EUROPEAN JOURNAL OF BIOMEDICAL AND PHARMACEUTICAL SCIENCES
http://www.ejbps.com

ISSN 2349-8870
Volume: 6
Issue: 13
71-81
Year: 2019

THE INFLUENCE OF THE MOLAR BAND AND BUCCAL TUBE ON PERIODONTAL HEALTH IN ORTHODONTIC TREATMENT

Taleb A. A. Alqarari[1] and Jing Mao*[1]

Department of Stomatology, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, Hubei 430030, China.

***Corresponding Author: Prof. Jing Mao**
Department of Stomatology, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, Hubei 430030, China.

Article Received on 29/09/2019 **Article Revised on 19/10/2019** **Article Accepted on 09/11/2019**

ABSTRACT

Objective: To compare the side effects of the molar bands and buccal tubes on periodontal health during orthodontic treatment. **Methods:** We selected 42 patients who treated with the fixed appliance, they all in the age range of 12-16 years, for each one choosing two first molars and one of the second molars in mandible as research objects, it is a total of 126 teeth. We bonded both a buccal tube and a molar band in the first molars of mandible, using the stochastic method to decide which side bonded the buccal tube, bonding it in the middle of the tooth crown. Take the second molar in buccal tube side as a blank control. In this study, 84 first molars were selected as the experimental group and 42 second molars without fixed appliance as the control group. The experimental group can be divided into two small groups. Buccal group, the 42 molars which used the buccal tubes in orthodontic treatment. Moral group, the 42 teeth which used the molar bands in orthodontic treatment. Furthermore. Gingival index and Pocket probing depth were recorded for clinical evaluation, IL-1 and IL-6 were tested use ELISA in this study. **Results:** In this study, there was no difference in all of the indexes among three groups before our experiment. After placing fixed appliance 6 months and 12 months, both of the experimental groups (buccal tube group and molar band group) showed that GI, PPD continued to increase. Compared with baseline, they had significant difference ($P < 0.05$), although there was no significant difference between buccal tube group and molar band group ($P>0.05$), the mean value of the GI, PPD in molar band group was larger than buccal tube group. After placing fixed appliance 6 months and 12 months, both of the experimental groups showed that IL-1 and IL-6 continued to increase. Compared with baseline, they had a significant difference ($P < 0.05$). In addition, there was a significant difference between IL-1 and IL-6 between buccal tube group and molar band group ($P<0.01$). It showed that inflammatory substances in the gingiva groove liquid increased during the treatment. **Conclusion:** In orthodontic treatment, buccal tubes caused less inflammatory response on periodontal health than molar bands, which was helpful to achieve better treatment effect. We concluded that buccal tube was more conducive to the periodontal health than molar band.

KEYWORDS: Buccal tube; Molar band; Periodontal index; Interleukin-1; Interleukin-6
Classification: R783.5.

INTRODUCTION

Gingivitis, especially in the region of the interdental papilla, has been observed in teeth banded for orthodontic purpose.[1,2] These have been associated with higher plaque rates,[3-5] gingival rates and probing depth[6-10], Plaque accumulation can be due to the bands placed which make plaque control difficult. Theoretically, therefore, controlling plaque could prevent gingivitis and periodontitis. Gingivitis is one of many periodontal diseases that affect the health of the periodontium.[11] Periodontal diseases are often classified according to their severity. They range from mild gingivitis; to more severe periodontitis.[12] It is well established that the patients who undergo orthodontic treatment have a high susceptibility to present plaque accumulation on their teeth because of the presence of bands, brackets, wires, and other orthodontic attachments. Recent studies show that the most important etiological factor of periodontal disease is plaque deposition around gingival margin.[13] Orthodontic treatment is a double-action procedure, regarding the periodontal tissues, which may be sometimes very significant in increasing the periodontal health status, and sometimes a harmful procedure which can be followed by several types of periodontal complications, namely: gingival recessions, bone dehiscence, gingival invaginations and/or the formation of gingival pockets, established that bacterial plaque is the primary etiologic factor in the development of gingival inflammation and periodontitis.[14] The quantity and the quality of plaque are known to be influenced by

many factors, including surface characteristics.[15-17] Especially surface roughness and high surface free energies were found to be positively correlated with plaque growth and maturation.[18] Gingival inflammation is known to further increase this.[19-21] In addition to the total amount of bacteria, the ratio between the aerobic and anaerobic bacteria is also an important marker for plaque pathogenicity.[22] The placement of orthodontic bands and buccal tubes and brackets influences plaque growth and maturation because of the abovementioned factors. Significant differences in biofilm formation on bonded teeth compared with control teeth were reported.[23] Most studies reporting on gingival changes after bracket placement suggested only temporary reversible periodontal changes.[24,25] Another study, however, reported significant attachment loss during orthodontic treatment.[26] Another way of studying the changes during orthodontic therapy is by analysis of the composition of the gingival crevicular fluid (GCF). GCF reflects the immune and inflammatory reactions from host-parasite interactions[27,28] and biomechanical stresses.[29,30] It is a noninvasive method, and until now, many substances involved in the inflammatory process and produced by the periodontal ligament cells in sufficient amounts to diffuse into the GCF have already been studied.[31] Giannopoulou et al compared the GCF composition of orthodontic patients with that of non orthodontic controls. However, the wide inter-subject differences in GCF composition made reasonable comparisons difficult and warrant prospective intra subject studies. Among many inflammatory and immune mediators identified in GCF, cytokines have attracted particular attention. Interleukin-6 (IL-6) is known to be a major modulator of inflammation in chronic local inflammatory reactions. It regulates the immune cell recruitment in the transition from the acute (recruitment of neutrophils) to the chronic (recruitment of monocytes) form of inflammation.[32] interleukin IL-1, IL-6, and tumor necrosis factor TNF-α are key mediators involved in a variety of immune and acute-phase inflammatory response activities.[33-36] The RANK/RANKL (receptor activator of nuclear factor κB and RANK ligand) pathway is also essential for osteoclast differentiation in inflammatory arthritis.[37] Orthodontic tooth movement is induced by mechanical stimuli and facilitated by remodeling of the periodontal ligament (PDL) and alveolar bone. A precondition for those remodeling activities, and ultimately tooth displacement, is the occurrence of an inflammatory process. Vascular and cellular changes were the first events to be recognized and described, and a number of inflammatory mediators, growth factors, and neuropeptides have been demonstrated in periodontal supporting tissues. Their increased levels during orthodontic tooth movement have led to the assumption that interactions between cells producing these substances, such as nerve, immune, and endocrine system cells regulate the biological responses that occur following the application of Orthodontic forces.[38] IL-1 exists in two forms, α and β[39], of which IL-1β is the form mainly involved in bone metabolism, stimulation of bone resorption[40,41] and inhibition of bone formation.[42] IL-1β also plays a central role in the inflammatory process. The staining of feline PDL cells for IL-1β showed the presence of bound signal complexes in the plasma membrane, which was expected, as it is known that receptors for IL-1β are present on fibroblasts.[43] Lo et al.[44] suggested that large amounts of IL-1β are present in inflamed gingival tissues and found that both macrophages and neutrophils are predominate in IL-1β production in inflamed gingival tissues. Kanda-Nakamura et al[45] also suggested that the response of gingival fibroblasts to IL-1 may represent a mechanism for amplification of gingival inflammation. Further, IL-1β may act synergistically with TNF-α22 as a powerful inducer of IL-6.[46,47] IL-6, a multifunctional cytokine previously referred to as B cell stimulatory factor 2, hepatocyte stimulating factor, or interferon- β2, is produced by both lymphoid and non-lymphoid cells[48] and can apparently induce osteoclastic bone resorption through an effect on osteoclastogenesis.[49-51] IL-6 was found to be produced by the human foreskin fibroblast line FS-4 and its synthesis was stimulated by treatment with IL-1.[52,53] The identification of IL-6 in human gingival tissues and cells implicates this lymphokine as a participant in the molecular events associated with inflammatory periodontal diseases 5. Irwin and Myrillas[54] reviewed the biological functions of IL-6 and found them to be specifically related to tissue destruction in the periodontal site. In addition, Yakovlev et al[55] found that levels of IL-1 β and IL-6 were significantly higher in inflamed as compared with non-inflamed gingival tissues in young adults, while Shimizu et al[56] demonstrated that IL-6 was produced by IL-1β from PDL cells.

The aim of this study is to compare the side effects of molar band and buccal tube on periodontal health during orthodontic treatment and determine the significative changes of IL-1 and IL-6 in GCF(Gingival Crevicular Fluid) with molar band and buccal tube in orthodontic treatment.

MATERIALS AND METHODS

1 Material agent

Molar bands and buccal tubes, Hangzhou Xinya dental material company.

Absorbent paper points no.30 Medical devises Co., Ltd. Of Tianjin Daya Ding.

Fuchsin basic sodium tetraborate Shanghai rex co-perfect instrument co., td.

Eppendorf Low temperature 5804R Eppendorf AG, Germany.

12 pipettes Eppendorf AG, Germany Microplate reader MB-530, Shenzhenshihui Technology Development Corporation Automatic plate washer PW-812, Shenzhenshihui Technology Development Corporation Micropipette Shanghai Instrument Co., Ltd.

2 Clinical data collection

Clinical data collection was in the Orthodontics Department, from June 2016 to end March 2018, for effect of the molar band and buccal tube on periodontal health in orthodontics. 42 patients, 126 molars for this study were selected from the one who attended to the orthodontic clinic. They were all in the age range of 12-16 years. There was no evidence of decalcification on their teeth. There was no medical problems, evidence of antibiotic therapy or topical chemical agents during this study.

In this study, 84 first molars were selected as experimental group and 42 second molars as control group. All samples were taken from mandibular. For experimental groups, we can also divided into two another two groups.

GroupA, cases using buccal tubes for the orthodontic treatment.

GroupB, cases using molar bands for the orthodontic treatment.

Control group: second molars without fixed appliance.

3 Clinical examination

Gingival index and Pocket probing depth were recorded for clinical evaluation. These baseline readings were taken before fixing the molar bands and buccal tubes in the experimental group, also before treatment in the control group.

2.3.1 Gingival index: Gingival health condition of the right lower first molar was assessed using the criteria of the Gingival Index system (GI).

2.3.2 Pocket probing depth: Pocket probing depth was measured to the nearest millimeter using Williams's calibrated periodontal probe. The distance from the gingival margin to the base of the pocket is measured. All measurements were taken prior to the collection of gingival crevicular fluid. In order to minimize potential technique errors and inter-examiner variability

4 Gingival Crevicular Fluid Collection

The selected sites were isolated by cotton rolls, rinsed gently with water and dried with a gentle air spray directed perpendicular to the gingival margin. A saliva ejector was used to avoid salivary contamination of the samples. Gentle removal of the supragingival plaque was completed utilizing dry gauze, and a sterile filter paper strip was gently inserted into the entrance of the selected site until the first sign of resistance was felt. The strip was held in place for 30 seconds Figure1 and Figure2. Attempts were made to avoid the insertion of the strips to the full depth of the pocket, to minimize the risk of contaminating the GCF with blood. Strips contaminated with blood were discarded and an alternative site was sampled Paper points were immersed into sterile Eppendorf vials and stored at−70◦C until further analysis.

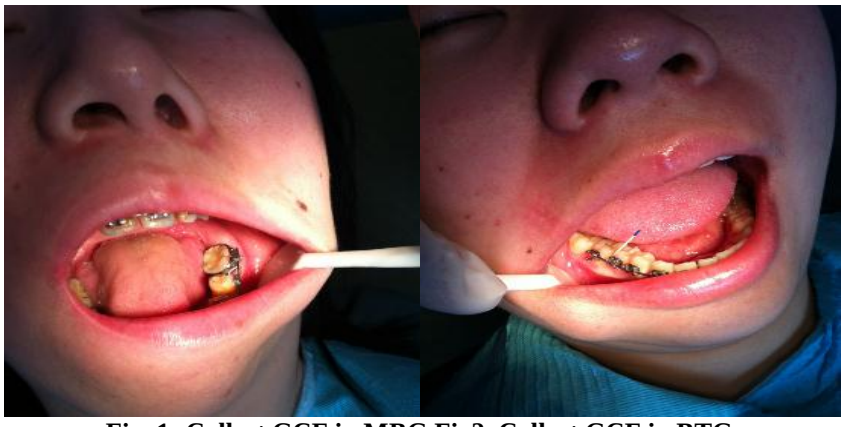

Fig. 1: Collect GCF in MBG Fig2. Collect GCF in BTG.

5 Measurement of IL-1 and IL-6 level

The amounts of IL-1 and IL-6 were measured by enzyme-linked immunosorbent assay sample (ELISA). Bring all reagents and samples to room temperature before use. Centrifuge the sample again after thawing before the assay. It is recommended that all samples and standards be assayed in duplicate.

Prepare all reagents, working standards, and samples as directed in the previous sections. Refer to the Assay Layout Sheet to determine the number of wells to be used and put any remaining wells and the desiccant back into the pouch and seal the Ziploc, store unused wells at 4°C. Add 100 µL of standard and sample per well. Cover with the adhesive strip provided. Incubate for 2 hours at 37°C. A plate layout is provided to record standards and samples assayed.

Remove the liquid of each well, don't wash. Add 100 µL of Biotin-antibody (1x) to each well. Cover with a new adhesive strip. Incubate for 1 hour at 37°C. (Biotin-antibody (1x) may appear cloudy. Warm up to room temperature and mix gently until solution appears uniform).

Aspirate each well and wash, repeating the process two times for a total of three washes. Wash by filling each well with Wash Buffer (200 µL) using a squirt bottle, multi-channel pipette, manifold dispenser, or auto washer, and let it stand for 2 minutes, complete removal

of the liquid at each step is essential to good performance. After the last wash, remove any remaining wash Buffer by aspirating or decanting. Invert the plate and blot it against clean paper towels. Add 100 µL of HRP-avidin (1x) to each well. Cover the micro titer plate with a new adhesive strip. Incubate for 1 hour at 37°C.

Repeat the aspiration/wash process for five times as in step 6. Add 90 µL of TMB Substrate to each well. Incubate for 15-30 minutes at 37°C. Protect from light. Add 50 µL of Stop Solution to each well, gently tap the plate to ensure thorough mixing.

Determine the optical density of each well within 5 minutes, using a micro plate reader set to 450 nm. If wavelength correction is available, set to 540 nm or 570 nm. Subtract readings at 540 nm or 570 nm from the readings at 450 nm. This subtraction will correct for optical imperfections in the plate. Readings made directly at 450 nm without correction may be higher and less accurate.

6 Statistical analysis

IL-1, IL-6 levels of pre/post treatment in each patient were compared by using a paired t-test and to compare the data between two groups t-test were used. For more than 2 groups ANOVA test was used.

RESULTS

Table 1: Gingival Index (GI) Measurement During The Study Period (Mean ± S.D).

Phase of treatment	Control group	Buccal tube	Molar band
Before treatment	1.00 ±0. 40	1.03 ±0. 42	1.04 ±0. 43
After 6 months	1.02 ±0. 41	1.64±0.33	1.85±0.34
After 12 months	1.05±0.37	1.71±0.36	1.89±0.39

Table 1 shows that the gum of three groups of subjects was in good condition before treatment, the luster was normal and there was no difference in Gingival index. After placing fixed appliance 6 months, the oral hygiene turned to be poor, the gum was bleeding when we checked the healthy status used a periodontal probe. Compared with baseline, both of the experimental groups had significant difference ($P < 0.01$, $T=1.700$). While there is no significant difference between buccal tube group and molar band group($P = 2.453 > 0.05$, $T=4.365$).

The oral health status was worse after 12 months, GI continued to rise. Compared with baseline, both of the experimental groups had significant difference ($P < 0.01$, $T=1.701$). While there is no significant difference between buccal tube group and molar band group($P = 1.573 > 0.05$, $T=5.032$).

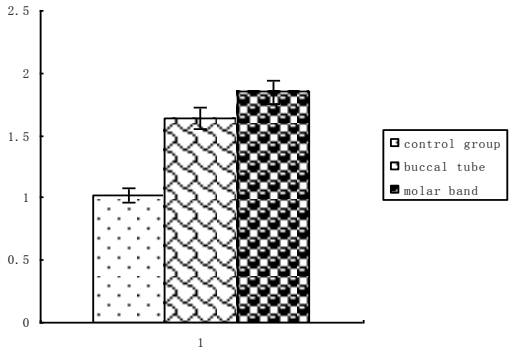

Fig. 1: The comparison of Gingival index among three groups after 6 months treatment.

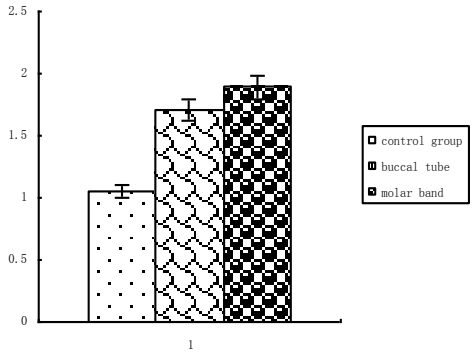

Fig. 2: The comparison of Gingival index among three groups after 12 months treatment.

Table 2: Pocket Probing Depth Measurement During The Study Period (PPD) (Mean ± S.D).

Phase of treatment	Control group	Buccal tube	Molar band
Before treatment	2.00±0.33	2.03±0.38	2.05±0.32
After 6 months	2.05±0. 31	2.70±0.32	3.60±0.35
After 12 months	2.10±0.30	3.35±0.33	3.84±0.29

Table 2 shows that the gum of three groups of subjects was in good condition before treatment, the luster was normal and there was no difference in Pocket probing depth. After placing fixed appliance 6 months, the oral hygiene turned to be poor, there were periodontal pockets in molar band groups, PPD increased significantly. Compared with baseline, both of the experimental groups had significant difference ($P < 0.01$, $T=1.750$). While there is no significant difference between buccal tube group and molar band group($P = 2.530 > 0.05$, $T=4.210$).

The oral health status was worse after 12 months, PPD continued to rise, there were periodontal pockets in molar band group and tube group. Compared with baseline, both of the experimental groups had significant difference ($P < 0.01$, $T=1.740$). While there is no significant difference between buccal tube group and molar band group($P = 1.514 > 0.05$, $T=5.213$).

Compared the average periodontal pocket depths, there was more destruction in periodontal tissue health in molar band group than buccal tube group.

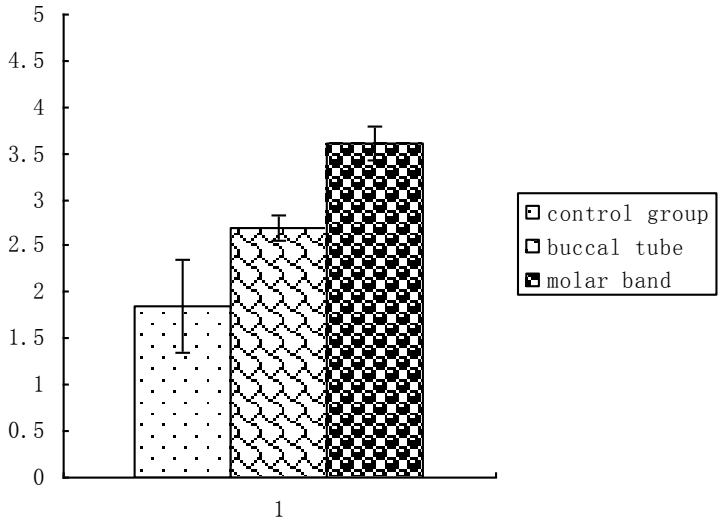

Fig. 3: The comparison of Pocket probing depth among three groups after 6 months treatment.

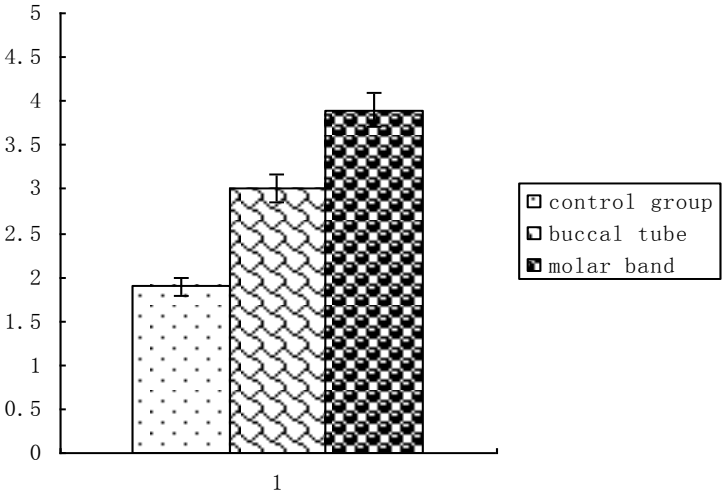

Fig. 4: The comparison of Pocket probing depth among three groups after 12 treatment months treatment.

Table 3: IL-1 Measurement During The Study Period (Mean ± S.D).

Phase of treatment	Control group	Buccal tube	Molar band
Before treatment	0.311±0.010	0.314±0.010	0.318±0.090
After 6 months	0.315±0.012	0.700±0.010	1.007±0.014
After 12 months	0.319±0.080	0.810±0.013	1.390±0.015

Table 3 shows that the content of IL-1 was normal in gingiva groove liquid, there was no difference in the mean value of the three groups of subjects. After placing a fixed appliance for 6 months, the oral hygiene turned to be poor, IL-1 increased significantly. Compared with baseline, both of the experimental groups had significant difference ($P < 0.01$, $T=1.708$) and there was a significant difference between the buccal tube group and molar band group ($P = 0.001<0.01$, $T=1.705$).

The oral health status was worse after 12 months, IL-1 continued to rise, Compared with baseline, both of the experimental groups had significant difference ($P < 0.01$, $T=1.705$) and there was significant difference between buccal tube group and molar band group($P = 0.0012<0.01$, $T=1.714$).

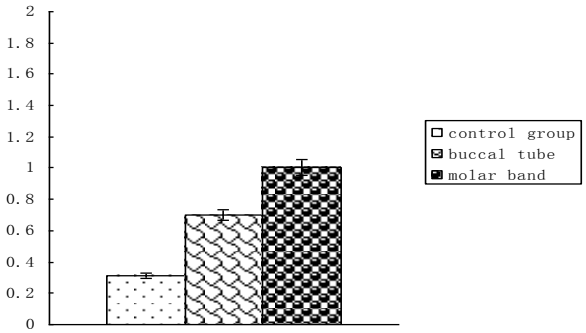

Fig 5: The comparison of IL-1among threegroups after 6 months treatment.

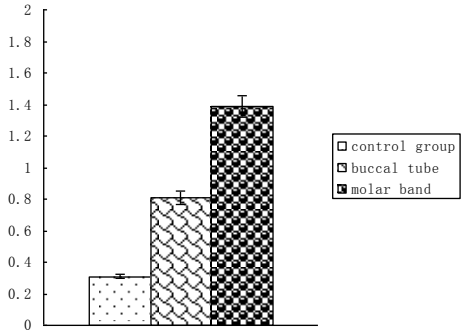

Fig 6: The comparison of IL-1 among three groups after 12 months treatment.

Table 4: IL-6 Measurement During the Study Period (Mean ± S.D).

Phase of treatment	Control group	Buccal tube	Molar band
Before treatment	0.212 ±0.010	0.216 ±0.009	0.213±0.010
After 6 months	0.214 ±0.011	0.549 ±0.010	0.938 ±0.009
After 12 months	0.219 ±0.009	0.607± 0.008	1.279± 0.011

Table 4 shows that the content of IL-6 was normal in gingiva groove liquid, there was no difference in the mean value of the three groups of subjects. After placing a fixed appliance for 6 months, the oral hygiene turned to be poor, IL-6 increased significantly. Compared with baseline, both of the experimental groups had significant difference ($P < 0.01$, $T=1.702$) and there was a significant difference between buccal tube group and molar band group($P = 0.001<0.01$, $T=1.708$).

The oral health status was worse after 12 months, IL-6 continued to rise. Compared with baseline, both of the experimental groups had significant difference ($P < 0.01$, $T=1.701$) and there was a significant difference between buccal tube group and molar band group($P = 0.0013<0.01$,$T=1.514$).

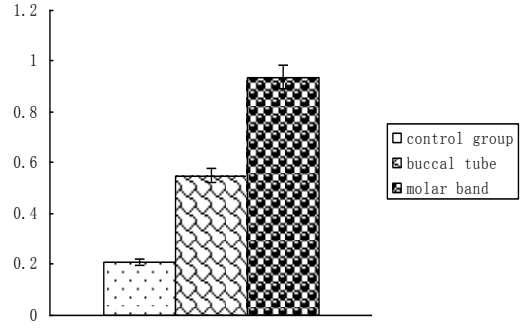

Fig 7: The comparison of IL-6 among three groups after 6 months treatment.

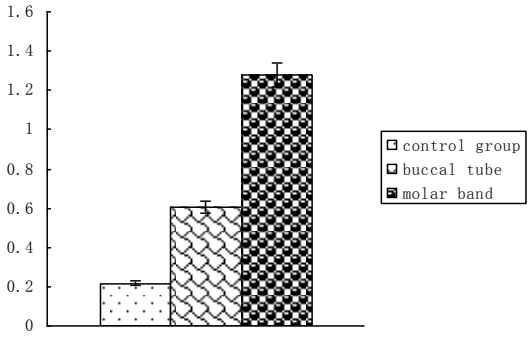

Fig 8: The comparison of IL-6 among three groups after 12 months treatment.

DISCUSSION

In this study, we bonded both buccal tube and molar band in the mouth of one patient, effectively avoiding the experimental error caused by individual differences. Before the treatment, dentists would mission the patients who consciously paid attention to oral health, each index in the treatment of the first a few months always cannot reflect the real situation of the oral, so we chose 6 months after treatment to test the indicators in the study. After we placed the fixed appliance 12 months, some patients finished their therapy and the molars of the majority of the patients had moved to some extent. When testing in this stage, the indicators can reflect the influence by buccal tubes and molar bands on periodontal health more effectively. By clinical examination combined with experimental data, we can evaluate the influence by buccal tubes and molar bands on periodontal health comprehensively. With the development of adhesive materials, we used glass-ionomer to bond the molar band, it has a better performance. To pay attention to the edge of the molar band and buccal tube, clean the adhesive materials, we effectively avoid the stimulation of the periodontal tissue. Compared with molar band, buccal tube is easier to fall off from the tooth surface, but we only consider the side effects on periodontal health in this study. After placing fixed appliance 6 months, there were periodontal pockets in molar band group, and after 12 months, there were periodontal pockets in both molar band group and tube group. This may be caused by irritation for the gum, which is called Pseudo periodontal pocket. We bonded the buccal tube in the middle of the tooth crown, neither near by the maxillary teeth, avoiding the occlusal interferences, nor near the gum to avoid stimulating it. If the buccal tube fell off from the tooth, we rebonded it immediately, so it would had less affection to the experiment.

In this study we demonstrated that Gingival index (GI) and Pocket probing depth (PPD) have been increased after 6 and 12 months after treatment with molar bands and buccal tubes. This increase in GI and PPD was higher in molar band group than buccal tube group. On the other hand, inflammation which was investigated through the measurement of inflammatory cytokines indicated that there was increase in both IL-1 and IL-6 during the course of treatment. This increase in IL-1 and IL-6 was also higher in molar band than buccal tube. These results indicated that both treatment (molar bands and buccal tubes) had an inflammatory effect on the health of orthodontic teeth. However, the effect was less upon using buccal tubes. Our study also matched the previous studies. For example, Naga, Sri M. and Sosa K. V. showed that individuals with orthodontics molar bands have more plaque accumulation, gingivitis and gingival enlargement than individuals without any treatment. Alexander, Boyd, and Baumrind also found more plaque accumulation and gingival inflammation around banded molars.

The results of the present study tend in general to confirm the hypotheses and finding of other similar studies. The data revealed that[57] molar bands and buccal tubes had significantly more plaque accumulation and gingival inflammation than bonded molars[58] molar bands showed more plaque accumulation and gingival inflammation than buccal tubes before and with advancing time during orthodontic treatment. An alternative possible explanation for increasing pocket depth is the mechanical injury[59] caused by the subgingival placement of orthodontic bands. Many of our findings were in agreement with those of previous studies. These included the observations that molars with orthodontic bands had more plaque accumulation, gingival inflammation.[60,61] Further, they had a quantitative and qualitative different type of bacterial flora whose presence was positively associated with gingival inflammation. Only mesiobuccal surfaces were used as study sites. They may have led to under estimation of the actual amount of periodontal inflammation. However, a previous study[62] revealed evidence that distal proximal surfaces show recordings of periodontal destruction similar to those of mesial surfaces. Buccal surfaces were not sampled because these surfaces tend to show less periodontal inflammation than proximal sites[63,64] and they are more likely to show teeth brush abrasion.[65] In this study, molar band group which had the greatest loss of attachment were invariably the subjects with the poorest

plaque removal and highest levels of gingival inflammation. This observation was in agreement with those of previous studies.[66-69] However, it has been reported that when loss of attachment measurements are made in inflamed tissue, the probe tip tends to penetrate through the secular epithelium into connective tissue. Many of our findings consistent with those of previous studies. These include the observations that molars with orthodontic bands have more plaque accumulation in gingival inflammation.[70,71] and loss of attachment. Only mesiobuccal surfaces were used as study sites.

In this study, an effort was made to investigate an important clinical question without conscious bias and using a reasonably well-balanced prospective design. We believed that this effort was fairly successful and this study had produced useful information which augments our previous knowledge. However, the study had several consequential flaws which we indicated in an attempt to create improved conditions for further research in this area.

In this study we demonstrated that through the measurement of inflammatory cytokines indicated that there was increase in both IL-1 and IL-6 during the course of treatment. This increase in IL-1 and IL-6 was also higher in molar band than buccal tube. These results indicated that both treatments (molar band and buccal tube) have an inflammatory effect on periodontal health.

Our study also matches previous studies Irwin and Myrillas[72] reviewed the biological functions of IL-6 and found them to be specifically related to tissue destruction in the periodontal site. In addition, Yakovlev et al[73] found that levels of IL-1 β and IL-6 were significantly higher in inflamed as compared with non-inflamed gingival tissues in young adults, while Shimizu et al.[74] demonstrated that IL-6 was produced by IL-1β from PDL cells. Macrophages have the ability to produce cytokines, such as IL-1β and IL-6, the levels of which are known to increase during orthodontic tooth movement.[75] The number and distribution patterns of RANKL and RANK-expressing osteoclasts change when excessive orthodontic force is applied to periodontal tissue, and IL-1β and TNF- α were shown to be expressed in osteoclasts in pathological status rat periodontal tissues.[76] Shiotani et al.[77] also showed the presence of RANKL in periodontal tissues during experimental tooth movement of rat molars. Therefore, it is suggested that RANKL is regulated by inflammatory cytokines in the PDL in response to mechanical stress. The peripheral sensory nervous system contributes to the development of acute and chronic inflammatory processes through local release of neuropeptides. SP, a sensory neuropeptide released from the peripheral endings of sensory nerves during inflammation, can modify the secretion of pro-inflammation cytokines from immunocompetent cells and has also been reported to induce the secretion of IL-1 β, IL-6, and TNF-α from monocytes.[78,79] CGRP, another major sensory neuropeptide, has also been found to evoke the release of IL-6 and IL-8 from synovial fibroblasts in patients with rheumatoid arthritis.[80] In another study, SP and CGRP both stimulated the release of IL-6 and TNF- α from a human bronchial epithelial cell line.[81] Further, we recently reported that CGRP and SP significantly stimulated the production of PGE 2, IL-1 β, IL-6, TNF- α, and RANKL in HDP cells.[82,83] Based on these results, we consider that CGRP and SP may be involved in pulpal inflammation that occurs during orthodontic tooth movement.

Compared with molar band, buccal tube is easier to fall off from the tooth surface, but we only consider the side effects on periodontal health in this study. After placing fixed appliance 6 months, there were periodontal pockets in molar band group, and after 12 months, there were periodontal pockets in both molar band group and tube group. This may be caused by irritation of the gums, which is called Pseudo periodontal pocket.

Of course, there was a defect in our experiment. We used orthodontic brackets which were made in China, if we chose imported brackets, such as 3M, which can fit the tooth surface better, we would make better results. In fact, at the end of the treatment for three months, we should still follow the indicators. But time is limited in this experiment, we failed to achieve that goal.

CONCLUSION

1. In orthodontic treatment, buccal tubes caused less inflammatory response on periodontal health than molar bands, which was helpful to achieve better treatment effect.
2. We concluded that buccal tube was more conducive to periodontal health than molar band.

REFERENCES

1. Ong MMA, Wang H. Periodontic. and orthodontic treatment in adults. Am [J] Orthod Dentofacial Orthop, 2002; 12(2): 420–428.
2. Chambrone L, Goldenberg FC. Resposta dos tecidosmucogengivais em dentes submet- idos a tratamento ortodontico. [J] Bras Ortodon Ortop Facial, 2006; 11: 315–32.
3. Rego RO, Oliveira CA, dos Santos-Pinto A, et al. Clinical and microbiological studies of children and adolescents receiving orthodontic treatment. Am [J] Dent., 2010; 23: 317–323.
4. Demling A, Heuer W, Elter C, et al. Analysis of supra- and subgingival long term biofilm formation on orthodontic bands. Eur [J] Orthod, 2009; 31: 202–206.
5. Banks P, Macfarlane TV. Bonded versus banded first molar attachments: a randomized controlled clinical trial. [J]Orthod, 2007; 34: 128–136.
6. Paschos E, Limbach M, Teichmann M, et al. Orthodontic attachments and chlorhexidine-containing varnish effects on gingival health. Angle Orthod, 2008; 78: 908–916.

7. Matos M, Vianna MIP, Pitta A. Controle quı´mico e mecaˆ nicode placa em pacientes ortodoˆ nticos. Uma ana´ lise porgrupo de dentes de acordo com o acesso´ rio ortodo ˆ nticoempregado. R Dental Press Ortodon Ortop Facial, 2003; 8: 87–93.
8. Kim K, Heimisdottir K, Gebauer U, et al. Clinical and microbiological findings at sites treated with orthodontic fixed appliances in adolescents. Am [J] Orthod Dentofacial Orthop, 2010; 137: 223–228.
9. Ristic M, Svabic MV, Sasic M, et al. Effects of fixed orthodontic appliances on subgingival microflora. Int [J] Dent Hyg., 2008; 6: 129–136.
10. Gastel JV, Quirynen M, Teughe W, et al. Longitudinal changes in microbiology and clinical periodontal variables after placement of fixed orthodontic appliances.[J] Periodontol, 2008; 79: 2078–2086.
11. Stuteville OH. Injuries caused by orthodontic appliances and Methods of preventing these injuries. [J]ADA, 1937; 24: 1494–507.
12. Skillen WG. Krivanek FJ. Effects of orthodontic appliances on gingival tissues. Northw Uni Bull, 1938; 38: 18–22.
13. Brandtzaeg P. Local factors of resistance in the gingival area. [J] Periodont Res., 1966; 1: 19–42,
14. L €oe H. Physiology of the gingival pocket. Acad Rev Calif Acad Periodontal, 1965; 13: 6-14.
15. Quirynen M, Marechal M, Busscher H, et al. The in fl uence of surface characteristics on the early bacterial colonization of intra-oral hard surfaces[J] Clin Dent., 1988; 1(Suppl A): A14-9.
16. Quirynen M, Marechal M, Busscher HJ, et al. The in fl uence of surface free-energy on planimet-ric plaque growth in man. J Dent Res., 1989; 68: 796-9.
17. Quirynen M, Marechal M, Busscher HJ.et al. The influence of surface free energy and surface roughness on early plaque formation. An in vivo study in man.[J] Clin Periodontol, 1990; 17: 138-44.
18. Quirynen M, Bollen CM. The in fluence of surface roughness and surface-free energy on supra- and subgingival plaque formation in man. A review of the literature. [J] Clin Periodontol, 1995; 22: 1-14.
19. Quirynen M, Dekeyser C, van Steenberghe D. The influence of gingival in flammation, tooth type, and timing on the rate of plaque formation. [J] Periodontol, 1991; 62: 219-22.
20. Ramberg P, Axelsson P, Lindhe J. Plaque formation at healthy andinflamed gingival sites in young individuals. [J] Clin Periodontol, 1995; 22: 85-8.
21. Rowshani B, Timmerman MF, Van der Velden U. Plaque development in relation to the periodontal condition and bacterial load of the saliva. [J] Clin Periodontol, 2004; 31: 214-8.
22. Socransky SS, Haffajee AD, Smith C, et al. Relation of counts of microbial species to clinical status at the sampled site. [J] Clin Periodontol, 1991; 18: 766-75.
23. van Gastel JL, Quirynen M, Teughels W, et al. Influence of bracket design on microbial and periodontal parameters invivo. [J] Clin Periodontol, 2007; 34: 423-31.
24. Gomes SC, Varela CC, da Veiga SL, et al. Periodontal conditions in subjects following orthodontic therapy. A preliminary study. Eur[J] Orthod, 2007; 29: 477-81.
25. Thomson WM. Orthodontic treatment outcomes in the long term:findings from a longitudinal study of New Zealanders. Angle Or-thod, 2002; 72: 449-55.
26. Janson G, Bombonatti R, Brandao AG, et al. Comparative radiographic evaluation of the alveolar bone crest after orthodontic treatment. Am [J] Orthod Dentofacial Orthop, 2003; 124: 157-64.
27. Lamster IB. The host response in gingival crevicular fluid: potentialapplications in periodontitis clinical trials. [J] Periodontol, 1992; 63: 1117-23.
28. McCulloch CA. Host enzymes in gingival crevicularfluid as diagnostic indicators of periodontitis. [J] Clin Periodontol, 1994; 21: 497-506.
29. Ren Y, Maltha JC, Van 't Hof MA, et al. Cytokine levels in crevicular fluid are less responsive to orthodontic force in adults than in juveniles.[J] Clin Periodontol, 2002; 29: 757-62.
30. Ren Y, Hazemeijer H, de Haan B, et al. Cytokine profiles in crevicular fluid during orthodontic tooth movement of short and long durations. [J] Periodontol, 2007; 78: 453-8.
31. Giannopoulou C, Mombelli A, Tsinidou K, et al. Detection of gingival crevicular fluid cytokines in children and adolescents with and without fixed orthodontic appliances. Acta Odontol Scand, 2008; 66: 169-73.
32. Romano M, Sironi M, Toniatti C, et al. Role of IL-6 and its soluble receptor in induction of chemokines and leukocyte recruitment. Immunity, 1997; 6: 315-25.
33. Dinarello C. A. (1989): Interleukin-1 and its biologically related cytokines. Adv. Immunol., 44: 153–205.
34. Egan C. G., Lockhart J. C., Ferrell W. R., et al. Pathophysiological basis of acute inflammatory hyper aemia in the rat knee: roles of cyclo-oxygenase-1 and -2. [J]Physiol., 539(Pt 2): 579–587.
35. Heinrich P.C., Castell J.V. and Andus T. Interleukin-6 and the acute phase response. Biochem. [J]., 1990; 265: 621–636.
36. Spears R., Oakes R., Bellinger LL. et al. Tumour necrosis factor-alpha and apoptosis in the rat temporomandibular joint. Arch. Oral Biol., 2003; 48(12): 825-34.
37. Walsh N. C. and Gravallese E. M: Bone loss in inflamma-tory arthritis: mechanisms and treatment strategies. Curr. Opin. Rheumatol, 2004; 16: 419–427.
38. Vandevska-Radunovic V.: Neural modulation of inflammatory reactions in dental tissues incident to orthodontic tooth movement. A review of the literature. Eur. [J]. Orthod, 1999; 21: 231–247.
39. Dinarello C. A.: Interleukin-1. Dig. Dis. Sci., 1988; 33(suppl.): 25S–35S.

40. Gowen M., Wood D. D., Ihrie E. J., et al: An interleukin 1 like factor stimulates bone resorp-tion in vitro. Nature, 1983; 306: 378–380.
41. Heath J. K., Saklatvala J., Meikle M. C., et al: Pig interleukin 1 (catabolin) is a potent stimulator of bone resorption in vitro. Calcif. Tissue Int., 1985; 37: 95–97.
42. Stashenko P., Obernesser M. S. and Dewhirst F. E.: Effect of immune cytokines on bone. Immunol. Invest, 1989; 18: 239–249.
43. Dinarello C. A. and Savage N.: Interleukin-1 and its receptor. Crit. Rev. Immunol, 1989; 9: 1–20.
44. Lo Y. J., Liu C. M., Wong M. Y., et al: Interleukin 1beta-secreting cells in inflamed gingival tissue of adult periodontitis patients. Cytokine, 1999; 11: 626–633.
45. Kanda-Nakamura C., Izumi Y. and Sueda T.: Increased expression of interleukin-1 receptors on fibroblasts derived from inflamed gingiva. [J] Periodontol, 1996; 67: 1267–1273.
46. Linkhart T. A., Linkhart S. G., MacCharles D. C., et al: Interleukin-6 messenger RNA expressionand interleukin-6 protein secretion in cells isolated from normal human bone: regulation by interleukin-1. J. Bone Miner. Res., 1991; 6: 1285–1294.
47. van Damme J., Cayphas S., Van Snick J., et al: Purification and characterization of human fibroblast-derived hybridoma growth factor identical to T-cell-derived B-cell stimulatory factor-2 (interleukin-6). Eur. J. Biochem, 1987; 168: 543–550.
48. Hirano T. and Kishimoto T. Interleukin-6. In Roberts A.B. (ed.): Handbook of experimental pharmacology. Springer, Berlin, 1990; 633–665.
49. Ishimi Y., Miyaura C., Jin C. H., et al. IL-6 is produced by osteoblasts and induces bone resorption. [J] Immunol, 1990; 145: 3297–3303.
50. Kurihara N., Bertolini D., Suda T., et al. IL-6 stimulates osteoclast-like multinucleated cell formation in long term human marrow cultures by inducing IL-1 release. [J] Immunol, 1990; 144: 4226–4230.
51. Lowik C. W., van der Pluijm G., Bloys H., et al.: Parathyroid hormone (PTH) and PTH-like protein (PLP) stimulate interleukin-6 production by osteogenic cells: a possible role of interleukin-6 in osteoclas-togenesis. Biochem. Biophys. Res. Commun, 1989; 162: 1546–1552.
52. Bartold P. M. and Haynes D. R. Interleukin-6 production by human gingival fibroblasts. J. Periodontal. Res., 1991; 26: 339–345.
53. May L. T., Ghrayeb J., Santhanam U., et al. Synthesis and secretion of multiple forms of beta 2-inter-feron/B-cell differentiation factor 2/hepatocyte-stimulating factor by human fibroblasts and monocytes. J. Biol. Chem., 1988; 263: 7760–7766.
54. Irwin C. R. and Myrillas T. T. The role of IL-6 in the pathogenesis of periodontal disease. Oral Dis., 1998; 4: 43–47.
55. Yakovlev E., Kalichman I., Pisanti S., Shoshan S. and Barak V. Levels of cytokines and collagen type I and type III as a function of age in human gingivitis. J. Periodontol, 1996; 67: 788–793.
56. Shimizu N., Ogura N., Yamaguchi M., Goseki T., Shibata Y., Abiko Y., Iwasawa T. and Takiguchi H. Stimulation by interleukin-1 of interleukin-6 production by human periodontal ligament cells. Arch. Oral Biol., 1992; 37: 743–748.
57. Butler J, Dowling P. Orthodontic bonded retainers. [J] Ir Dent Assoc., 2005; 51(1): 29-32.
58. Newmwan M, Takei H, Carranza F. Carranza's clinical periodontology. 9th ed. Saunders, 2003; 191-193.
59. Lundstrom F, Hamp S-E, Nyman S. Systematic plaque control in children undergoing long-term orthodontic treatment. Eur[J] Orthod, 1980; 2: 27-39.
60. Kloehn JS, Pfeifer JS. The effect of orthodontic treatment on the periodontium. Angle Orthod, 1974; 44: 127-134.
61. Pearson LE. Gingival height of the lower central incisors in the orthodontically treated and untreated. Angle Orthod, 1968; 28: 337-339.
62. Schei O, Waerhaug J, Lovadal A, et al. Alveolar bone loss as related to oral hygiene and age. [J] Periodontol, 1959; 20: 7-16.
63. Anerud A, Loe H, Boyen H, et al. The natural history of periodontal disease in man. Changes in gingival health and oral hygiene before 40 years of age. J Periodontal Res., 1979; 14: 526-540.
64. Clerehugh V, Lennon MA, Worthington HV. Aspects of the validity of buccal loss of attachment ≥ 1mm in studies of early periodontitis. [J] Periodontol, 1988; 14: 207-210.
65. Zachrisson BU. Cause and prevention of injuries to teeth and supporting structures during orthodontic treatment. Am [J] Orthod, 1976; 69: 285-300.
66. Sjolien T, Zachrisson BU. Periodontal bone support and tooth length An orthodontically treated and untreated persons. Am [J] Orthod, 1973; 64: 28-37.
67. Zachrisson BU, Alnaes L. Periodontal condition in orthodontically treated and untreated individuals. I. Loss of attachment, gingival pocket depth and clinical crown height. ANGLE Oarisoo, 1973; 43-402- 411.
68. Hamp 5, Lundstroin F, Nyman 5. Periodontal conditions in adolescents subjected to multiband orthodontic treatment with controlled oral hygiene. Eur [J]Orthod, 1982; 4: 77-86.
69. Boyd RL, LeggottPj, Quinnk5, et al. Periodontal implications of orthodontic treatmentin adults with reduced or noimal periodontal tissues versus those of adolescents. Am *I* Orthod Dentofacial Orthop, 1989; 96: 191-199.
70. Pearson LE. Gingival height of the lower central incisorsin the orthodonticaliy treatedand untreated. ANGLE ORTtIoO, 1968; 28: 337-339.
71. Kennedy DB, joondeph DR. Osterberg 5K, et al. The effect of extraction and orthodontic treatment

on dentoalveolar support. Am[J] Orthod, 1983; 84: 183-190.
72. Irwin C. R. and Myrillas T. T. The role of IL-6 in the pathogenesis of periodontal disease. Oral Dis., 1998; 4: 43–47.
73. Yakovlev E., Kalichman I., Pisanti S., et al. Levels of cytokines and collagen type I and type III as afunction of age in human gingivitis. [J]. Periodontol, 1996; 67: 788–793.
74. Shimizu N., Ogura N., Yamaguchi M., et al. Stimulation by interleukin-1 of interleukin-6 production by human periodontal ligament cells. Arch. Oral Biol., 1992; 37: 743–748.
75. Saito M., Saito S., Ngan P.W., et al.: Interleukin 1 beta and prostaglandin E are involved in the response of periodontal cells to mechanical stress in vivo and in vitro. Am. [J]. Orthod. Dentofacial Orthop, 1991; 99: 226–240.
76. Ogasawara T., Yoshimine Y., Kiyoshima T., et al. In situ expression of RANKL, RANK, osteoprotegerin and cytokines in osteoclasts of rat periodontal tissue. [J]. Periodontal Res., 2004; 39: 42–49.
77. Shiotani A., Shibasaki Y. and Sasaki T. Localization of receptor activator of NFkappaB ligand, RANKL, in periodontal tissues during experimental movement of rat molars. [J]. Electron Microsc., 2001; 50: 365–369.
78. Lotz M., Vaughan J. H. and Carson D. A. Effect of neuropeptides on production of inflammatory cytokines by human monocytes. Science, 1988; 241: 1218–1221.
79. Rameshwar P., Ganea D. and Gascon P. In vitrostimulatory effect of substance P on hematopoiesis. Blood, 1993; 81: 391–398.
80. Raap T., Justen H. P., Miller L. E., et al.: Neurotransmitter modulation of interleukin 6 (IL-6) and IL-8 secretion of synovial fibroblasts in patients with rheumatoid arthritis compared to osteoarthritis. [J]. Rheumatol, 2000; 27: 2558–2565.
81. Veronesi B., Carter J. D., Devlin R. B., et al. Neuropeptides and capsaicin stimulate the release of inflammatory cytokines in a human bronchial epithelial cell. Neuropeptides, 1999; 33: 447–456.
82. Kojima T., Yamaguchi M. and Kasai K. Substance P stimulates release of RANKL via COX-2 expression in human dental pulp cells. Inflamm. Res., 2006; 55(2): 78-84.
83. Yamaguchi M., Kojima T., Kanekawa M. et al. Neuropeptides stimulate production of interleukin-1 beta, interleukin-6, and tumor necrosis factor-alpha in human dental pulp cells. Inflamm. Res., 2004; 53: 199–204.