transplanted dense connective tissue in directing the phenotypic expression of overlying epithelium.

CONCLUSION

In conclusion, the results of this short-term study suggest that SCTGs applied for non-root coverage indications in areas with thin gingival tissues result in a more significant gain in tissue thickness rather than a substantial increase in KGW. Histologically, it appears that palatal connective tissue grafts tend to maintain their intrinsic characteristics of dense connective tissue in the short healing period following transplantation into areas of alveolar mucosa. This is not however associated with phenotypic expression of keratinization by the overlying epithelium within the entire extent of the graft.

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The authors reported no conflicts of interest related to this study.

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Effect of different surface treatments on tensile bond strength between resin acrylic teeth and denture base material

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ABSTRACT

Objectives: A study was carried out to assess the tensile bond strength between resin acrylic teeth and denture base material after applying different surface treatments.
Materials & Methods: Thirty resin acrylic central incisor denture teeth were cut at the neck (ridge lap surface). These teeth were then allocated into three groups of different surface treatments: teeth in the first group received no treatment; teeth in the second group were surface treated with a groove and reinforced with a metal wire; teeth in the third group were reinforced with a glass fiber. Each group was processed by a water bath.
Results: The results showed that all treated groups improved significantly the tensile bond strength ($P<0.01$), while the third glass fiber group reported the highest mean values.
Conclusions: Higher bond strength of acrylic denture base to the acrylic teeth was obtained with a metal wire as compared to the untreated group. The addition of glass fiber can significantly enhance the tensile bond strength between resin acrylic teeth and denture base material.
Key words: Acrylic resin, acrylic teeth, glass fiber, and tensile strength.

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INTRODUCTION

Acrylic resin is the most commonly used restorative material in removable complete and partial dentures. Such materials to be polymerized require the activation of an initiator; such as benzoyl peroxide; to free radicals, in an addition polymer. The heat is usually generated either by a hot water bath or by microwaves.[1] The most commonly used method to process poly methyl methacrylate resin is a water bath.[2,3]

Plastic denture teeth are often preferred over porcelain teeth as they are easier to adjust and can bind chemically to the denture base materials. Heat-cured acrylic resins are the materials of choice for denture bases.[4,5] The failure rate of acrylic resin dentures due to fracture has been reported to be high.[6] The most common type of failure encountered was de-bonding or fracture of the teeth.[7] The lack of adequate bonding at the tooth-base interface was due to:

a- Contamination of the surface, particularly by wax and possibly by sodium alginate mold seal.
b- The difference in the structure

of the components due to their different processing routes.[8]

The failure of the bond between acrylic resin teeth and denture base material remains a significant problem. The literature indicates that the chemical and mechanical surface treatments of the denture tooth surface prior to bonding will enhance the bond strength.[9] The current study aims to evaluate the effect of different surface treatments (reinforced metal & glass fiber) on the tensile bond strength between acrylic teeth and acrylic resin denture base material.

MATERIALS AND METHODS

Materials

The materials used in this study are summarized in Table 1.

Table 1. Materials used in the study.

Materials	Trade name	Manufacture	Batch number
Acrylic teeth	-	China	1003
Hot-cure resin (liquid)	Super acryl plus	Czech Republic	Lot#RR142,B
Pink hot-cure resin (powder)	Super acryl plus	Czech Republic	Lot#RR142,B
Molding wax	Poly wax Toughened modeling wax	Turkey	12-2014
Stainless steel wire	Pigeon dental	Dentirak	Lot#130520
Reinforced glass fiber	Vetrotex –ocv reinforced	Molding Ltd.,UK	PP.6018770

Methods

Grouping of the specimens

Thirty teeth from acrylic resin were selected and randomly divided into three different groups according to the surface treatment received:

Group 1 (n=10): the teeth did not receive any treatment.

Group 2 (n=10): the teeth were surface treated with a groove preparation and reinforced with a metal wire.

Group 3 (n=10): the teeth were surface treated with a monomer and reinforced with glass fiber.

Specimen preparation

Teeth preparation

Thirty central incisors were selected and were cut at the neck (gingival portion) as follows:

A rubber mold-casting ring was used to prepare (round form) stone bases. According to the manufacturer's instructions, dental stone was mixed and poured in the rubber mold. Approximately 4mm of the incisor portion of each central incisor was embedded in the stone mixture in such a way that the gingival portion of the tooth is parallel to the horizontal plane.10The stone base with the teeth were fixed on a surveyor table by a screw. A grasping unit was designed and prepared for this study to hold a portable engine hand piece in a fixed position (parallel to the vertical arm and perpendicular to the surveyor table) that allows horizontal movement of the hand piece with surveyor arm. The surveyor arm also allows a vertical movement of the hand piece. A portable engine hand piece was fixed on the surveyor arm at constant speed (4000 rpm); using one stone disc for each tooth to cut in a one way direction to the ridge lap portion.

Mold preparation

All groups: A wax mold made form rectangular shape, dimensions of (17mm, 10mm, 7mm, 9mm, 3mm) 5 as shown in the (Fig. 1 A). The teeth were waxed on the beveled surface of a rectangular wax block. The slope of the beveled surface aligned each denture tooth so that the long axis of the tooth was at 45 degrees from the base of the wax block. (Figs. 1 B&C).

Group 1 (n=10): the teeth did not receive any additional treatment.

Group 2 (n=10): A cut was placed at the neck (gingival portion) using a fissure bur for each tooth and in one-way direction to the ridge lap portion. Next, a stainless steel wire of (9 mm) diameter and (6 mm) length was selected. Half of the wire length was fixed in the groove via adhesive material and the rest was embedded in the wax pattern.[11]

Group 3 (n=10): An electronic balance was utilized to weigh the amount of fibers that will be used in this study. (Fig. 1 D) Short fibers (0.375 grams) were added randomly to the acrylic powder and mixed together. The acrylic monomer was then added to

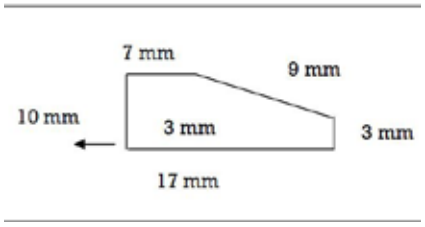

Fig. 1A. Wax mold for a rectangular wax block.

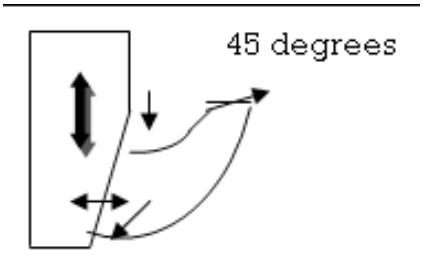

Fig. 1B. Slope of the beveled surface aligned to the denture tooth.

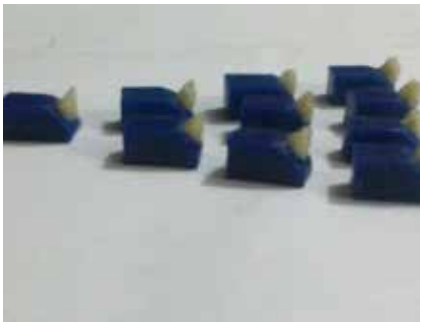

Fig. 1C. Resin acrylic teeth fixation.

Fig. 1D. Reinforced Glass fiber.

Fig. 1E. The wax pattern when inserted.

Fig. 1F. Group 2 after flasking in stone.

Fig. 1G. Groups 1 and 3 after flasking.

Fig. 1H. Specimens after finishing and polishing.

the powder and mixed using a cement spatula. When the mixture reached a dough stage, the acrylic was packed within the stone mold, cured and finished.[12]

The conventional flasking technique for complete dentures was then followed for the mold preparation in all groups. The lower half of the flask was completely filled with dental stone, which was mixed according to the manufacturer's instructions 100gm/31ml. The wax patterns were then inserted to one half of their depth (Fig. 1 E). The stone and wax pattern were lubricated with separating medium and allowed to dry. The upper half of the flask was placed over the lower half and then filled with stone. The mold was left to set for 60 minutes before moving to the next step, (Fig.s1 F&G).

Pink heat cure acrylic powder with liquid was mixed according to manufacturer's instructions 3gm/1ml. The teeth were properly positioned within the mold. When the mixture reached a dough stage, the acrylic dough was then placed inside the mold. The two halves of the flask were finally closed under pressure until metal-to-metal contact was established and left under pressure at 20 bars for 5 minutes.[13] The curing was done in a thermostatically controlled water bath at 74°C for two and a half hours and then in 100°C for a 30 minutes.[14] Heat source was then switched on and left for 90 minutes and then boiled for 30 minutes. After that, the flask was allowed to cool down in the water bath.[15] Once cured; the specimens were removed carefully from the stone mold. The samples were then finished and polished (Fig. 1 H).[16] Before testing, all specimens were stored in distilled water 37°C for 10 days.[5]

Tensile Bond Strength Test

All specimens were tested in the same day. The specimens were held in a metal fixture (Fig. 2). The metal fixture was firmly held to an Instron universal testing machine model 1195 (Instron, corporation, canton mass). The compressive load was applied with a loading testing machine at 45 degrees to the long axis of each denture tooth on the palatal surface. Tensile bond strength test was then performed using a 200 kg load cell with a crosshead speed of 0.5 mm/min with a chart speed of 20 mm/min. Specimens were loaded until they fractured. The data was recorded in kilograms. The tensile bond strength was calculated according to the formula below:

$$T.S \frac{\frac{FF}{\pi 2}}{\pi = \frac{22}{7}} \frac{XD \frac{\pi 2}{\pi} XD}{= \frac{22}{7}}$$

D (diameter) =5mm, S = 19.64 mm2

TS = tensile strength (N/mm2)

F= Force at failure (N).

S= area of cross section.17

RESULTS

All values of tensile bond strength were shown in Table 2. Results showed that the specimens without surface treatment had low tensile bond strength of 0.19 N/mm2. The specimens that were treated with reinforced metal wire had a mean value of 0.23 N/mm^2. The specimens reinforced with glass fiber showed the highest tensile bond strength value of 0.26N/mm^2. The ANOVA test indicated that there were highly significant differences between all groups as shown in (Table 3) and the least significant test (LSD) showed the statistical significance in between the 3 groups (Table 4).

Table 2. Descriptive statistics of mean distribution of tensile bond strength among studied groups.

Groups	Mean (N/mm^2)	Standard deviation	Standard error	Minimum	Maximum
Group 1 (Control)	0.19	0.021	0.006	0.16	0.22
Group 2	0.23	0.0205	0.0065	0.21	0.26
Group 3	0.26	0.0188	0.0059	0.24	0.29

Table 3. ANOVA test between groups tensile bond strength among studies groups.

	F-test	P-value	Sig
Between Groups	29.73	P<0.01	HS

Table 4. Least significant difference (LSD) test for tensile bond strength among studied groups.

(LSD) test			
Studied Groups	Group 1	Group 2	Group 3
Group 1	-	HS	HS
Group 2		-	S
Group 3			-

DISCUSSION

Many studies have been carried out to assess and improve the bond strength of acrylic teeth to denture base material. Bond strength can be affected by several factors, including cross linking of the materials, availability of the monomer, and the degree of contamination during processing. Several procedures for repairing acrylic dentures are employed by Japanese dental laboratories, to repair using wrought wire. The denture is prepared by cutting a groove at the broken area. The groove is made slightly wider than the wrought wire and deep enough so that the wire can be completely seated in the groove and fixed in it. The joint design is important in this procedure and linked to the success of the repair.[18,19]

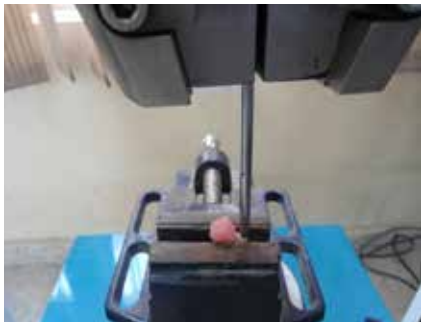

Fig. 2. specimen with chucks in place ready for testing

In this study, the bond strength between resin acrylic teeth and denture base resins were evaluated during tension. Results showed that most of the tensile bond strength is mostly around

or denture teeth or determined by them. This is best explained by the differences in the experimental design. The result of this study showed the tensile bond strength of acrylic teeth, as a function of different reinforcement to denture base resin was significantly at (Pvalue<0.01). This result is in agreement with Takahashi et al and others.[5,20,21] While our results disagree with Geert & Jooste and others.[22,23,24] A possible explanation for such differences in the results is the difference in the experimental design. The previous studies applied compression load at 45 degrees to the palatal surface of denture teeth mounted on a rectangular resin block (thickness was not mentioned). In this study the resin acrylic teeth after cutting were allocated into three groups previously mentioned. The results were statistically significant in the surface treated groups (p value<0.01), while the control group showed a non-significant difference at (P value>0.01) in improving tensile bond strength mean value as shown in (tables 2, 3, &4). This is agreement with Takahashi et al and others.[5,21, 25]

The results of the current study revealed that there were significant differences among the tested groups. The groups reinforced with a metal wire had a higher mean value of tensile bond strength than untreated group. This may be due to adding of reinforced metal wire to give support to the bond between acrylic teeth and acrylic resin denture base and gave it a higher tendency and stability to withstand higher tensile bond strength. This study agreed with Polyzios et al11 and Suzuki.[26]

The samples that were surface treated by addition of glass fiber had higher mean values of tensile bond strength when compared with control group. This can be partly explained by the presence of glass fiber in resin ensuring the transfer of the load from matrix to fiber. This will lead to an increase in the strength of the resin and allow the resin to tolerate the force of tension more than the samples that had no fiber in their structure. Adequate quantity of fiber present in resin can increase the acrylic strength. This is in agreement with Ehasn[12] and disagreement with Polyzios et al.[11]

CONCLUSION

The following conclusions can be drawn:

1. A higher bond strength of acrylic denture base to the acrylic teeth was obtained with metal wire as compared to untreated group.

2. The addition of glass fiber can enhance the tensile bond strength.

Aknowledgments:
The authors reported no conflicts of interest related to this study.

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Musculoskeletal disorders among Lebanese dentists

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ABSTRACT

Objective: Dentists are exposed to musculoskeletal disorders (MSD), which are related to the work environment. The objective of this survey is to determine the prevalence of MSD among dentists in Lebanon.

Material and Methods: A questionnaire was distributed by the authors at the 23rd Beirut International Dental Meeting. The recorded data was analyzed with SPSS 20. P-value< 0.05 was considered to be statistically significant.

Results: 219 dental practitioners participated in the study of which 92.7% suffered from MSD and 40% had a prevalence of at least one MSD symptom over the past twelve months. Most common areas affected by MSD in order of magnitude were lower back (61.8%), neck (51.5%), shoulders (39.5), fingers (14.1%), wrist (11.8%), and elbow (8.6%). Females had a higher frequency than males. Permanent Pain was inversely proportional to the physical activity.

Conclusions: High prevalence of MSD exists among dental practitioners affecting the daily practice. Further long-term studies are needed to identify the specific risk factors for MSD so as to introduce effective remedial measures.

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INTRODUCTION

The World Health Organization (WHO) and United Nations recognize musculoskeletal disorders as the most common causes of long-term pain and disability which affect hundreds of millions of people.[1] Literature review across the world has shown a high prevalence of MSD among dentists.[2] A study conducted in New Zealand reported the prevalence of MSD in the neck region among dentist population as 57%, and in the lower back and shoulder as 52%.[3] Similar results were found by Thornton et al among dentists in US.[4] In Western countries, due to its high prevalence, the awareness of MSD in the dental profession has increased.[4] However, in China, where the population amounts to about 1.3 billion people, there is a lack of research about the prevalence of MSD among dentists.[5] In India as well the prevalence of MSD among dental practitioners is not well documented.[2]

Dental professionals spend their work day in an awkward, static position preforming precise procedures in a small workspace, the patient's mouth.[6] MSDs are characterized by the presence of

discomfort, disability or persistent pain in the joints, muscles, tendons, and other soft parts, caused or aggravated by repeated movements and prolonged awkward or forced body postures.[2]

The key of prevention is good posture during an activity. A good or "neutral" posture means that the joints are being used near the middle of their full range of motion.[7]

Dentists frequently assume static postures that require contraction of muscles to hold the body motionless while resisting gravity.[6] The further away from neutral position; the more strain is put on the muscles, tendons and ligaments around the joint.[7]

This survey will focus on the prevalence of MSD among dentists by specialty, highlighting how lifestyle such as doing physical activity could influence the frequency of pain, and can deduce if Lebanese dentists are well informed or not about ergonomic posture and lifestyle needed to avoid MSD.

MATERIALS AND METHODS

A pilot study was performed on five dental practitioners before commencing the study. This questionnaire recorded the prevalence of MSD in terms of musculoskeletal symptoms (location of pain in the neck, shoulders, lower back, fingers, elbow and wrist) and the frequency of pain (permanent, occasional, or rare) in the preceding 12 months.

A cross sectional descriptive study was then conducted from the 26th of September till the 3rd of October 2013 to assess the prevalence of MSD among dental practitioners in Lebanon. The purpose of the study was explained to the participants. The authors distributed questionnaires at the 26th Beirut International Dental Meeting.

The method for answering was explained and the questionnaire was filled and returned in the same day. The recording included demographic variables such as age, gender, years of experience, specialty of the participants, duration of practice (working hours per day, working day per week), and whether the subjects were using indirect vision or direct vision, their position of work (sitting, standing or both). (Fig. 1)

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Introduction: Ce questionnaire vise à récolter les informations concernant les conditions de vie des praticiens en chirurgie dentaire, et leurs effets sur le risque de développement des troubles Musculosquelettiques "TMS".

1- Age: _____
2- Sexe: F____; M_____
3- Expérience: _____
4- Spécificité de l'exercice:
- Dentisterie général: ---------
- Chirurgie: --------- - Parodontologie:_____
- Endodontie: -------- - Pédodontie:_____
- Orthodontie: --------- - Prothése:_____

5-Nombre d'heures de travail en moyenne par jour: ---------
6-Nombre de jours de travail par semaine:----------
7-Modalités de l exercice:
- Vision directe: -------
- Vision indirecte: ------

8-Position de travail:
- Debout: -------
- Assis: --------
- Alternance de position debout et assis: _____

9-Cible des douleurs:
- Doigts:_____
- Poignets:_____
- Coude:_____
- Cou: _____
- Epaules:_____
- Bas du dos:_____

10- Fréquence des douleurs :
- permanentes:_____
- occasionnelles:_____
- rares:_____
- jamais ressenties:_____

11- Activités sportives:
- Tennis:_____
- Marche:_____
- Jogging:_____
- Natation:_____
- Musculation: _____
- Stretching:_____
- Aucune activité physique:_____

Fig. 1 the questionnaire that was distributed at the 23rd Beirut International Dental Meeting.

Statistical Analysis: Statistical package for social sciences (SPSS) version 20 was used to determine frequency distributions, means and proportions. Comparison of proportions was done using Fischer's exact test. P-value <0.05 was considered to statistically significant.

RESULTS

Out of the 230 questionnaires distributed, 219 were available to participate in the study. 11 practitioners refused to participate. Demographic details are shown in table 1. The majority of participants were males (68%), the mean age of the participants was 43. The major work by general dentists is restorative, prosthodontics, in addition to other services. General dentists were accounted to 67% of the studied subjects and remaining were specialists. The average duration of practice was 18.6 years. Most of the practitioners work 7 hours per day and 5 days per week. 64% of dentists work in the seated position and 52% use both of direct and indirect vision. There is no statically significant of MSD by age, years of practice. Working hours per day seem to have relation with neck pain.

Prevalence of MSD: Overall 40% of the practitioners had a prevalence of at least one MSD symptom. The most common areas affected with MSD in order of magnitude were low back (61.8%), neck (51.4%), shoulders (39.5%), fingers (14.1%), wrist (11.8%), elbow (8.6%). (Fig. 1)

Prevalence of MSD in the involved area was higher for female dentists than males. Females suffer much more from males in both area neck and wrist.

Table1. Distribution of Dentists by demographic characteristics in relation to the prevalence of MSD.

	N	%
Gender		
Male	147	67.4%
Female	71	32.6%
Total	218	100.0%
Specialty		
General Dentistry	144	67.3%
Surgery	19	8.9%
Endodontic	5	2.3%
Orthodontic	28	13.1%
Pediatric Dentistry	6	2.8%
Periodontology	12	5.6%
Total	214	100.0%
Vision		
Direct	86	39.4%
Indirect	18	8.3%
Both	114	52.3%
Total	218	100.0%
Posture		
Sitting	138	64.5%
Standing	11	5.1%
Both	65	30.4%
Total	214	100.0%

Age: Mean (SD); Min-Max 43.1(10.7); 22- 70
Years of experience: Mean (SD); Min-Max 18.7 (9.8); 1- 45
Daily Working Hours: Mean (SD); Min-Max 7.4 (1.98); 2- 12
Weekly Working Days: Mean (SD); Min-Max 5.2(0.82); 2-6

DISCUSSION

Published literature has shown a high prevalence of MSD among dentists.[8,9] This has been due to the prolonged static postures, repetitive movements, vibrations and force, which are considered as risk factors for MSD.[8-10]

To avoid musculoskeletal disease, dentists must respect the ergonomic rules. MSD pain is often the direct result of the body positioning and movements made by dentists in their daily work.

There are many studies

concerning MSD among dentists. A targeted look at the upper, neck, shoulders, and wrists are common in all studies.

When dentistry changed from a standing job to seated one, the prevalence of pain in the neck and shoulder became more pronounced.[11] Although this change in posture is not the focus of our study but this survey shows that Lebanese dentists suffer more from back pain than other areas. Also musculoskeletal pain in the lower back remains a constant cause of loss of work for dentists.[11]

In a study by Biller, 65% of dentist surveyed, complained from a back pain.10 A study by McGill et al reported that 81% of dentists surveyed, complained from neck, shoulder, wrist and back pain.[12] This one shows that 61.8% of Lebanese dentists (experienced and non experienced dentists) reported a back pain. The prevalence of back pain in Australia is (64%), Queens Land Australia (53%), Denmark (50%), in Teheran (32.3%).[13]

Some studies show that General practitioners tend to be predisposed to neck and lower back pain due to their prolonged static postures and fewer repetitive motions while working.[2] Other studies have shown that Orthodontists have predominantly lower back pain because of the repeated forward positioning of head and bending of low back during clinical procedures.[5] The present study shows the highest prevalence of back pain is among pediatric and general dentists.

These examples show how switching to a seated position has made little to no difference with the frequency of pain among dentists in the lower back region.

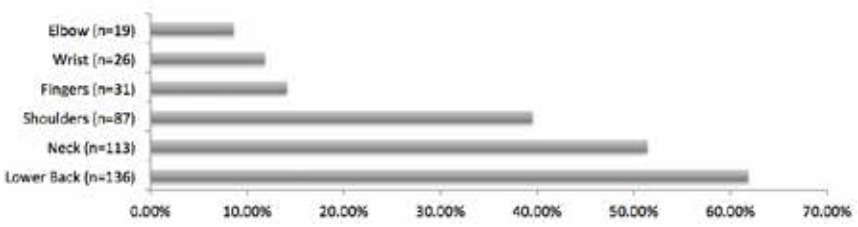

Fig. 2. Prevalence of MSD in relation to the area involved.

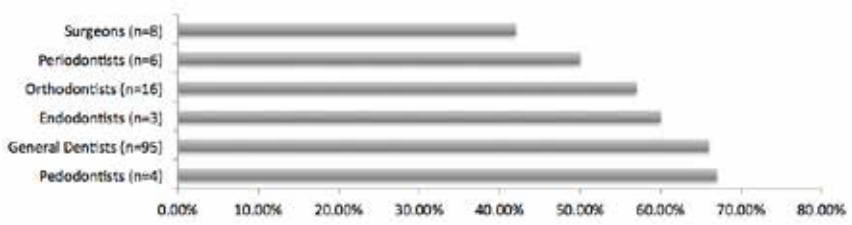

Fig. 3A. MSD in the low back area is the highest among pediatric dentists and general dentists.

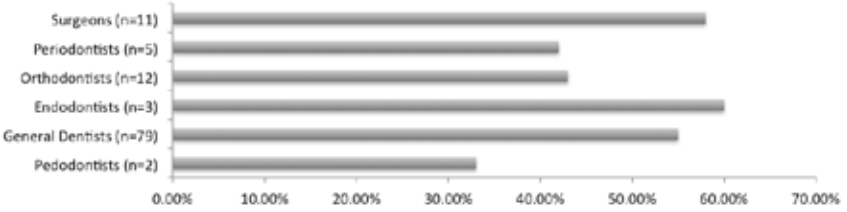

Fig. 3B. MSD in the neck area is the highest among endodontists and surgeons.

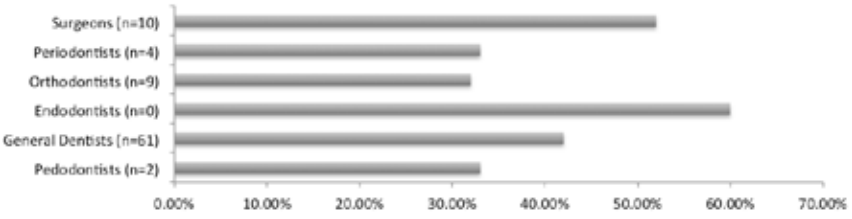

Fig. 3C. MSD in the shoulder area is the highest among surgeons and general dentists.

Another study was conducted to determine the prevalence of neck, shoulder, arm and hand discomfort in dentists and pharmacists. 51% of dentists compared to 23% of pharmacists reported shoulder symptoms. 44% of dentists and 26% of pharmacists suffered from neck pain. This indicates that shoulder-neck discomfort is more among dentists than pharmacists.14In this survey neck pain represents 51.4% among Lebanese dentists specially

females and endodontic dentists. Some international comparison shows a high prevalence of neck pain among dentist, for example in Queensland Australia (57%), in Denmark (65%), in Saudi Arabia (65%), Iran (47%).[13] A survey from the hospitals in Guangzhou, China shows that neck pain was the most prevalent musculoskeletal symptom in the 12 month period, as reported by 83.8% of the surveyed dentists.[15]

In this study the frequency of pain in the shoulder area was 39.5%, the most percentage of dentist who reported shoulder pain, were surgeon dentists. This finding is similar by dentists in China; the frequency of shoulder pain was permanent by surgeon Chinese dentists and represented 83.3% of MSD.[5] Other studies showed that Periodontists and dental hygienists are predisposed to neck, shoulder, and wrist pain due to the static postures combined with forceful, repetitive movements while performing procedures.[2]

There was not a big difference in predilection of MSD between specialist and general dentists. This lack of difference could be attributed to the fact that in Lebanon general dentists perform a wide range of services as part of their practice.

This study showed a higher prevalence of MSD among females, which is similar in Lithuania, New Zealand, and India.[2] This was attributed to the fact that women were more concerned about their health compared to men and tend to

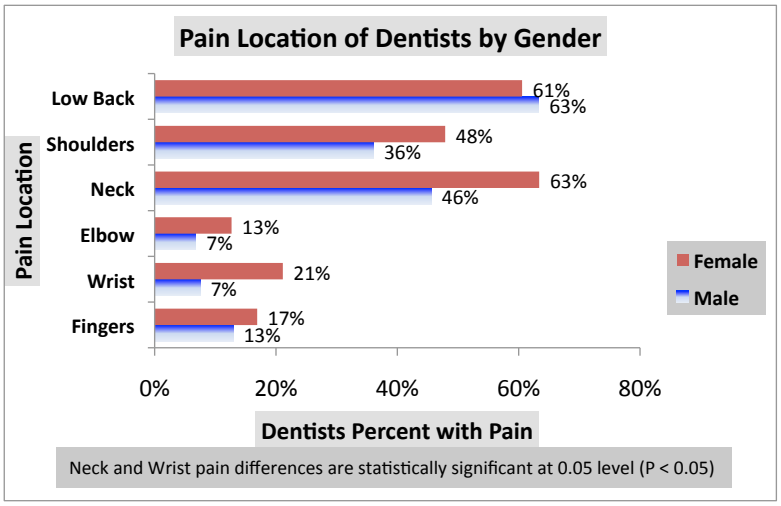

Fig. 4. Area wise predilection of MSD in relation to gender of the dental practitioners. Prevalence of MSD in the involved area was higher for female dentists than males. Females suffer much more from males in both area neck and wrist.

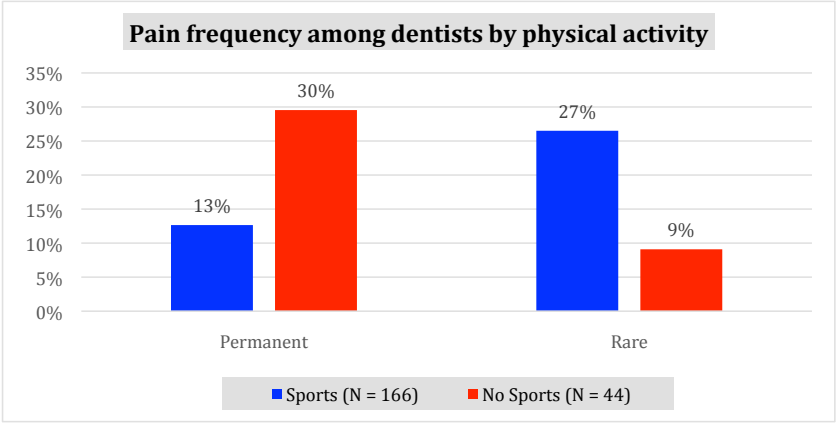

Fig. 5. Pain frequency among dentists by physical activity: the frequency of pain is decreasing with physical activities. The relation between pain and physical activities is inversely proportional.

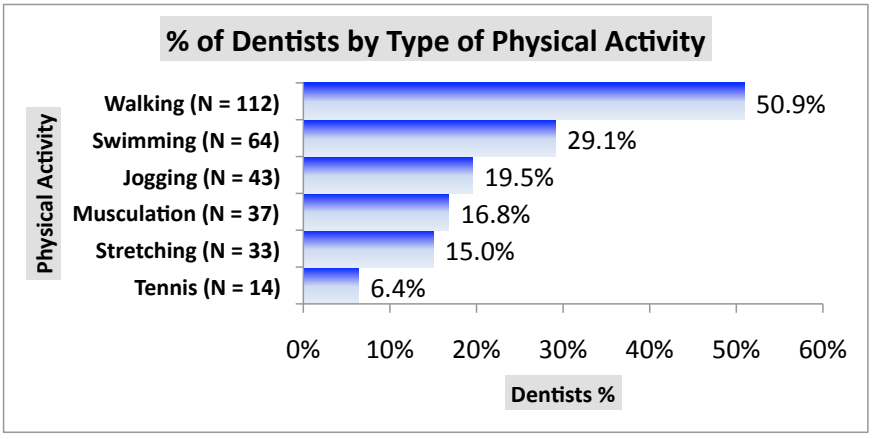

Fig. 6. Physical Activity among dentists. 50.9% of dentists choose walking as physical activity and 29% swimming.

report health problems more often.[2]

In this survey we found that daily working hours were significantly associated with the presence of neck pain. This finding is the same by Chinese dentists.[5]

Overall, this study demonstrates that there is a high percentage of MSD symptoms 92.7%, depending on age and years of experience. This can show that Lebanese dentists don't have the knowledge in preventing and managing work-related musculoskeletal problems in their practice.

RECOMMENDATIONS

Some strategies must be learned and respected in the dental practice such as:

Avoid static postures: dentists should vary their work positions to shift the workload from one group of muscles to another.[16,17]

Alternate between standing and sitting: this alternating let one group of muscles rest, while the workload is shifted to another group of muscles.[18]

Reposition the feet: the changes in foot position can also shift the workload from one group of low back muscles to another.[19]

Position patients at the proper height: generally, patients should be placed in a semi supine position for mandibular and maxillary procedures.[19]

Avoid twisting: dentists should position instruments within easy to reach and avoid twisting or reaching across the body. Repeated twisting in one direction may result in muscle imbalances leading to low back pain.[13]

The use of loupes and surgical telescopes are able to maintain a neutral working posture while increasing their visual acuity, level of motor control, and diagnostic ability. [5]

In dentistry, stress is also a factor of MSD because stress can elicit muscular contraction and pain, especially in the trapezius muscle. Physical activity is primordial to reduce stress and decrease stress-related muscular tension.[6] This survey shows that when dentists are used to do physical activity, the repercussion of MSD becomes rare. A survey in china has also identified a significant association between regular exercise and lower occurrence of neck pain.[5]

Stretching can be incorporated into daily routine. It increases blood flow to muscles, increasing production of joint synovial fluid, reducing formation of trigger points, increasing nutrient supply to vertebral disks.[6] Mcclean et al reported that by complying with regularly scheduled micro breaks, the subjects had less discomfort.[17] Other authors have also pointed out to the efficacy of using micro pauses and stretching during dental procedure.[5,20]

Dental operators can be taught to manage and prevent injuries effectively. They can educate themselves and their staff members by using preventive approaches before painful episodes occur.

CONCLUSION

Overall, this review demonstrates that MSD represents a significant barrier for the dental profession. There is a 92.7%, of MSD symptoms in the studied population, depending on age and years of experience. Promoting prevention programs regarding ergonomic postures must be a necessity among dentists during their clinical practices.

Aknowledgments:
The authors reported no conflicts of interest related to this study.

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Difference in attachment loss between grooved and non-grooved root surfaces of mandibular premolars

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ABSTRACT

PURPOSE: Radicular grooves have been demonstrated to have a significant impact on periodontal attachment loss. The purpose of the present study is to compare the difference in attachment loss between grooved and non-grooved root surfaces of mandibular premolars.

Material and Methods: 30 freshly extracted and periodontally involved first and second mandibular premolars were selected for this study. Loss of attachment was measured from the CEJ to the most coronal level of stained periodontal ligament on the mesial and distal radicular surfaces. Comparison of attachment loss between interproximal grooved and non-grooved surfaces was performed using the paired t-test.

Results: Mean attachment loss on the grooved and non-grooved root surfaces was 8.4±1.9 mm and 6.4±1.7 mm respectively. The difference was statistically significant (P<0.001). Groove position on the root surface was directly related to the location and maximum attachment loss.

Discussion and Conclusions: The present study demonstrates the negative effect of proximal root grooves on attachment loss in mandibular premolars and highlights the importance of radicular anatomy in site-specific periodontal attachment loss.

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INTRODUCTION

Abundant evidence implicates microorganisms as the primary etiologic factor in various forms of periodontal diseases. Local anatomic and developmental variations and abnormalities of root anatomy are considered important co-factors in periodontal disease process.[1-6] The significance of these root features has been emphasized by The American Academy of Periodontology which added to its classification the category "Acquired Deformities and Conditions"[7] that includes anatomical tooth factors. Root characteristics- such as root concavities and grooves, cervical enamel projections and enamel pearls and others- may compromise patient's self care, interfere with the accessibility for adequate root surface instrumentation[8,9] and affect the therapeutic outcome.

Proximal root concavities are among the most frequent and significant root abnormalities. The significance of root grooves in periodontal attachment loss has been demonstrated in several studies. In a clinical investigation,

Bačic et al[10] reported that grooved teeth presented significantly higher plaque, gingival and periodontal disease index scores than non-grooved teeth. A significant positive association has also been demonstrated between the presence of palato-gingival grooves, a specific anatomic abnormality of the maxillary incisors, and the frequency of probing depths greater than 4 mm.[11] In addition to the presence of grooves as a risk factor for site-specific disease, root groove morphology has been positively associated with the severity of attachment loss.[5]

In addition to their effect on plaque accumulation and progression of attachment loss, radicular grooves can also have a significant impact on the outcome of non surgical and surgical periodontal therapy. Badersten et al[12] reported that following non surgical therapy in non-molar teeth, proximal surfaces with root grooves had a lower incidence of probing attachment gain and a higher incidence of probing attachment loss than sites without concavities. Furthermore, since more cementum tend to be present over root concavities than over the adjacent convexities,[13,14] prolonged contamination of thick cementum in radicular concavities has been suggested to affect the success of adequate root surface debridement.[15] In periodontal regenerative therapy, the adaptation of expanded polytetrafluoroethylene (ePTFE) membranes against root trunk concavities of multirooted teeth was demonstrated to be poor and to interfere with adequate coronal sealing of the membrane against the root surface resulting in scarce regenerative outcome.[16] In a 6-month clinical study, the results of guided tissue regeneration in maxillary premolars with infrabony defects were not superior to open debridement without membrane.[17] This could be attributed to the presence of deep interproximal root concavities on the premolars which may annul the membrane effect.

Knowledge of root anatomy is therefore a critical element influencing clinical decisions relative to diagnosis, treatment, maintenance, and prognosis of individual teeth.[18] While abundant information has been published relative to root concavities in the anterior teeth, molars and maxillary first premolars, limited data is currently available relative to the role of root concavities in periodontal attachment loss in mandibular premolars. The aims of the present study are to evaluate the relationship between root concavity and periodontal attachment loss in mandibular premolars extracted for periodontal reasons and assess differences in attachment loss between grooved and non-grooved root surfaces.

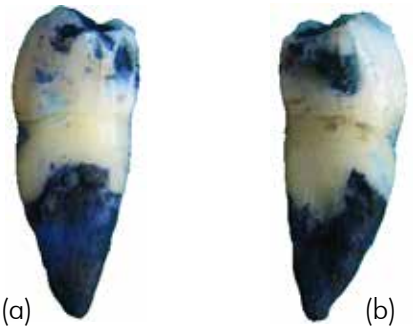

Fig 1. Photographs of the mesial and distal surfaces of a mandibular second premolar illustrating the difference in clinical attachment loss between the grooved (a) and non-grooved (b) surfaces in the same tooth. The greatest loss of attachment occurred in relation to the groove longitudinal axis.

MATERIALS AND METHODS

Tooth selection and root preparation

Thirty periodontally involved first and second mandibular premolars, freshly extracted for periodontal reasons, were selected for the study based on the following inclusion criteria:

- presence of a groove on one of the proximal surfaces and a flat or rounded surface on the other proximal aspect
- intact cemento-enamel junction (CEJ)
- absence of restorations or caries extending beyond the CEJ
- absence of visible damage resulting from the extraction procedure

The teeth were gently scaled and cleaned under running tap water and stained with 0.1% toluidine blue for 10 seconds to allow periodontal attachment visualization.

Evaluated parameters

Loss of attachment was measured along the long axis of the root from the CEJ to the most coronal level of the stained periodontal ligament on both mesial and distal aspects of all teeth using a digital caliper (Mitutoyo, Japan) up to 0.1 mm. On the grooved surface, the measurement was made along the axis of the groove. A corresponding measurement was recorded at the middle of the opposing flat or convex surface. All measurements were performed by one calibrated examiner.

Statistical analysis

The t-test at $P<0.001$ was used to evaluate the difference in attachment loss between grooved and non-grooved root surfaces.

RESULTS

Mean attachment loss on the grooved and non-grooved root surfaces was 8.4 ± 1.9 mm and 6.4 ± 1.7 mm respectively (fig. 1). The difference was statistically significant ($P<0.001$). Groove position on the root surface was directly related to the location and maximum attachment loss.

DISCUSSION

The present study evaluated by direct measurements on 30 mandibular first and second premolars, extracted for severe periodontal bone loss, the possible influence of anatomic proximal root grooves on attachment loss. The impact of proximal concavities was confirmed by the significant difference in attachment loss between grooved and non-grooved surfaces. These results are in line with the findings of previously published reports.[5,11,19,20] Furthermore, groove location was directly related to the location and maximum attachment loss, in accordance with the conclusions of Leknes et al[5] in maxillary and mandibular single-rooted teeth.

CONCLUSION

In conclusion, the present study emphasizes the need to recognize proximal root concavities of mandibular premolars in the periodontal management of periodontally involved patients as they may influence the site-specific progression of periodontal attachment loss.

Aknowledgments:

The authors reported no conflicts of interest related to this study.

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Correlation between the level of salivary immunoglobulin A and mutans streptococci among Lebanese preschool children with early childhood caries

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ABSTRACT

Background: Early childhood caries (ECC) is a public dental health problem that has been affecting preschool children all over the world. Its prevention must be a priority for all professionals in the medical and dental community. We questioned if the immune system impacts the development of ECC in children with poor eating habits. Method: Fifty nine Lebanese preschool children with similar alimentation were divided into 2 groups: Group 1 comprised children with no cavities and group two included children with ECC. We measured the mutans streptococci (SM) and Immunoglobulin A (IgA) levels collected from saliva and dental plaque and compared them between the groups. Results: There was a significant difference in SM levels between the 2 groups ($p<0.001$). There was a marginal correlation between salivary total IgA and SM collected from dental plaque ($r=0.33$, $p=0.077$) and an absence of correlation for the salivary SM ($p=0.35$).

Conclusion: In our patient population, we failed to find a significant relationship between salivary markers of immune system and the development of ECC. Further studies should be investigated. Fighting ECC remains in its prevention.

Keywords: Early childhood caries, streptococcus mutans, immunoglobulin A, prevention.

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INTRODUCTION

Saliva plays an essential role in maintaining a good oral health. It controls metabolism, adhesion and proliferation of local microorganisms, improves the elimination of residues of carbohydrates, ensures the remineralization of teeth and neutralizes the organic acids produced by cariogenic microorganisms causing the demineralization of tooth enamel.[1] Despite the reduction in the prevalence of tooth decays in the recent years, it remains a major problem in many developing countries[2-4] especially in young children where it is known as early childhood caries (ECC).[5,6] In Lebanon, according to a study by Chedid et al in 2011, more than half of the children aged between 1 and 4 years present a high risk of caries with 74.7% having at least one decay.[7]

ECC is a special form of carious lesions.8 It is encountered in patients where the food mode is characterized by bottle and breast-feeding in an inappropriate way.[3,5,9] In 2008, Caplan et al. defined "inappropriate feeding" as the frequent use of a bottle or glass containing milk or other

sweetened drinks before and after meals or at bedtime.[10]

The infectious nature of caries in general and ECC in particular implies a potential involvement of salivary antimicrobial components in the phenomenon of pathogenesis.[11-13] However, studies about the correlation between these immunological factors (immunoglobulin G, M, A) and tooth decay in children are inconclusive, with a variety of results based on different methods of sampling and assay used.[14-16]

The American Academy of Pediatric Dentistry considered, in a report published in 2003, that the presence of any decayed deciduous tooth, missing or filled teeth in children aged less than seventy-one months, is a sign of ECC.[17,18] The answer to the question "what causes this condition?" is important and complex. The risk factors involved in ECC have been widely described in the literature.[6,9,19-24]

The objective of our study was to clarify the role of the salivary immune system in ECC, and its possible correlation with the rate of salivary streptococcus mutans (SM) and the number of cavities in a sample of young Lebanese preschoolers. In other words, we tried to provide an answer to the question: "why for a group of children from the same environment with the same eating habits, some present early childhood caries, while others do not?"

MATERIALS AND METHODS

The sample consisted of 59 children (30 boys and 29 girls) aged between 18 and 49 months. They were recruited randomly from nurseries and dental offices. The inclusion criteria were: absence of antibiotic intake for the last two weeks at least, good health, no previous dental treatment and inappropriate diet, consisting of a frequent use of bottle or glass containing milk or other sweetened drink before and after meals or to bedtime. Before any dental and immunological examination were performed, an informed consent was signed by the parents.

A six-part questionnaire was completed for each child by one of the parents concerning his/her identity, siblings, socioeconomic background, medical and dental history.

The dental clinical examination was performed by a single operator and was limited to a visual examination of the teeth using a mirror and a probe at daylight. Decayed teeth were recorded. No radiological examination was performed.

After dental examination, children were divided into two groups depending on whether they have ECC (caries group) or not (no caries group).

The SM Dentocult Strip Mutans test 25,26 was used to detect SM in the saliva and plaque. This method is based on the ability of the microorganisms to adhere and grow on strips delivered with the kit after incubation. Using a sterile probe, the plaque was removed from the interdental surfaces and gently spread over the ribbed strip. Plaque collections were denoted by P1, P2, P3 and P4 corresponding to the teeth number 51, 61, 64, 84 respectively.

Saliva was collected by impregnation. After an incubation period of forty-eight hours at a temperature of 37 degrees Celsius, the scores of SM were recorded by comparing the culture media to a charter issued with the microbiological test kit.

The N-latex IgA test was carried out in the laboratory of chemistry at the American University of Beirut Medical Center. After the collection of all the samples, the tubes were analyzed in a machine called "nephelometry" (the nephelometry technique is a test that quickly and accurately measure the serum concentration of antibodies .

For each variable P1 to P4 and S, the children were classified by categories: "low score" category if the value of the variable is between zero and one and the category "high score" if the variable is equal to two or three.[27]

Data analysis

Descriptive statistics consisted of means and standard deviations of the variables: age, IgA, number of caries and proportions of the variables: gender, saliva (S) and plaque (P1 to P4). Since IgA and Age did not follow a normal distribution, the test rank sum Wilcoxon test was used to compare these variables between both groups. In addition, the Spearman correlation coefficient was applied to the variables in each group. The chi-square test or Fisher's exact test was used

to determine the relationship between the different categories (gender, P1 to P4, S) and the presence or absence of caries with bar charts. All analysis was done using SPSS version 16. A 5% level was used to declare significance and a 10% level was used to declare marginal significance.

RESULTS

Table 1 contains summary statistics for each group along with the comparisons between the groups. There were 13 (43.3%) girls in the control group and 16 (53.3%) girls in the "caries" group. Gender difference was not statistically significant (p=0.438). The average age (29.3) in the control group was significantly lower than that (39.7) in the "caries" group (p<0.001).

Although the "caries" group had a higher average IgA than that of the control group (23.6 vs, 30.4), the difference was not statistically significant (p=0.17). As for S and P1-P4, we found that most people scored in the lower range (0 or 1) on those variables in the control group and in the higher range (2 and 3) in the "caries" group. The differences between the two groups were significant for all those variables. Figure 1 supports the latter result. Moreover, the average P was significantly higher (p<0.001) in the "caries" group (average=2.6) as compared to the control group (average=1.1).

In both groups, there was no significant correlation between IgA and Age (p=0.619 for the control group and p=0.849 for the "caries" groups). In the control group, there was no significant correlation (p=0.94) between IgA and average P, however in the "caries" groups the correlation was positive (r=0.33) and marginally significant (p=0.077). As for the number of caries, it was not significantly correlated (p=0.633) with IgA. It is worthy to note that there was also a marginally significant (p=0.064) positive correlation (r=0.343) between the number of caries and Age. The results of comparing IgA among children with low vs. high P1-P4 in the "caries" group are summarized in table 2. It can be seen that for all the variables the mean IgA was always higher in the high category as compared

Table 1. Variable comparisons between the two groups

Variable		No Caries (N=30)	Caries (N=30)	p-value
Gender [n(%)		13(43.3)	16(53.3)	0.438
[(female		29.3(5.4)	39.7(7.0)	<0.001
Age				
IgA		23.6(17.9)	30.4(47.8)	0.17
S				<0.001
	0	21 (70.0%)	0 (0.0%)	
	1	2 (6.7%)	1 (3.3%)	
	2	6 (20.0%)	19 (63.3%)	
	3	1 (3.3%)	10 (33.3%)	
P1				<0.001
	0	10 (33.3%)	1 (3.3%)	
	1	8 (26.7%)	2 (6.7%)	
	2	3 (10.0%)	2 (6.7%)	
	3	9 (30.0%)	25 (83.3%)	
P2				<0.001
	0	8 (26.7%)	0 (0.0%)	
	1	7 (23.3%)	1 (3.3%)	
	2	8 (26.7%)	6 (20.0%)	
	3	7 (23.3%)	23 (76.7%)	
P3				<0.001
	0	16 (53.3%)	0 (0.0%)	
	1	4 (13.3%)	2 (6.7%)	
	2	4 (13.3%)	8 (26.7%)	
	3	6 (20.0%)	20 (66.7%)	
P4				<0.001
	0	20 (66.7%)	0 (0.0%)	
	1	4 (13.3%)	6 (20.0%)	
	2	4 (13.3%)	12 (40.0%)	
	3	2 (6.7%)	12 (40.0%)	
Average P		1.1 (0.9)	2.6 (0.4)	<0.001

to the low category. However, marginal significance was only seen for P1 (p=0.085) and P4 (p=0.095). In figure 2 we plotted the means for each the variable for the two categories and it confirmed our results above. A similar comparison was done for the control group and none of the variables reached statistical or marginal significance (table 2).

As for S, there was no significant difference in IgA between the "low" and the "high" S categories in both of the groups (p=0.35 for the "caries" group and p=0.933 for the "control" group).

The difference between genders was not statistically significant, as we obtained 43.3% of girls in the control group and 53.3% of girls in the affected group (p = 0.438).

As for the age of the children, there was a statistically significant difference between the control group (29.3 months) and the ECC group (39.7 months) with a p < 0.001.

Table 2. Different levels of the average of the total IgA (mg/l) corresponding to low and high scores for the variables: P1 to P4 and saliva (S) in the "caries" group.

Variable	Group	Total IgA (mg/l) Mean (SD)	P value
P1	Low score High score	14.4 (8.5) 33.6 (10.3)	0.085
P2	Low score High score	22.6 (9.3) 32.8 (11.1)	0.924
P3	Low score High score	24.4 (7.4) 33.4 (12.7)	0.983
P4	Low score High score	18.9 (5.0) 47.6 (20.0)	0.095
S	Low score High score	34.9 (12.4) 21.4 (8.5)	0.35
	Low score		

Table 3. Different levels of the average of the total IgA (mg/l) corresponding to high and low scores for the variables: P1 to P4 and saliva (S) in the "control" group.

Variable	Group	Total IgA (mg/l) Mean (SD)	P value
P1	Low score High score	21.5 (3.1) 28.2 (8.4)	0.618
P2	Low score High score	20.6 (3.0) 33.3 (9.7)	0.161
P3	Low score High score	22.4 (2.7) 28.0 (13.0)	0.601
P4	Low score High score	24.4 (3.4) 11.8 (13.0)	0.255
S	Low score High score	23.64 (3.4) 21.00 (--)	0.933
	Low score		

DISCUSSION

The comparison of two populations with similar inappropriate diet habits but with different dental statuses (one population is free from decay while the second is affected by the ECC) has not been described in the literature and this was our main goal. The originality of our work lies in the choice of the sample and particularly the control group.

According to the primary statistical analysis, the difference between genders was not statistically significant, similar to what was reported by deFarias and Bazerra in 2003.[13] On the other hand, there was a statistically significant difference in age between the control group (29.3 months) and the caries group (39.7 months) with a p<0.001.

Regarding our first objective to evaluate and compare SM levels taken from the saliva and plaque, we obtained a significant difference between the two groups for all variables (p<0.001). This allows us to confirm the direct relationship between the level of SM and dental caries. These results are similar to those of Seki et al.[25]

As for the evaluation and comparison of the level of total IgA between the two groups of children, we found out a higher level of total IgA in the caries group compared to the control group (30.6 mg/l against 23.6

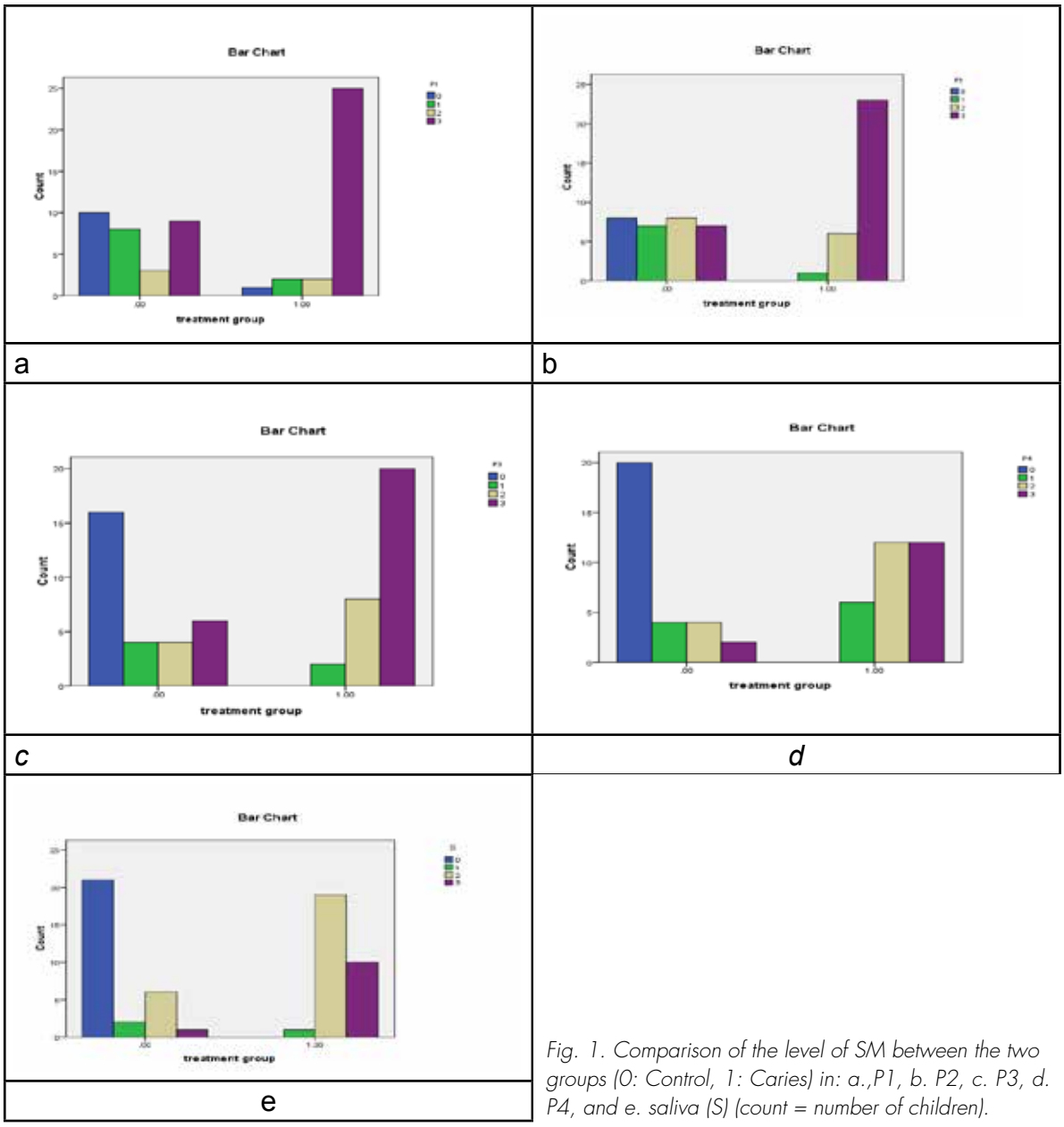

Fig. 1. Comparison of the level of SM between the two groups (0: Control, 1: Caries) in: a.,P1, b. P2, c. P3, d. P4, and e. saliva (S) (count = number of children).

mg/l). Nevertheless, the individual values that we obtained were very variable: one of the children had a level of 251 mg/l whereas the average was 89.6 in the caries group. His score of SM was 3 as he was a premature infant (this factor is considered promoting decay disease).[28] However, the difference was not significant (p = 0.17). Some authors confirm the positive correlation between these two variables (IgA and caries) such as deFarias and Bazerra 200313, while others have found conflicting results.[29,30]

In order to obtain more conclusive results, we grouped the low scores (0 - 1) and high scores (2 - 3) of the SM together. By comparing the level of total IgA with the low and high scores collected from the saliva, we have not obtained any correlation in the groups (p = 0.933 for the control group) (p = 0.35 for the group "caries ").

As for the level of IgA with the low and high scores of the SM collected from the plaque, it showed a positive but marginally significant correlation for the average P: r= 0.33, p=0.077 and P1: p= 0.085 and P4: p = 0.095 independently. In addition, for all the variables of the plaque,

the mean IgA in children at high score (2 and 3) was higher than the lowest scores (0 and 1), but with no significant correlation was obtained.

The same comparison was applied for the group "control" and no significant correlation was obtained. The study of the correlation between the levels of IgA and each of the following variables: age, average P and the number of cavities for each of the 2 groups showed:

- Significant correlation between the level of IgA and the age for the two groups.
- No correlation with the average of P for the control group while for the caries group a positive correlation (r=0.33) was revealed, but marginally significant p = 0.077. As for the number of cavities, the correlation was irrelevant (p = 0.633).

The lack of correlation between the level of IgA and the saliva should not exclude the role of the immune system as our results could be explained by the following factors:

a- The assessment was limited to the rate of total IgA and not that of the SM specific IgA.[31,32]

b- The lack of standardization and reproducibility problems.

c- The effect of salivary flow on the composition of saliva that was not considered in our study.[32]

d- The immaturity of the immune system in children.[33]

e- High levels of salivary IgA may be the reflection of past exposure to SM.[34,35]

The marginal correlation that was obtained by comparing the level of total IgA and scores of SM taken from the plaque, opens up new research perspectives.

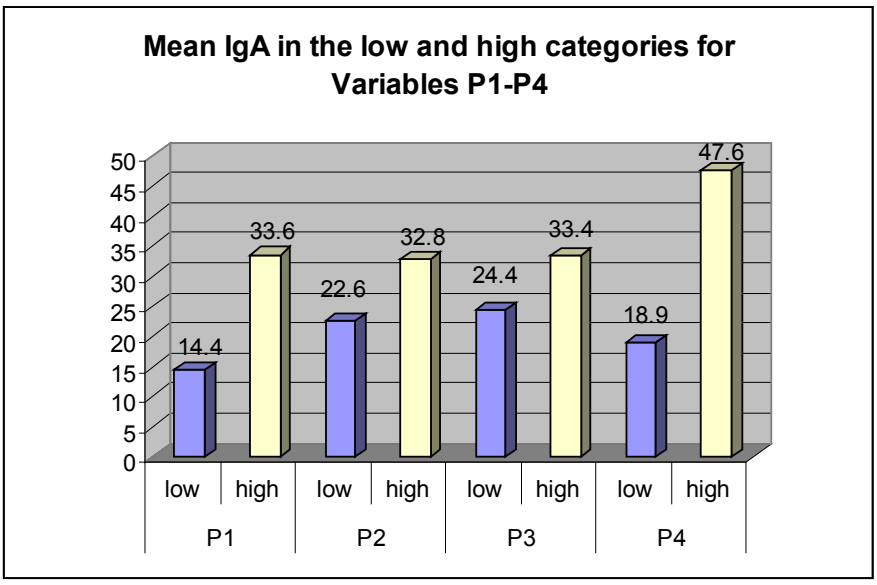

Fig. 2. Comparison of IgA level in low vs. high score of SM, in P1 to P4

CONCLUSION

The particularity of our work lies in the choice of the population. The direct connection between decays in general and ECC in particular and the high levels of SM was evident. Thus, the lack of correlation between IgA and SM collected from the saliva does not eliminate the importance of the preventive role of salivary immune system. The marginal correlation between IgA and SM collected in the plaque, shows a boundary link and needs further research. Prevention, by means commonly recognized as dental hygiene tools and control of diet, stays after all, the basic of all therapeutic measures.

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Establishment of supracrestal tissue dimensions following crown lengthening procedures in humans

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ABSTRACT

Background: The standard application of a mean value of 2.04 mm for the biologic width has been demonstrated to result in inconsistent amounts of tooth extension following crown lengthening procedure (CLP). BW has been recently replaced by the height of supracrestal gingival tissues (SGT) that includes the BW and sulcular depth. The question whether SGT dimension established at tissue maturation postoperatively is not significantly different from the pre-surgical dimension has not been fully answered.

Objectives: The aims of the present prospective clinical investigation are to compare the preoperative and the 24-week height of SGT buccally, lingually/palatally and interproximally following surgical CLP and assess the temporal changes in the gingival marginal level from right after surgery to the 24-week healing time.

Material and Methods: Twenty adult systemically healthy patients requiring CLP were recruited for the study. CLP was performed at 33 teeth and the patients followed up to 24 weeks. The following parameters were recorded using a customized stent for measurement reproducibility: stent-gingival margin (SGM) (baseline, right after surgery, and 24 weeks), stent-bone crest (SBC) (baseline and 24 weeks).

Results: There were no statistically significant differences between SGT at baseline and at 24 weeks for any of the 4 tooth sides. Significant differences were found between SGM right after surgery and SGM at 24 weeks for the mesial and distal sides ($P<0.001$) in contrast to the buccal and lingual/palatal sides where such differences were not detected ($P=0.63$ and 0.64 respectively).

Conclusions: The preliminary results of this investigation suggest the following: 1) The presurgical SGT dimension can be used as a guideline measurement in crown lengthening as it is re-established apically with a similar apico-coronal dimension 24 weeks postoperatively; 2) over time, crown height extension is more significantly reduced at the interproximal aspects suggesting greater postsurgical tissue rebound interproximally.

Key Words: crown lengthening procedure, supracrestal gingival tissues, biologic width, osseous resection

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INTRODUCTION

The creation of an adequate space for the proper placement of prosthetic margins on a compromised tooth can be achieved surgically (crown lengthening procedure), orthodontically,[1] or by combination of both.[2] Clinical crown lengthening is defined as a surgical procedure that aims at exposing sound tooth structure for restorative purposes via apical repositioning of the gingival tissue with or without removal of alveolar bone.[2] Crown lengthening procedure (CLP) has been generally performed applying the concept of the biologic width (BW) that stems from an early histologic description in cadavers and uses a mean value of 2.04 mm as the indicator for the amount of osseous resection needed during surgery. This average value has been challenged in various clinical investigations where inconsistent amounts of crown extension have been reported following surgical CLP.[4-8]

The total height of supracrestal gingival tissues (SGT) which includes the BW plus the sulcular depth has been proposed as a more representative dimension

to apply in CLP as it accounts for the variability of sulcular depth. SGT measurement rather than that of BW could also eliminate the inherent inaccuracies in locating the bottom of the sulcus.9 SGT values have been demonstrated to vary between individuals, arches, gingival morphotypes, tooth types, and sites.[6,10,11] This variability of SGT underlines the need for patient-customized surgical crown extension based on individual soft and hard tissue parameters.

While several clinical studies seem to agree that SGT is a predetermined dimension that re-establishes itself at tissue maturation postsurgically,[12,13,14] other investigations have reported that SGT reformed at tissue maturation postoperatively is significantly different from that recorded at baseline prior to CLP.[6,9,15] These inconsistencies warrant additional research to further investigate this issue.

The objectives of the present prospective clinical study are to compare the preoperative and the 24-week dimensions of SGT buccally, lingually/palatally and interproximally following surgical CLP and assess temporal alterations of the gingival margin between baseline and the 24-week healing period.

MATERIALS AND METHODS

Study population

Twenty adult systemically healthy patients were recruited from a private clinical practice and the Department of Periodontology (Lebanese University, School of Dentistry, Hadath, Lebanon). All patients were informed about the study aims and requirements and signed the written consent form approved by the Scientific Review Board of the institution.

To be included in the study, the patients had to fulfill the following inclusion criteria: 1) age above 18 years; 2) adequate oral hygiene levels evidenced by a FMPS $\leq$ 20% after initial prophylaxis;[16] 3) teeth requiring crown lengthening that do not present pathologic tooth mobility, unfavorable crown-to-root ratio, short root trunk or furcation involvement.

Exclusion criteria included: 1) absence of teeth adjacent to those requiring CLP; 2) presence of attachment loss due to chronic or aggressive periodontitis; 3) ongoing orthodontic therapy; 4) altered passive eruption at the involved teeth; 4) medications known to be associated with gingival overgrowth such as cyclosporin, calcium channel blockers, diphenylidantoin, etc.; and 5) smoking.

The study population provided a total of 33 experimental teeth requiring CLP and 28 control teeth which shared a proximal surface with the experimental teeth.

Experimental procedures

One to 2 months prior to baseline examination, all patients received a comprehensive dental examination, oral hygiene instructions, and full-mouth prophylaxis. All experimental teeth had temporary provisional restorations made (Fig.1a). Tooth preparation was not modified until the end of the experimental period and final clinical evaluation.

At baseline alginate impressions of the surgical areas were made to provide casts for the fabrication of acrylic stents allowing reproducibility of clinical and surgical measurements from fixed reference points throughout the follow-up period. For this purpose, a modified copy of the temporary crown (MSC) with no interproximal contacts and extending to but not apically to the gingival margin was fabricated with 4 vertical slots parallel to the long axis of the experimental teeth allowing the insertion of a periodontal probe 0.5 mm in diameter ;one mid-buccally, one mid-lingually/palatally, and 2 interproximally at the mesial and distal surfaces corresponding to the contact point locations (Figs.1b-1c).

At surgery, plaque index PI (Silness & Löe)[17] and gingival index GI (Löe & Silness)[18] were evaluated in the surgical area. Following local anesthesia and prior to flap reflection, the distance between the coronal edge of the MSC and the gingival margin (SGM0) was recorded using the MSC at the experimental teeth requiring crown lengthening: This measurement was made at 4 sites (mid-buccal, mid-lingual/palatal and mid-interproximal) at all experimental teeth using a calibrated periodontal probe and rounded to the nearest 0.5 mm. The same measurement SGM0 was similarly recorded on the control teeth using available fixed anatomical reference points on the crown.

A split-full thickness flaps were elevated on the buccal and lingual/palatal aspects of the experimental and adjacent teeth (Fig.1d). Following removal of the

supracrestal soft tissues, the direct bone level was measured to the nearest 0.5 mm form the reference stent to the crestal bone (SBC0) at the mid-buccal, mid-lingual/palatal and mid-interproximal aspects of the experimental teeth.

The height of supracrestal tissues (SGT0) was then calculated by subtracting the SBC0 measurement from that of the free gingival margin SGM0 recorded just prior to flap elevation.

Osteoplasty and ostectomy were then carried out using coarse round diamond burs mounted on a high-speed hand piece with copious water irrigation and hand chisels. The amount of bone removal during CLP was dictated by the assumption that pre-surgical SGT will re-establish itself postsurgically, i.e. the distance between the definitive restorative margin and the newly established osseous crest will be equal to SGT height measured prior to flap elevation (Fig.1e).

The buccal and lingual flaps were subsequently apically positioned ensuring maximum coverage of the buccal and lingual/palatal crestal bone using 5-0 monofilament interrupted vertical mattress sutures. At this time, the distance between the stent and the sutured gingival margin (SGM1) was measured at the individual 4 aspects of the experimental teeth (Figs.1f-1g).

Follow-up clinical measurements

Sutures were removed 10 days following surgery. No further modification of tooth preparation or alteration in the existing margins of the temporary restoration was made during the 6-month follow-up period.

Plaque and gingival indices were re-evaluated in the surgical area at 12 and 24 weeks. The distance between the coronal edge of the MSC and the gingival margin was recorded as previously described at 24 weeks (SGM24) (Fig.1h).

In addition, at the 24-week evaluation period, the distance between the stent and the alveolar crest (SBC24) at the mid-buccal, mid-lingual/palatal, and mid-interproximal surfaces of the experimental and adjacent teeth was recorded under light local anesthesia using the MSC (Fig.1i). SGT24 was then calculated as described above.

Statistical analysis

Data were summarized using means and standard deviations for continuous variables. The paired t-test was applied to compare baseline and 24-week postoperative values of SGT separately for buccal, lingual/palatal and interproximal measurements. In case the variables did not follow a normal distribution, the nonparametric Wilcoxon signed rank test was used. Friedman's test was applied to detect changes in SGM values from right after surgery till 24 weeks.

RESULTS

All patients maintained adequate levels of oral hygiene and low gingival indices throughout the early and late healing periods.

At baseline, comparison of SGT measurements at the 4 sides of all experimental and control teeth is summarized in Table 1. SGT at the buccal aspect was significantly lower than the other tooth sides ($P<0.05$). No significant differences were found between SGT values at lingual/palatal, mesial and distal aspects ($P>0.05$). When SGT was compared for the 33 experimental teeth between baseline and 24 weeks postoperatively separately for each side of the teeth (mesial, buccal, distal, and lingual/palatal) and for the average SGT (mean SGT value for all 4 surfaces), the results (Table 1) demonstrated no statistically significant differences between SGT at baseline and at 24 weeks for any of the 4 sides or for the overall average ($P>0.05$ for the paired t-test and non-parametric Wilcoxon signed rank test). Similar findings were observed for the control teeth at individual sites and mean all around SGT values. P values were all superior to 0.05 indicating statistically non significant differences between baseline and the 24-week SGT dimensions at the control teeth.

Over time comparisons of mean SGM for the experimental teeth at the individual sites (mesial, buccal, distal, and lingual) were computed and summarized in Table 2. Friedman's test was used for detecting changes in SGM from right after surgery until 24 weeks postoperatively. Statistically significant differences were found between SGM1 and

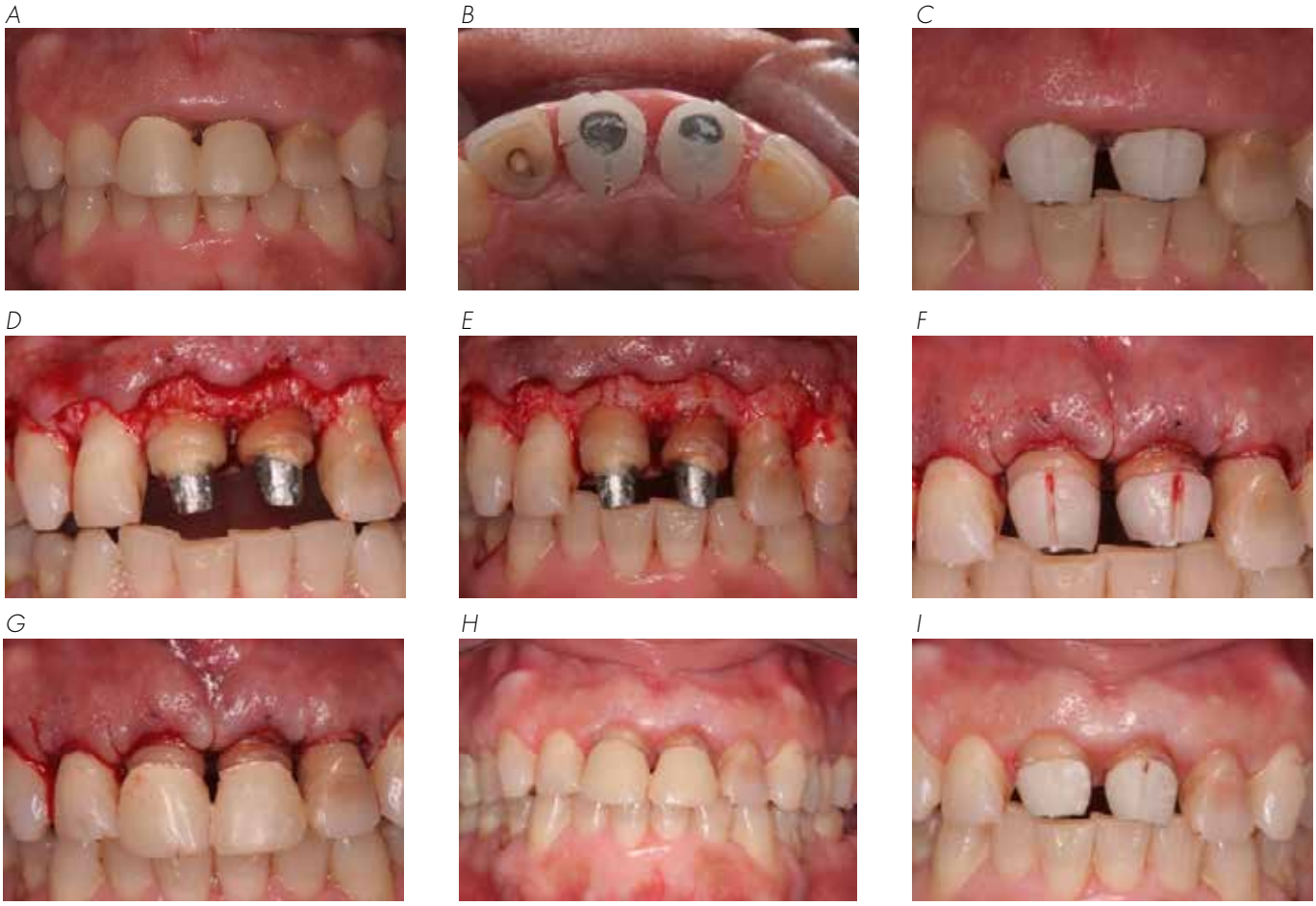

Fig. 1. Clinical pictures showing the experimental step-by-step procedure.

SGM24 for the mesial and distal sides ($P<0.05$). In contrast, no significant differences were evidenced between SGM1 and SGM24 for the buccal and lingual/palatal sides ($P=0.63$ and 0.64 respectively).

Similar trend was found at the control teeth relative to SGM values. Friedman's test was used for detecting changes in SGM from right after surgery till 24 weeks post surgery. There was a significant decrease in SGM over time at the mesial and distal sides of the teeth ($P=0.006$ and 0.001, respectively) while no significant change in SGM dimensions was found at the buccal and lingual aspects after surgery.

DISCUSSION

In the present study, significantly lower SGT values were observed at the mid-buccal sites when compared with the interproximal and lingual sites. Similar conclusions can be extrapolated from the findings of Barboza et al11 although the authors did not perform comparative statistical tests. Mean SGT values ranging between 2.2 mm and 3.0 mm at the central buccal sites versus average measurements ranging between 2.6 mm and 3.2 mm at the lingual sites and between 3.4 mm and 4.2 mm at the interproximal surfaces were reported. Similarly, Perez et al[6] found mean SGT dimensions of 3.47 mm at mid- buccal and mid-lingual sites versus averages ranging between 3.47 mm and 4.05 at the line angles of proximal surfaces. Kois[19] attributed this discrepancy between buccal and interproximal sites to the difference in the amount of scalloping between bone and gingiva. No such site-level variation in SGT measurements could be demonstrated in the human histologic study conducted by Vacek et al[7] When comparing the dentogingival tissue dimensions between tooth surfaces, the authors concluded that no statistically significant differences were evident for any of the tissue dimensions including epithelial

Table 1. Comparison of SGT values at experimental teeth before surgery and 24 weeks postoperatively.

N= 33	Baseline Mean (SD) (mm)	24 Weeks After Surgery Mean (SD) (mm)	P-value
SGT Mesial	3.24 (0.82)	3.54 (0.50)	0.105
SGT Buccal	3.00 (0.91)	2.82 (0.50)	0.361
SGT Distal	3.16 (1.11)	3.42 (0.66)	0.293
SGT Lingual	3.56 (1.10)	3.34 (0.66)	0.283
Overall SGT	3.24 (0.62)	3.28 (0.32)	0.756

Table 2. Comparison of SGM values at experimental teeth between baseline and the 24-week evaluation interval.

N=23	Baseline (SGM0) Mean (SD) (mm)	Right After Surgery (SGM1) Mean (SD) (mm)	24 Weeks (SGM24) Mean (SD) (mm)	P-value*
Mesial	2.80 (0.90)	5.07 (0.84)	3.64 (1.01)	<0.05
Buccal	4.93 (1.17)	6.70 (1.00)	6.39 (1.28)	0.63
Distal	3.20 (1.30)	5.30 (0.96)	4.13 (1.03)	<0.05
Lingual	4.26 (1.69)	5.65 (1.33)	5.11 (1.45)	0.64

* P-value for the t-test between SGM1 and SGM24

attachment, connective tissue attachment and sulcus depth.

When considering the mean figures of SGT at the site-level, the present study reported average mid-buccal values inferior or equal to 3 mm. These figures are similar to those demonstrated by Tristão et al.[10] in a histomorphometric evaluation of the midbuccal SGT in humans (2.75 ± 0.59 mm). Conversely, Barboza et al11 Perez et al,[6] reported higher figures for the mid-facial surfaces (3.47 mm). The differences between the SGT dimensions observed in the present study and those in the 2 above mentioned reports could be attributed to the location of the measurement site (mid-interproximal versus line-angles), to differences in tooth types, differences in gingival morphotypes (thick versus thin), and measurement rounding (rounding up to the nearest 0.5 mm versus the nearest millimeter).

In the present study, the corono-apical dimension of SGTs re-established itself postsurgically at all sites in the experimental and control teeth. This finding is in line with the conclusions of Lanning et al[14] but in contradiction with those of Perez et al[6] and Arora et al[9] who demonstrated that the mean SGT values obtained 6 months postsurgically were reduced when compared to preoperative dimensions. These differences could be related to: 1) measurement rounding; 2) the surgical procedure itself where ostectomy and osteoplasty would have resulted in greater changes in the local anatomic tooth-related and bone-related variables; 3) tissue biotype where thin gingival morphotypes tend to have more postoperative recession when compared to thicker biotypes; and

4) differences in postsurgical bone resorption as a result of osseous recontouring.[4,20-22] The results of the present study and those of Lanning et al14 tend to confirm the hypothesis of a genetically predetermined SGT height to be used as a guideline in CLP procedures.

Tissue rebound associated with a coronal displacement of the gingival margin was observed in the postoperative period, mainly at the interproximal sites that demonstrated significantly greater creeping than both buccal and lingual surfaces. Postsurgical tissue rebound has been documented in a number of studies.[5,8] No documentation is currently available relative to the significant differences in gingival tissue rebound between buccal/lingual and interproximal sites following CLP. This differentiated behavior can be attributed to the inherently greater SGT interproximal values.

Based on the preliminary results of this clinical study, it can be concluded that the presurgical SGT dimension can be used as a guideline measurement in crown lengthening as it is re-established apically with a similar apico-coronal dimension 24 weeks following CLP. In addition, greater postsurgical tissue rebound should be expected interproximally than at the buccal and lingual/palatal surfaces.

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The authors reported no conflicts of interest related to this study.

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Hidden correlation between Obstructive Sleep Apnea, temporomandibular disorders and sleep bruxism: a literature review

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ABSTRACT

In contemporary dentistry, trained dental professionals play an important role in the early assessment and treatment of some Obstructive Sleep Apnea, as well as in the management of sleep bruxism and temporomandibular disorders. There are a number of questions that arise due to the commonalities existing between the aforementioned medical conditions which may suggest a possible new approach in the clinical evaluation of these disorders. In this paper, we address these issues through a detailed exploration of hidden correlation based on a peer reviewed literature.

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INTRODUCTION

Sleep Disordered Breathing (SDB), is a common disorder that has numerous clinical presentations. The most frequently identified are Obstructive Sleep Apnea (OSA) and Upper Airway Resistance Syndrome (UARS).[1-3]

During the last decade, many studies have identified certain relationship between OSA, Temporomandibular Disorders (TMD) and Sleep Bruxism (SB), but it is still not yet fully understood.[4] In this paper, we try to enlighten the causal relationship and recognize the association between these clinical disorders.

Obstructive sleep apnea

OSA is one common sleep disorder characterized by recurring collapse of the upper airway during sleep, resulting in sleep fragmentation and oxygen desaturation. OSA is defined as the occurrence of 5 or more episodes of complete (apnea) or partial (hypopnea) upper airway obstruction per hour of sleep.[5]

Obstructive Sleep Apnea Syndrome (OSAS) is a frequent respiratory sleep disturbance that affects 4% to 9% population and this high prevalence increases the

probability that patients with OSAS could also present TMD and SB.5

Sleep bruxism and Obstructive sleep apnea

Sleep bruxism

Sleep Bruxism (SB) is defined as a stereotyped movement disorder characterized by rhythmic masticatory muscle activity (RMMA) associated with tooth grinding (TG) and occasional tooth clenching.[6] The most important clinical manifestation of bruxism is tooth wear (TW) described in the literature as the loss of the constitution of the tooth and has been classified as being caused by attrition, abrasion, erosion, or a combination of these factors.[7] As it relates to SB, tooth wear is reported to additionally cause tooth mobility, temperature hypersensitivity, and tooth fracture.[8] It has also been postulated that much of the wear could be erosion rather than attrition.[9] Interestingly, those two factors are interwoven in the bruxism triad patient (sleep bruxism, sleep disturbance, and sleep-related gastroesophageal reflux disease), magnifying the wear in this patient population.[9]

Etiological factors of sleep bruxism

In the literature, many etiological factors have been linked to SB such as nicotine, ethanol, caffeine intake, occlusal discrepancies, stress, psychological, alterations in neurochemicals, interactions related to airway patency and salivary flow, genetic and familial predisposition, and occlusal factors.[10-18]

Prevalence of bruxism in obstructive sleep apnea

In 2001, Ohayon et al[12] reported that the highest prevalence of SB was between the ages of 19 and 44 years. There was no sexual dimorphism and it was found to significantly decrease with age. The authors also found, by applying the International Classification of Sleep Disorders (ICSD) criteria, that half of tooth grinders met the diagnosis of sleep disorders (SD) and adversely were affected by their tooth grinding, with reports of muscular discomfort, disturbance of a partner's sleep, or a need for dental treatment. At least one third of patients with bruxism in the general population may also have SDB conditions such as sleep apnea, periodic leg movement during sleep, and headache.[19,20]

In a study involving 1,042 subjects in 2013, Maluly et al found out a prevalence of self-reported SB of 12.5%. When a self-reported questionnaire was combined with polysomnographic recording, which is considered as the gold standard tool for sleep bruxism diagnosis, the prevalence was 5.5%.[21] In 2014, Hesselbacher et al reported that the prevalence of self-reported SB is significantly higher in OSA patients (26%) than in general population.[22] In addition to OSA, 50% of UARS patients complain of SB,[19] which was considered as risk and/or perpetuation factor of TMD.[23-25] Furthermore, Shedden Mora et al[26] suggested a common link between nocturnal masseter muscle activity (NMMA), somatization, and symptom intensity in chronic TMD.

Protective hypothesis of bruxism in obstructive sleep apnea

The hypothesis regarding the relation between bruxism as a reflexive mechanism and apnea severity is that bruxism tend to improve or protect airway dimensions. The rise in CO_2 level (hypercapnia) causes the ventilatory stimuli to increase the activity of both masseter muscles by stabilizing the mandible and genioglossus by dilating the upper airway more efficiently,[27-30] especially in 20- to 40-year-old subjects,[9] and thus delaying OSA development. With age, the neurochemical ability to brux decreases, and consequently, the reflexive mechanism cannot overcome the additional airway obstructions due to the loss of muscle tone, weight gain.[9]

In 2000, Sjöholm et al considered that mild apnea patients had more bruxing events than moderate apnea patients.[31] This was explained by the limited correlation between bruxing and apnea. Later in 2013, Saito et al found that in patients with concomitant OSAS and SB, most SB events occurred after sleep OSA events, suggesting that SB events occurring close to OSA events is a secondary form of SB.[32] In addition, Hosoya et al found out that patients with OSAS have a high risk of SB.[33] This relationship suggests that improvement in OSAS might prevent exacerbations of SB. It was observed that the frequency

of sleep apnea increased as the frequency of bruxism increase.[19,34] While the causal relationship is not based on evidence, OSAS has been known as the highest risk factor for tooth grinding during sleep.[35,36]

Temporomandibular disorders and obstructive sleep apnea

Temporomandibular disorders (TMDs) might include many clinical problems that involve the masticatory muscles pain (13%), the temporomandibular joint pain (9%) and the condyle-articular disk complex (16%).[37-39]

They can be influenced by age,40psychological stress,[41] gender,[42] depression,[43] sleep problems,[44] general health,[41] anatomic changes, trauma, ligament laxity, occlusal imbalances and parafunctional habits.[39] Kou et al and Wu et al demonstrated that estrogen receptors have been detected in TMJ tissues and play an important role in joint inflammation.[45,46]

Many factors might contribute to TMD including:[47,48]

Initiating factors: lead to the onset of the symptoms and are related primarily to trauma or adverse loading of the masticatory system.

Perpetuating factors: include behavioral, social, emotional and cognitive factors.

Predisposing factors: are pathophysiologic, psychological or structural processes that alter the masticatory system sufficiently to increase the risk of development of temporomandibular disorders.

The primary clinical signs and symptoms of TMD are pain in the facial region and TMJ, limited asymmetric mandibular movement, TMJ sounds, headache, and sleep disturbances.[4]

The prevalence of temporomandibular joint and muscle disorder (TMJD) is between 5% and 12%.[49] TMJ disorders are at least twice as prevalent in women as men and the two peak periods of incidence are in puberty and during menopause.[50]

The Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) are often used to diagnose most common TMDs.[4,51] RDC/TMD is a widely used diagnostic system for TMD[51,52] and has two assessment components:

Axis I, a clinical and radiographic assessment designed to differentiate myofascial pain, disc displacement, and arthralgia, arthritis, and arthrosis.

Axis II, evaluates psychological status and pain-related disability.

In 2009, Smith et al found that 28% of patients diagnosed with the based myofascial pain's RDC/TMD were identified by polysomnography as having OSA.[53]

The prevalence of TMD and OSAS are high in the general population. Cunali et al reported that 36.8% of the OSAS patients presented TMD,[4] while other studies observed that 4.9% of TMD patients had some form of OSA.[54]

The Orofacial Pain Prospective Evaluation and Risk Assessment (OPPERA) is a prospective clinical study to identify risk factors that contribute to someone developing a TMJ disorder. In 2013, Sanders in a OPPERA cohort study, demonstrated that OSA symptoms are associated with both first-onset TMD and chronic TMD.[55]

Autonomic nervous system activity

According to Diatchenko et al[56] individuals who are genetically predisposed to an increased sensitivity to catecholamines are at increased risk of developing first-onset TMD. Likewise, Huynh et al[57] and Carra et al[58] have reported that SB subjects have a higher sleep-time sympathetic-cardiac activity than healthy subjects. In 2012, Schames et al[59] reported also that SB triggers the parasympathetic trigeminal cardiac reflex leading to bradycardia. They considered that the rhythmic masticatory muscle activity slows the heart rate when brain microarousals in OSA cause tachycardia. Sanders et al[55] hypothesized that the increased stimulation of the sympathetic nervous system observed in OSA underlies an increased prevalence of TMD. These findings would warrant further investigations on the sympathetic activity and its correlation with TMD, SB and OSA.

Manadibular advancement devices

Oral appliance therapy has been demonstrated an effective alternative in treating OSAS, especially in mild and moderate OSA situations and in patients unwilling or unable to tolerate

Continuous Positive Airway Pressure (CPAP). The aim of these appliances is to relieve the upper airway obstructions by holding the mandible in a forward position.[38]

Mandibular advancement devices (MAD) have reported to reduce bruxism events 50% to 83%.[60] These devices may also be used in SB patients without respiratory disturbances. A statistically significant reduction (39% and 47%) of SB episodes per hour was recorded with the MAD at protrusion of 25% and 75%, respectively,[61] providing further evidence that improving airway patency is an important treatment strategy for bruxism even in a normal patient population.

Pain or discomfort in the Temporo-Mandibular Joint (TMJ) has been reported as temporary side effects during short and medium periods of oral appliance wear. Long-term side effects are characterized by occlusal changes without the presence of pain.[4,62] Standardized criteria for TMD diagnosis, such as the RDC/TMD, should be part of the examination procedure for OSAS patients referred for oral appliance treatment.[4,54] In 2012, Doff suggested that the possible development of TMDs or temporary pain of the temporomandibular complex is not a contra-indication for oral appliance therapy in OSAS patients.[38]

CONCLUSION

When treating TMD and SB patients, dental professionals should take into account sleep disturbances in order to improve treatment prognosis. In addition, they should not forget that due to the high prevalence of TMD and its correlation to OSA, patients can present both disorders at the same time. Adequate treatment planning will maximize benefits for the patients.

Since bruxism could be a normal physiologic reactive protective mechanism, clinicians might be able to predict the possibility, in a young patient population, that bruxism could be linked to apnea and therefore a further assessment of the SB and OSA relationship is recommended.

To date, our literature review showed that further collective research works need to be done taking into consideration the diagnostic methodology, both for TMD and SB and their relation to OSA, in order to facilitate the job and unify the data collection according to a standardized protocol.

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Dental students and teachers confronted to science

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INTRODUCTION

Most changes in therapy come nowadays from new products and their inherent techniques as they are introduced to the market.[1] Clinicians (and patients) can be overwhelmed by marketing strategies and advertisements, some obvious and some not so obvious (i.e., sponsored clinical case reports in non-peer-reviewed journals). Some are even backed with small case studies published in peer-reviewed journals but which are at lower levels of evidence and their validity is often subject to discussion. This seems contradictory with the evidence-based dentistry concept (EBD) to which dentistry gradually shifted to in the 1990's[2] As the name suggests, EBD implies guiding clinical decisions and therapy by evidence obtained from bench-top or clinical research.

Problems with EBD

One of the obvious problems in the internet age is the quantity of information available to the reader. Scientific information is growing at an exponential rate and as an example, a current search on Pubmed with "dentistry" as a keyword will yield around 468,761 papers,[3] which is why students (and clinicians) are generally confronted to an abundance of papers when looking up a particular subject. A search on Google Scholar for the same keyword will yield 1,330,000 results,[4] and on Google more than 101 million hits.[5] The quality of the information is another issue; as all information cannot be fully trusted and should be critically appraised prior to adopting the conclusions it reaches. Kurichi and Sonnad estimate that twenty-seven percent of studies published between 1985 and 2003 in Surgery journals included incorrect selection or reporting of statistical methods.[6] Molckovsky et al.[7] reported that error rates in high-impact oncology journals averaged 4%, which they considered to be likely an underestimate, as errors noticed by readers were not consistently reported, and errata published by journals were not systematically noticed or read. They concluded that the propagation of serious errors would decrease but still continue after publication of errata. Errors (voluntary or not) serious enough to cause retraction of published research papers are also present and put patients at risk by influencing treatments in the period before the retraction.[8] Nevertheless, the overall quality of publication seems to be improving with time as Gibson and Harrison reported an 8-fold increase in randomized controlled trials between 1989-1993 and 1999-2008.[9]

Problems with readers

It just so happens that one of the key skills needed for evidence-based practice is the ability to critically appraise information,[10] which is why students are confronted to scientific requirements such as reading scientific and analyzing manuscripts, commenting on methodology and/or results. Critical reading is unfortunately not a skill routinely taught at undergraduate level and often timidly introduced at postgraduate level. This results in the fact that students often have insufficient knowledge in research methodology and statistics to be able to discriminate between good research, poor research and downright misleading research. Furthermore, exposure of students (and even teachers) to courses and workshops aiming to improve their critical appraisal skills is generally limited. This point has been reported and highlighted in several publications that unanimously concluded that readers, whether students or doctors, are generally poorly prepared to assess papers, which requires proficiency in research methodology and core statistical concepts.[11-14] In this aspect, it would seem that faculty members responsible for teaching research are challenged to develop creative and interesting approaches to avoid students' perceptions of research as boring, dull, not for them, or irrelevant to what they really want to do. As for statistics, Reed et al.[11] concluded that a physician who comfortably comprehends the appropriate use of descriptive statistics Student's t-test, Pearson's chi-square/Fisher's Exact test will be able to read and interpret at least 70% of the published medical literature. Educational efforts should focus on appropriate study design and analysis.

From another standpoint, it has been noticed that primary research and "evidence-based" papers seem to be less attractive to readers than narrative reviews and editorials[15] and that clinicians prefer throwaway over peer-reviewed journals.16These findings may seem disappointing as we know that it is important for readers to critically appraise the primary research

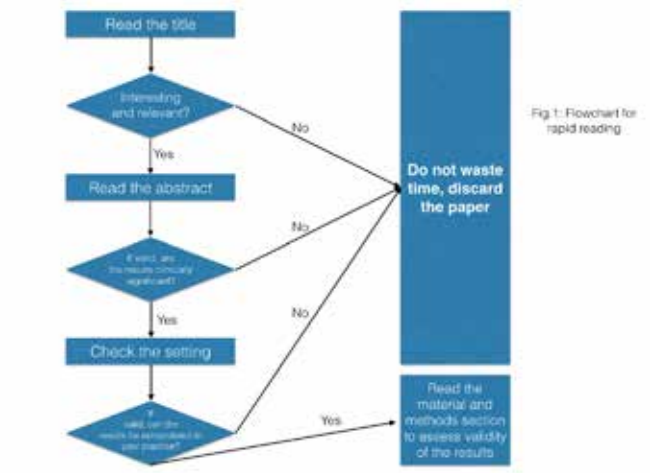

Fig.1: Flowchart for rapid reading

data. It remains an undeniable fact that the readability of articles has received little attention. Authors and journal editors must keep in mind that articles are not meant to be written, but read. They must take steps to make research papers and systematic reviews more attractive to readers, which could include for example using simpler language.

Problems with reading techniques

The criteria for rapid reading[17] aim at optimizing reading time by separating the wheat that will require a more careful read, from the chaff that will be readily eliminated. One approach would establish usefulness of the information based on the title and the abstract (Fig.1). Step 1 would be appraising the title: does the title indicate that the paper is pertinent to the information requested by the reader? In the affirmative, step 2 would be to appraise the abstract. The issue is not to establish whether the results are true or not, but rather if they convey any clinical significance that would affect the reader's clinical behavior. Generally speaking, if this is not the case the reader will probably decide not to read the paper in depth. Conversely, readers often decide to modify their practice based on reading abstracts that describe clinically significant differences. Unfortunately, it has been established that if the reader makes decisions based on reading the abstract alone, biased or inappropriate conclusions may be drawn, particularly with regards to study limitations, adverse events,

Criteria	Y	N	I	N/A	N/U
Design					
Are the aims clearly stated?	☐	☐	☐	☐	☐
Does the observation technique allow to detect what is measured?	☐	☐	☐	☐	☐
Sampling					
Are the inclusion criteria well described and adequate?	☐	☐	☐	☐	☐
Are the exclusion criteria well described and adequate?	☐	☐	☐	☐	☐
Is the recruitment process well described?	☐	☐	☐	☐	☐
What is the randomization process?	☐	☐	☐	☐	☐
Are ethical guidelines followed?	☐	☐	☐	☐	☐
Implementation					
Is the study standardized?	☐	☐	☐	☐	☐
Are there any drop-outs and is the reason given?	☐	☐	☐	☐	☐
Are all the devices/materials listed?	☐	☐	☐	☐	☐
Are the methods/materials well described?	☐	☐	☐	☐	☐
Statistical Analysis					
Is the data collection method well described?	☐	☐	☐	☐	☐
Are the statistical tests consistent with data type/study design?	☐	☐	☐	☐	☐
Other criteria					
Are selection/measurement/design biases addressed?	☐	☐	☐	☐	☐
Are selection/measurement/design biases addressed?	☐	☐	☐	☐	☐

Fig.2: Sample of reviewing chart. Y=Yes, N=No, I=Incomplete, N/A=Not available, N/U=Not understood

and subject dropouts or losses, and that irrespective of article type, journal, or level of evidence.18As a general rule, one may decide to dismiss a paper based on the abstract, but accepting the conclusions of a paper based on the abstract alone without going back to the materials and methods section to put the results back into perspective is hazardous at best. Step 3 would assess the context of the study to compare it with the reader's context and establish whether their conclusions would be applicable in the reader's clinical setting. For instance if a study was conducted in a hospital environment, it would understandably not necessarily be possible to extrapolate the results to a general practitioner's office hence reducing the reader's interest for that particular paper.

The criteria for in-depth reading are generally more numerous and involve various points ranging from the writing style to the validity of the statistical analysis. They are in fact similar (or should be similar) to those used by reviewers. The use of evaluation charts (fig.2), guide the reader and simplify the process but this reading technique is highly time consuming. Nevertheless, in-depth reading is essential for evaluating the results published by the authors modulated by the conditions described in the materials and methods section, which in turn will allow to safely and effectively extrapolate the results to ensure the optimal approach to specific clinical situations.

CONCLUSION

If validity and proper design are imperative to achieve evidence-based dentistry that uses relevant, high quality, clinically oriented research that provides better information for the clinician and better treatment for the patient, critical appraisal of said research by the reader should be equally efficient. Criticism should however be structured constructively as quite often the quantity of work behind publications is considerable and generally commands respect. The temptation to go all out on criticism should thus be refrained for as Dale Carnegie once said: "Any fool can criticize, condemn, and complain; and most fools do."

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