Biomechanical effects of fixed partial denture therapy on strain patterns of the mandible

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Statement of problem. The mandibular posterior 3-unit fixed partial denture (FPD) is a conventional prosthodontic therapy and presumably has an effect on the direction and magnitude of occlusal forces and, thus, on the biomechanical environment of the mandible, which may in turn affect bone structure. However, the impact of FPD therapy on mandibular biomechanics is unknown.

Purpose. The purpose of this study was to test the hypothesis that 3-unit FPD therapy alters strain patterns in the mandible during loading.

Material and methods. Four human cadaver mandibles missing first molars were bilaterally fixed and artificially loaded on each tooth individually. Surface cortical bone strains were measured with multiple strain gauges during loading of up to 250 N. Next, 3-unit FPDs with a chamfer finish line were fabricated using Type IV gold alloy. Strain measurements were conducted in the same manner to assess differences in strain patterns before and after therapy. Paired-sample tests for metric and angular data were used to assess difference in strain pattern before and after therapies (α=.05).

Results. When loading was applied on the teeth not involved in FPD therapy, no differences were found before and after FPD placement. When the posterior retainers were loaded, the strain distribution differed (*P*=.01); on the buccal cortices, strain levels increased posteriorly but decreased significantly anteriorly. However, these differences were less than 100 $\mu\epsilon$, and the overall deformation pattern of the mandible after the FPD therapy was similar to that before FPD therapy. Strain distributions when the pontic was loaded were similar to those when the posterior retainer was loaded.

Conclusion. Three-unit FPD therapy did not alter the overall deformation pattern of the mandible during loading. (J Prosthet Dent 2006;95:55-62.)

CLINICAL IMPLICATIONS

Implant therapy has become a first choice for the replacement of a single missing tooth. However, the conventional 3-unit FPD is still applicable and required for situations in which patients are unable to accept implant therapy. This study suggests that the conventional 3-unit FPD does not alter the overall functional strain pattern of the mandible. It further indicates that the pontic biomechanically compensates for the functional loss due to tooth loss. The findings provide justification for 3-unit FPD therapy as an accepted treatment option for the restoration of a single missing tooth.

Recent advances in implant dentistry have resulted in implant therapy as the first choice for the replacement of a single missing tooth, which had been traditionally treated with a 3-unit fixed partial denture (FPD). Implant therapy seems to have clinical advantages over FPD therapy: no preparation of abutment teeth, no risk of developing secondary caries, better access for hygiene, less incidence of periodontal disease, and better preservation of alveolar bone. However, when the alveolar ridge is severely resorbed and the dentist is unable to place an implant in proper position, an implant site preparation surgery such as mandibular symphysis grafting, ramus grafting, sinus floor elevation, or guided bone regeneration is necessary to generate a predictable outcome. Even though implant therapy may be an ideal treatment modality for a single missing tooth, a 3-unit FPD is an option for patients who do not accept implant therapy due to severe systemic disease, financial status, surgery phobia, or other reasons. Mandibular posterior 3-unit FPD therapy is a reliable prosthetic therapy for a single missing molar. Although FPD therapy

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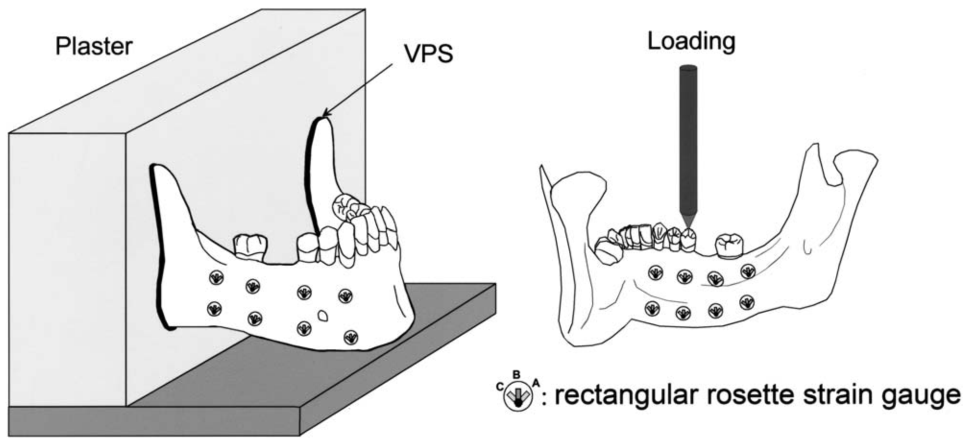

Fig. 1. Schematic drawing of mandible showing positions of 8 rosette strain gauges on buccal and lingual cortices. Rami of mandible were covered with VPS and fixed in plaster. Plaster was secured in loading apparatus. Static loading was applied to each tooth individually. Orientation of central element *(B)* of each rosette strain gauge is superoinferiorly aligned, corresponding to vertical axis of mandible corpus; alignment of 3 gauge elements C/B/A is as follows: *C*, Left; *B*, middle; *A*, right.

inherently has the disadvantage of unavoidable reduction of abutment teeth, treatment outcome can be excellent as long as standard clinical procedures are followed.[1-3] Despite the possible effect of an FPD on mandibular biomechanics and, thus, on mandibular bone structure, the biomechanical effects of FPD placement are unknown.

It is known that the mandible deforms during function.[4-9] This deformation generates functional stresses, which contribute to bone development and maintenance.[10-16] Because 3-unit FPD therapy results in a rigid connection of independent teeth, FPD placement may alter the stress distribution in the mandible during function, which in turn may alter bony structure around the abutment teeth. The alteration in functional stress distribution may affect the remodeling process of the mandible and its internal structure over the long term. Evidence to support this theory is the occurrence of subpontic osseous hyperplasia (SOH). SOH, known as osseous proliferation beneath the pontic of the FPD, is occasionally seen clinically and is usually found in the posterior region of the mandible.[17-20] Although the etiology of SOH is not clearly elucidated, the mechanical stimuli transmitted through an FPD during mastication is a likely candidate to induce SOH.[21] It is possible that the posterior 3-unit FPD modifies the patterns of mandibular bone strain, which then alters the cortical structure of the mandible, despite the absence of clinically observed changes in shape.

The objective of this study was to test the hypothesis that 3-unit FPD therapy alters the strain distribution of the mandible during simulated function. For this purpose, human cadaver mandibles were artificially loaded and surface cortical bone strain was measured before and after FPD placement.

MATERIAL AND METHODS

Surface bone strains were measured during artificial loading of cadaver mandibles missing 1 molar. FPD therapy was then performed on the same mandibles, and strains during loading were again recorded. Strains before and after FPD placement were compared to each other. Four fresh frozen human mandibles, from individuals who were 40 to 82 years of age at death, were obtained from the Gross Anatomy Laboratories at Baylor College of Dentistry. Causes of death were not related to primary bone diseases. The mandibles were completely dentate, although right first molars were missing. Sixteen rosette strain gauges (FRA-1-11-11; Tokyo Sokki Kenkyujo, Tokyo, Japan) were attached with cyanoacrylate cement (Zapit; Dental Ventures of America, Corona, Calif) onto the right side of the mandible: 8 on the lingual surface and 8 on the buccal surface (Fig. 1). On each surface, the upper 4 gauges were placed at the boundary between the alveolar bone and mandibular body, and the lower 4 were attached about 10 mm above the lower border of the mandible. The anteroposterior positions of the 4 gauges in each row were below the canine, second premolar, second molar, and distal to the second molar (Table I). The strain gauges were connected to strain conditioners and amplifiers (Model 2120; Vishay Micro-Measurements, Raleigh, NC) through a switch box. The mandibles were bilaterally fixed at the condyles and angles. In humans the condyles move slightly during mastication.[22,23] To simulate the in vivo situation, the condyles and angles were molded with vinyl polysiloxane (VPS) impression material (Reprosil; Dentsply Caulk, Milford, Del). The VPS layers were approximately 1.0 mm in thickness. Next, the covered portions of the mandibles were embedded into blocks of dental plaster (Plaster Lab; Garreco Inc, Heber Springs, Ark). Finally, the blocks supporting the mandibles were secured in the loading apparatus with screws. In this manner, the mandibles could deform with minimum restriction during loading (Fig. 1). Static loads were applied to each tooth individually. The loading positions were the buccal cusps of the left and right premolars, and the mesiobuccal cusp and the central fossa of the left and right second molar. The magnitude used was 150 N for the premolars and 250 N for the molars. A magnitude approximate to human physiological occlusal force was selected based on previous reports.[24,25] During loading, strain measurements were directly stored in a computer through an analog-digital converting board (DT2821; Data Translation, Marlboro, Mass), with the sampling rate of 62.5 Hz. Afterwards, maximum and minimum principal strains and their directions were calculated. A rosette strain gauge is composed of 3 linear gauges placed at 0-, 45-, and 90-degree positions (Fig. 1). By measuring strains of each gauge, maximum principal strain ϵ_1, minimum principal strain ϵ_2, and their angles were established through the following standard formulas[25]:

$$\epsilon 1 = \frac{1}{2}(\epsilon a + \epsilon c) + \frac{1}{2}\sqrt{(\epsilon a - \epsilon c)^2 + (2\epsilon b - \epsilon a - \epsilon c)^2}$$

$$\epsilon 2 = \frac{1}{2}(\epsilon a + \epsilon c) - \frac{1}{2}\sqrt{(\epsilon a - \epsilon c)^2 + (2\epsilon b - \epsilon a - \epsilon c)^2}$$

$$\tan 2\theta = \frac{2\epsilon b - \epsilon a - \epsilon c}{\epsilon a - \epsilon c}$$

where ϵa, ϵb, and ϵc are the strains of each gauge component, and θ is the angle between the direction of $\epsilon 1$ and the A-gauge (Fig. 1).

After the series of strain measurements of the intact mandible were completed, the mandibles were displaced from the loading apparatus and 3-unit FPD therapies were performed. Retainers were placed on the second premolar and molars. Teeth were prepared with a chamfer finish line. Impressions were made with VPS. The FPDs were cast in Type IV gold alloy (Ney-Oro CB; Dentsply Ceramco, Burlington, NJ) and cemented with glass-ionomer cement (Vitremer; 3M ESPE, St. Paul, Minn). Care was taken to protect the strain gauges during FPD placement, and no damage was apparent. The mandibles with the FPDs were then positioned in the loading apparatus and the strain measurements were performed as previously described. In addition, loads of 150 N and 250 N were placed on the buccal cusps and the central fossa of the pontic. Strain patterns were compared before and after the FPD therapy.

Data were stored in a computer spreadsheet (Microsoft Excel 2000; Microsoft, Redmond, Wash) and analyzed using a statistical analysis program (MINITAB 14; Minitab, State College, Pa). Strains in microstrain units of principal tension (maximum strain, ϵ_1) and compression (minimum strain, ϵ_2), and the orientation of principal tension were calculated by using the strain data analyzer software (DAS-100A 2003; Kyowa Electronic Instruments, Tokyo, Japan). The orientations of ϵ_1 were originally calculated in degrees relative to the A-element of each gauge, and the results were transformed to be relative to the B-element or the central element to indicate their relationships with anatomical features or the orientation of the mandible (Fig. 1). Descriptive statistics, including mean values and SDs, were calculated for all measurements. Overall differences before and after the FPD therapy were assessed with nonparametric Friedman analysis of variance (ANOVA) tests. Paired-sample *t* tests were used to compare strain magnitudes of specific sites either individually (before and after the FPD therapy) or within the group. Circular descriptive statistics, including angular mean values, circular standard errors, and Rayleigh's tests of uniformity,[26] were calculated with statistical analysis software (Oriana Statistical Analysis Program 2.02; Kovach Computing Services, Wales, UK). The Hotelling paired-sample test was used to compare orientations of principal strains. An alpha level of .05 was used for all statistical analyses.

Table I. Strain gauge sites

Gauge site	Surface	Level	Position
1	Lingual	Alveolar	Below the canine
2	Lingual	Alveolar	Below the second premolar
3	Lingual	Alveolar	Below the second molar
4	Lingual	Alveolar	Distal to the second molar
5	Lingual	Basal	Below the canine
6	Lingual	Basal	Below the second premolar
7	Lingual	Basal	Below the second molar
8	Lingual	Basal	Distal to the second molar
9	Buccal	Alveolar	Below the canine
10	Buccal	Alveolar	Below the second premolar
11	Buccal	Alveolar	Below the second molar
12	Buccal	Alveolar	Distal to the second molar
13	Buccal	Basal	Below the canine
14	Buccal	Basal	Below the second premolar
15	Buccal	Basal	Below the second molar
16	Buccal	Basal	Distal to the second molar

Reliability of strain measurements

The reliability of strain measurements was assessed by repeated loading on the same location. Linear and angular paired-sample tests were used to compare the magnitude and orientation of principal strains between repeated tests. There were no significant differences among the mean shear strains of all gauge sites, and no significant differences were found in ratios of tensile to compressive strain either, according to the

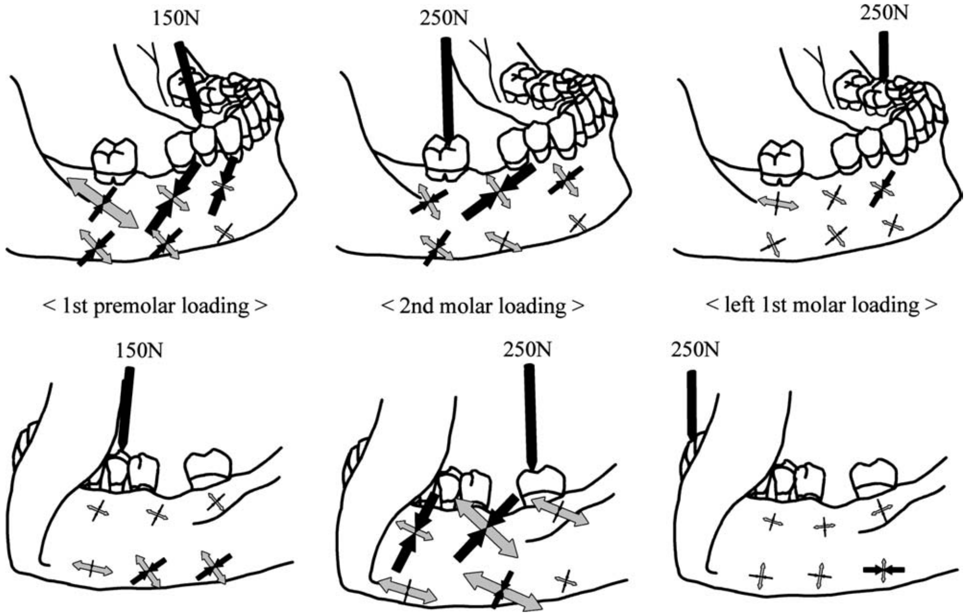

Fig. 2. Averaged principal strain distribution before FPD therapy. *Gray arrows* indicate tensile strain; *black arrows* indicate compressive strain. **Top row,** Buccal view. **Bottom row,** Lingual view. **Left,** 150-N load placed on buccal cusp of right first premolar. Greater strain in buccal cortex than in lingual. There was no significant difference in strain distribution between loads on right first and second premolars, irrespective of presence or absence of FPD. **Center,** 250-N load placed on central fossa of right second molar. Direction of tensile strain on buccal cortex was almost at right angles to that on lingual cortex indicating torsion. **Right,** 250-N load applied to buccal cusp of left first molar. Direction of tensile strain on buccal cortex was almost the same as that on lingual cortex. Likewise, direction of compressive strain was similar between buccal and lingual cortices, indicating dorsoventral bending. Similar strain distribution was found when left first molar was loaded after FPD therapy.

nonparametric Friedman ANOVA tests. There was also no significant difference in the angle of the principal strains. The grand mean of the coefficient of variation was 6.2%, which means that the overall error of the method was less than ±6.2%. Therefore, an overall accuracy of in vitro strain measurements in this study was at the level of 93.8%. There was also no significant difference in orientation of principal strains. For example, between 2 tests with loads of 150 N placed on the right central incisor, the differences in angles ranged from −5.6 to 7.7 degrees, and these differences were not statistically significant (Hotelling paired-sample test). Theoretically, the variance of the surface bone strain would be fully attributed to the error of method, since no biological variance was present, given the presumption that the mandible as a whole behaves identically under identical loading and boundary conditions.

RESULTS

Strain patterns before FPD therapy

When 150 N was applied to the buccal cusp of the right first premolar, there were significant differences in the magnitude of strain among mandibles (grand mean strain: mandible I, 125 ± 12 $\mu\epsilon$; mandible II, 126 ± 13 $\mu\epsilon$; mandible III, 279 ± 36 $\mu\epsilon$; mandible IV, 226 ± 22 $\mu\epsilon$; $P<.001$) and among sites (range of mean strain: from 70 $\mu\epsilon$ (gauge site 2) to 333 $\mu\epsilon$ (gauge site 11, $P<.001$). Larger strains were measured on the buccal than on the lingual cortex (mean strain: 246 ± 21 $\mu\epsilon$ versus 132 ± 12 $\mu\epsilon$, $P<.001$) (Fig. 2, *left*). There were significant differences in the magnitude of strain between sites on the buccal and lingual sides alike. For the buccal side of the alveolar bone, the tensile strain increased posteriorly (mean tensile strain: from 61 $\mu\epsilon$ to 416 $\mu\epsilon$) and the mode (ratio of ϵ_1/ϵ_2) increased from 0.19 to 1.83, while both the tensile and compressive strains showed the trend of increasing on the mandibular body (Fig. 2, *left*). This pattern was not clear on the lingual surface. There were also significant differences between the alveolar region and the basal portion of the mandible. On the lingual surface, the alveolar site had significantly lower strain than the site below it (mean strain: 98 ± 12 $\mu\epsilon$ versus 167 ± 18 $\mu\epsilon$; paired-sample *t* test, $P<.001$). On the buccal side, the situation was reversed (mean strain: 280 ± 35 $\mu\epsilon$ versus 212 ± 22 $\mu\epsilon$; paired-sample *t* test, $P=.001$).

There were no differences in strain patterns among the mandibles in terms of the orientations of the principal strains. Thirteen of 16 gauge sites had significant uniformity of the directions of the principal strains in all 4 mandibles (Rayleigh test of uniformity, $P=.04$); 3 other sites had a dominant orientation in at least 2 or 3 mandibles. The orientation of the principal strains was different between lingual and buccal sides. This is indicated by the posterosuperior direction of tensile strain on the buccal cortex compared to the anterosuperior direction of tensile strain on the lingual cortex (Fig. 2). Those differences in strain magnitude between mandibles and sites, and the uniformities in orientation of principal strains observed when the load of 150 N was placed on the buccal cusp of the first premolar were generally seen in all other loading experiments, irrespective of the presence or absence of an FPD.

The strain patterns with a load of 150 N on the buccal cusp of the right second premolar were almost the same as those with loading of the first premolar in terms of the directions of principle strains (Hotelling test, $P<.05$). There were no significant differences in magnitudes of shear strains. However, mean shear strains when loading on the second premolar showed a general trend in having lower value than those when loading on the first premolar (grand mean of shear strain: 330 ± 27 $\mu\epsilon$ versus 378 ± 31 $\mu\epsilon$; paired-sample *t* test, $P=.097$).

When a load of 250 N was placed on the central fossa of the right second molar, relatively larger strains were measured on the lingual cortices than on the buccal cortices (mean strain: 281 ± 15 $\mu\epsilon$ versus 245 ± 14 $\mu\epsilon$, respectively, $P=.19$) (Fig. 2, *middle*). Greater strain was also measured on the buccal alveolar bone than on the buccal mandible body (mean strain: 307 ± 16 $\mu\epsilon$ versus 183 ± 34 $\mu\epsilon$, $P<.001$), whereas on the lingual cortex, strain values were similar in both regions (mean strain: 309 ± 30 $\mu\epsilon$ versus 253 ± 27 $\mu\epsilon$, $P=.118$).

When the buccal cusp of the left first molar was loaded, relatively larger strains were detected on the right side of 2 mandibles, whereas small strains were measured for the other 2 mandibles. Strain patterns of the lingual cortex showed that tensile strains were dominant in the anteroposterior direction near the border of the mandibles, whereas compressive strain was dominant in alveolar bone (Fig. 2, *right*). A nearly identical strain pattern was found when loading was on the central fossa of the left first molar. The strain patterns when loaded on the buccal cusp or central fossa of the left second molar were similar to these when loaded on the left first molar as well. When the left first premolars were loaded, slightly larger strains were measured than those when loading was on the left molars. However, the directions of principal strain were comparable to those when a load was placed on the left molars. On the whole, regardless of loading position, the mandibles deformed in the same manner when the left posterior teeth were loaded, which was characterized with small strain.

Strain patterns after the FPD therapy and comparison with and without the FPD

No differences were detected in strain patterns before and after the FPD therapies when loading was on the right first premolar. There were no significant differences in the magnitude of shear strain (mean shear strain 365 ± 31 $\mu\epsilon$ versus 378 ± 31 $\mu\epsilon$). The angles of the principal strains were nearly the same. Sixty-two of 64 sites had angle differences of less than 10 degrees, and the differences in mode were insignificant as well (grand mean: 1.6 ± 0.2 versus 2.3 ± 0.9).

After the FPD therapies, there were no significant changes in strain patterns when a load of 150 N was placed on the anterior retainer (second premolar). There were no significant changes in the magnitude of shear strain (316 ± 26 $\mu\epsilon$ versus 330 ± 27 $\mu\epsilon$). The angles of the principal strains were similar at most of the sites. The angle differences in 53 of the 64 gauge sites in 4 mandibles was between −5 and 6 degrees, whereas a difference greater than 10 degrees was measured at only 11 gauge sites for 2 mandibles, and all of them were located on the lingual surface. The difference in mode was not significant (grand mean: 1.1 ± 0.2 versus 1.1 ± 0.1).

When the load was placed on the posterior retainer (second molar), differences were detected before and after FPD therapy. After FPD placement the magnitude of strain significantly decreased compared to that before FPD placement (mean grand shear strain: 344 ± 21 $\mu\epsilon$ versus 525 ± 33 $\mu\epsilon$; paired-sample *t* test, $P=.001$). However, there were area variations in the change in the magnitude of strains. A tendency was observed on the buccal cortices after FPD placement such that the absolute shear strains of the posterior 4 strain gauges increased by an average of 32 $\mu\epsilon$ or 4.3% from before FPD placement, and those of the anterior 2 gauges on the alveolar bone decreased by 166 $\mu\epsilon$ or 13.6% from before FPD placement (Fig. 3, *left*). On the lingual surface, relatively lower strain was observed on the alveolar bone compared to that before FPD placement (mean strain: 283 $\mu\epsilon$ versus 304 $\mu\epsilon$). No obvious change was observed in the mean strain of the mandibular corpus before and after therapy (272 $\mu\epsilon$ versus 268 $\mu\epsilon$). On the facial surface, both the alveolar region and the corpus had significantly lower strains compared to those before FPD placement (alveolar: 133 $\mu\epsilon$ versus 199 $\mu\epsilon$; corpus: 164 $\mu\epsilon$ versus 299 $\mu\epsilon$; $P=.02$ and .01, respectively). The directions of the principal strains were almost the same before and after FPD therapy. The angle difference in 47 of the 64 gauge sites in 4 mandibles was between −4 and 4 degrees; the difference of greater than 10 degrees was only measured at 5 gauge sites on 2 mandibles, and all of them were located on the lingual side.

When loaded on the central fossa of the pontic with a force of 250 N, strain patterns were similar to those when loaded on the posterior retainer. The magnitudes of strain were not significantly smaller compared to those when loaded on the posterior retainer (mean strain: 239 ± 20 $\mu\epsilon$ versus 263 ± 18 $\mu\epsilon$). The directions of the principal strains also did not differ (10.8 ± 2.3 degrees) (Fig. 3, *right*). There were no significant

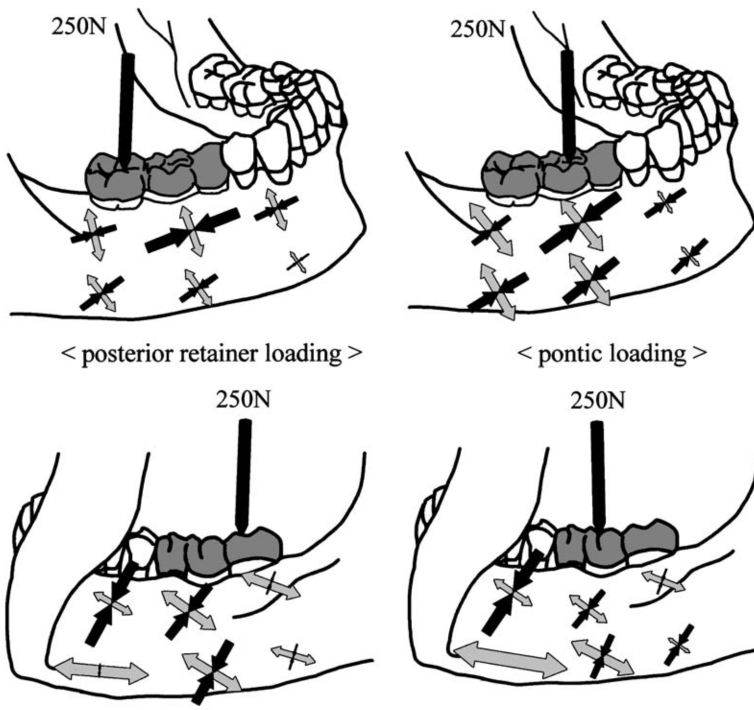

Fig. 3. Average principal strain distribution after FPD therapy. *Gray arrows* indicate tensile strain; *black arrows* indicate compressive strain. **Top row,** Buccal view. **Bottom row,** Lingual view. **Left,** 250-N load on posterior retainer. Larger strains were noted on buccal alveolar bone posteriorly. **Right,** 250-N load on pontic. Strain levels on both buccal and lingual cortices were similar to those when loaded on posterior retainer.

differences in strain patterns when loaded on the left posterior teeth with and without the FPD.

DISCUSSION

Physiological deformation of the mandible occurs during mastication.[5,6] Such deformation occurs as a result of 3 factors: the combination of external forces through the teeth, the contraction of masticatory muscles, and the reaction force around the temporomandibular joint. The teeth are thought to be an unstable factor because they can be affected by trauma, dental diseases, and subsequent dental treatments. One example is the loss of the dentition. The biomechanical condition of edentulous mandibles is different from that of dentate mandibles. Thus, the biomechanical environment in the oral cavity would not be uniform in the long term. Alterations of the biomechanical environment would result in changes in the deformation pattern of the mandible. Because bone deformation is considered to be an essential element for maintaining bone modeling and remodeling activity,[13] alterations in deformation patterns might influence bone mechanical properties over the long term. A study reported that differences exist in mechanical properties, such as elastic modulus and anisotropy, between dentate and edentulous mandibles.[16]

A conventional 3-unit FPD is a reliable treatment modality for a single missing tooth. The FPD restores the lost anatomical crown, thereby restoring masticatory efficiency and preventing tooth movement, such as tilting and extrusion of an opposing tooth. In a 3-unit FPD, because 2 independent teeth are rigidly connected to each other and 2 sets of roots support 3 anatomical crowns, it is reasonable to believe that deformation patterns of the mandible during function would be different from that without the FPD. Clinically, SOH is occasionally found in association with a 3-unit FPD. Although the precise etiology is unknown, the biomechanical effect of an FPD is postulated to be an etiologic factor.[21] For these reasons, the biomechanical effect of FPD therapy in terms of mandible deformation was investigated.

The shape of the mandible is not simple, and the inner architecture is complicated due to variations in cortical thickness, microstructure, and the associated material properties.[27,28] Such variations in internal architecture affect deformation patterns and make the deformation pattern of the mandibles difficult to predict.[29] In such situations, multiple rosette strain gauges may be used to assess the direction and magnitude of principal strains. To design a simulation model of mandible deformation in the present experiment, the area of the mandible to which medial pterygoid, masseter, and temporalis muscles attach was first covered by VPS, and then stabilized. This partial fixation of rami was considered to be enough to allow the mandible to deform without restriction during loading because dimensional change in posterior teeth in function has been reported to be approximately 500 μm.[22,23]

The results show that before the FPD therapy, torsion, resulting in inversion of alveolus and teeth and eversion of the inferior border, is the predominant pattern of deformation on the working side. This is indicated by the posterosuperior direction of tensile strain on the buccal cortex compared to the anterosuperior direction of tensile strain on the lingual cortex. Interestingly, this pattern of loading is found despite the lack of simulated muscle forces. In this experiment, the ramus was fixed, while the occlusal force was set parallel to the tooth axis. Thus, the torsion is inherent to the shape of the mandible under these loading conditions. This finding is consistent with that of Daegling and Hylander,[30] who performed an in vitro study to analyze torsion of the human mandible, and Korioth et al,[7] who performed finite element method simulation of a human mandible.

When strain patterns were compared before and after FPD therapy, it is clear that the FPD placement did not affect the strain patterns in the mandible when loaded on the teeth not involved in the FPD, including the left posterior teeth. However, the FPD therapy influences the strain magnitude on the alveolar bone when loaded on a posterior retainer. This is because, as the result of connecting 2 teeth rigidly, loading was distributed and transmitted to both retainers. The effect of connecting the teeth seems to be limited locally, because the direction of the principal strains was the same before and after FPD therapy. Accordingly, it can be argued that FPD therapy does not alter the overall deformation pattern of the mandible during loading. In the present study, information about alveolar bone deformation around retainers was not obtained because strain gauges were not attached to the region near the retainers. It is likely that FPD placement alters stress-strain distribution of peridental structures. Further research is needed to elucidate the possible biomechanical effect of FPD on local peridental microstructures.

Interestingly, the strain patterns when the pontic was loaded were similar to those when the posterior retainer was loaded. Normally, FPD therapy is recommended for more efficient mastication and to prevent collapse in occlusion from occurring. However, these data suggest that FPD therapy also contributes to normal bone physiology by providing similar mechanical stimuli for modeling and remodeling activity in the mandible. One of the advantages of a dental implant is that an implant transfers biomechanical stimuli directly to the peri-implant structure, thereby preventing bone resorption due to disuse. Since this study focused on the biomechanical effects of a 3-unit FPD on the mandible, a comparison cannot be made between implant and 3-unit FPD therapy in terms of their biomechanical influence. Nonetheless, the results of this study suggest that a 3-unit FPD may have similar positive biomechanical effects as an implant. Therefore, it is recommended that FPD or implant therapy be performed soon after tooth extraction to preserve the residual alveolar bone.

CONCLUSIONS

The biomechanical effects of 3-unit FPD therapy on the deformation patterns of human cadaver mandibles was assessed with in vitro strain gauge experiments and artificial loads. Within the limitations of this study, the following conclusions were drawn:

1. Three-unit FPD therapy does not alter the overall deformation patterns of the mandible during simulated function.
2. The working side of the mandible is subjected to torsion during loading.
3. The pontic contributes to physiological bone deformation during loading, as well as its conventional role in maintaining masticatory efficacy and occlusal stability.

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