A comparison of *in vitro* strain patterns of the mandible during loading before and after fixed partial denture therapy

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Abstract

This study determined whether the conventional mandibular posterior 3-unit fixed partial denture (FPD) influences the overall strain patterns of the mandible during artificial loading. Four unembalmed human cadaveric mandibles were used. The mandibles were missing 1st molars. Firstly, artificial loading up to 250N was applied to each tooth and strain measurements of the mandibles were performed with multiple strain gauge technique. Following FPD therapy replacing the 1st molar, strain measurements were carried out in the same manner to assess differences in strain patterns before and after therapy. No difference was detected before and after the FPD placement when loading was applied onto the teeth not involved in the FPD. Slight difference was found in strain gradient before and after the FPD therapy when the posterior retainer was loaded. Increased strain levels were detected posteriorly and decreased strain anteriorly on buccal cortex. Overall stain pattern of the mandible, however, was similar to that before the FPD therapy. Strain distribution when the pontic was loaded was similar to that when the posterior retainer was loaded.

Within the limitations of this study, the following conclusions were drawn.

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

Key words: fixed partial dentures, strain, mandible

INTRODUCTION

In the history of modern dentistry mandibular posterior 3-unit fixed partial denture (FPD) therapy serves as a predictable prosthetic treatment for one missing molar. Overall treatment outcome is usually outstanding as long as basic clinical procedures are followed.[1,2] The biomechanical effects of FPD therapy, however, still remain unknown because of the difficulties to assess the *in vivo* deformation patterns of the mandible during function. It is known that the mandible deforms during function.[3),4)] This deformation creates functional stress in the mandible and such stress contributes to bone development and maintenance.[5),6)] Since 3-unit FPD therapy is a rigid connection of independent teeth, FPD placement will alter the stress distribution of the mandible during chewing as well as the bony structure around the abutment teeth. The alteration in functional stress distribution would affect the remodeling process of the mandible, and could change the superstructure of the mandible after a long term service period. Evidence to support this theory is the occurrence of subpontic osseous hyperplasia (SOH). The SOH, known as osseous proliferation beneath the pontic of the FPD, is occasionally seen in routine clinical patients and usually is found in the posterior region of the mandible.[7),8)] Although etiology of the SOH is not clearly elucidated, the mechanical stimuli transmitted through the FPD during chewing is the most likely candidate to induce SOH.[9)] Thus, it could be possible that posterior 3-unit FPD modifies the ultra- or superstructure of the mandible even when no apparent change in shape

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is clinically observed.

In this study, we tested the hypothesis that the 3-unit FPD therapy would influence the strain patterns of the mandible during function. Surface strain on the human unembalmed cadaveric mandibles, when artificially loaded, was measured before and after the FPD therapy by using the multiple strain gauge technique and assessed if there was a difference.

MATERIAL AND METHODS

The research protocol used in this study was reviewed and approved by the Institutional Review Board, Baylor College of Dentistry, Texas A&M Health Science Center, Dallas, TX, prior to the initiation of the experiments.

First of all, strain patterns were measured for the unembalmed cadaveric mandibles missing 1st molars during artificial loadings. Next, FPD therapy was performed on the same mandible and strain patterns during loading were again recorded. Finally, the difference in strain patterns of the individual mandible was compared with and without the FPD.

Four adult human cadaveric mandibles were used. Their causes of death were not bone related diseases. The mandibles were missing first molars and other teeth were either intact or restored. Before use, all soft tissues were removed and the surfaces were prepared with fine abrasive papers and acetone. Sixteen rosette strain gauges (FRA-1-11-11, Tokyo Sokki Kenkyujo, Tokyo, Japan) were attached with cyanoacrylate cement onto the right side of the mandible: eight on the lingual aspect and eight on the buccal surface (Fig. 1).

On both aspects, the upper four gauges were located around the boundary between alveolar bone and mandibular body; the lower four were attached about 10 mm above the lower border of the mandible. The anteroposterial positions of the four gauges in each row were below the canine, second premolar, second molar, and retromolar region, respectively. The strain gauges were connected to a series of strain amplifiers (Model 2120, Micro Measurements, Raleigh, NC, USA) through a switch box. The mandibles were bilaterally semifixed at the condyles and angles so that they could deform with minimum restriction during loading. To do this, first, the condyles and angles were covered with heavy body polyvinylsiloxane impression material (Reprosil, Dentsply, Milford, DE, USA). The rubber layers were approximately 1.0 mm in thickness. The covered portions of the mandible were embedded into the block made of dental plaster.

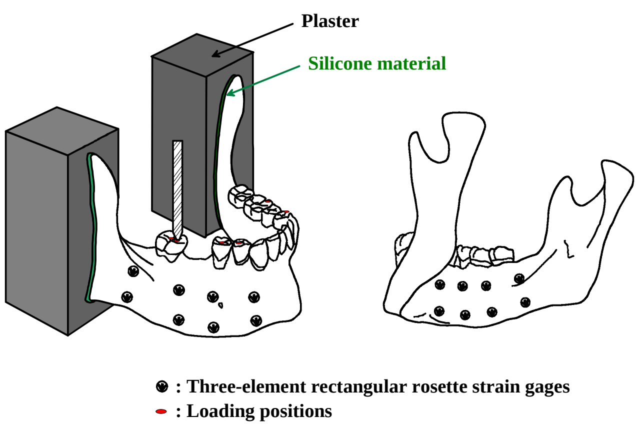

Fig. 1 Strain gauge sites on the mandible. The rami of the mandible were covered with silicone rubber and semifixed in a plaster.

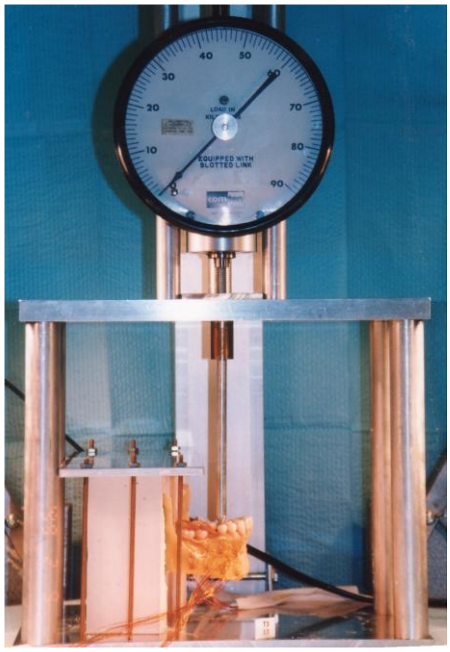

Fig. 2 Loading apparatus. The plaster holding the mandible was secured in a loading apparatus. Static loading was applied to each tooth individually.

The block supporting the mandible was then secured in the loading apparatus with screws (Fig. 2).

Static loadings were applied to the tooth individually; the loading positions were the buccal cusps of the right premolars, the mesiobuccal cusp and the central fossa of the right 2nd molar, and the mesiobuccal cusp of either left 1st or 2nd molar. Magnitude used was 150N for the premolars, 250N for the molars. During loading, strain measurements were performed. Strain data were directly stored in a personal computer via an analog-digital converting board with the sampling rate of 62.5Hz. Afterwards, maximum and minimum principal strains and their directions were calculated according to the standard formula described elsewhere.[10]

After the series of strain measurements with the mandible missing 1st molars were completed, the mandibles were displaced from the loading apparatus and were received 3-unit FPD therapies replacing the right first molar. The standard clinical and laboratory procedures were followed. Retainers were 2nd premolar and 2nd molar. Tooth preparations were done with chamfer. The FPDs were cast in gold alloy and cemented with glass ionomer cement (Vitremer, 3M, St. Paul, MN, USA). Since great care was taken to protect the strain gauges during the FPD therapy, they were not influenced by the procedures at all. The mandibles with the FPDs were then positioned again in the loading apparatus and the strain measurements were carried out in the completely same manner as those for the mandibles without the FPDs. In addition, loadings of 150N and 250N were placed onto the buccal cusps and the central fossa of the pontic, and strain patterns were determined. Since the strain gauges and their position were identical between the experiments with and without the FPD, in the end, differences in strain patterns were assessed before and after the FPD therapies.

RESULTS

There was no considerable difference in the strain patterns of the mandible among the four mandibles when loaded. In the result section, therefore, strain values were averaged and reported unless otherwise described. The mandible showing strain patterns in Figures 3 and 4 serves to represent other mandibles.

Strain patterns of the mandible without the FPD

When loading of 150N was applied onto the buccal cusp of the 1st premolar, larger strain values were measured on buccal than those on the lingual cortex (mean strain: 246$\mu\varepsilon$ vs. 132$\mu\varepsilon$). No major difference was found in strain patterns among the mandibles: tensile strains in posterosuperior direction and compressive strains at right angle to them on buccal cortex; small tensile strains in anterosuperior direction and small compressive strains at right angle on lingual cortex (Fig. 3A). The strain pattern when loading was placed on the buccal cusp of the 2nd premolar was almost the same as that with loading on the 1st premolar. When loading of 250N was placed onto the 2nd molar, both buccal and lingual cortices strained to the similar degree. Larger strain values were detected on all mandibles when loaded on central fossa compared to those when loaded on the mesiobuccal cusp (mean strain: 265$\mu\varepsilon$ vs. 238$\mu\varepsilon$). The strain pattern when loaded onto central fossa was shown in Fig. 3B. Greater strain was measured on buccal alveolar bone than on buccal mandible body, while on lingual cortex strain values were similar between them except for the area below the 2nd molar. When the left molar was loaded, considerable amount of strain was detected on the right side of the mandible in two cases; small strain was measured in other two mandibles. Strain patterns of lingual cortex showed compressive strain dominant in anteroposterior direction near the border of the mandibles, while tensile strain dominant in alveolar bone (Fig. 3C).

Strain patterns of the mandible with the FPD

There was no difference in strain patterns when loaded on the 1st premolar before and after the FPD therapies. When loaded on the anterior retainers, insignificant differences in strain patterns were observed before and after the FPD placement in all mandibles. When loading was placed on the posterior retainer, slight differences were detected before and after the FPD therapy. Tendency was observed on buccal cortex after the FPD insertion that absolute strain values of posterior strain gauges increased and those of anterior gauges on alveolar bone decreased. However, the direction of principal strain was almost the same before and after the FPD therapy (Fig. 4A). On lingual cortex, not observable difference

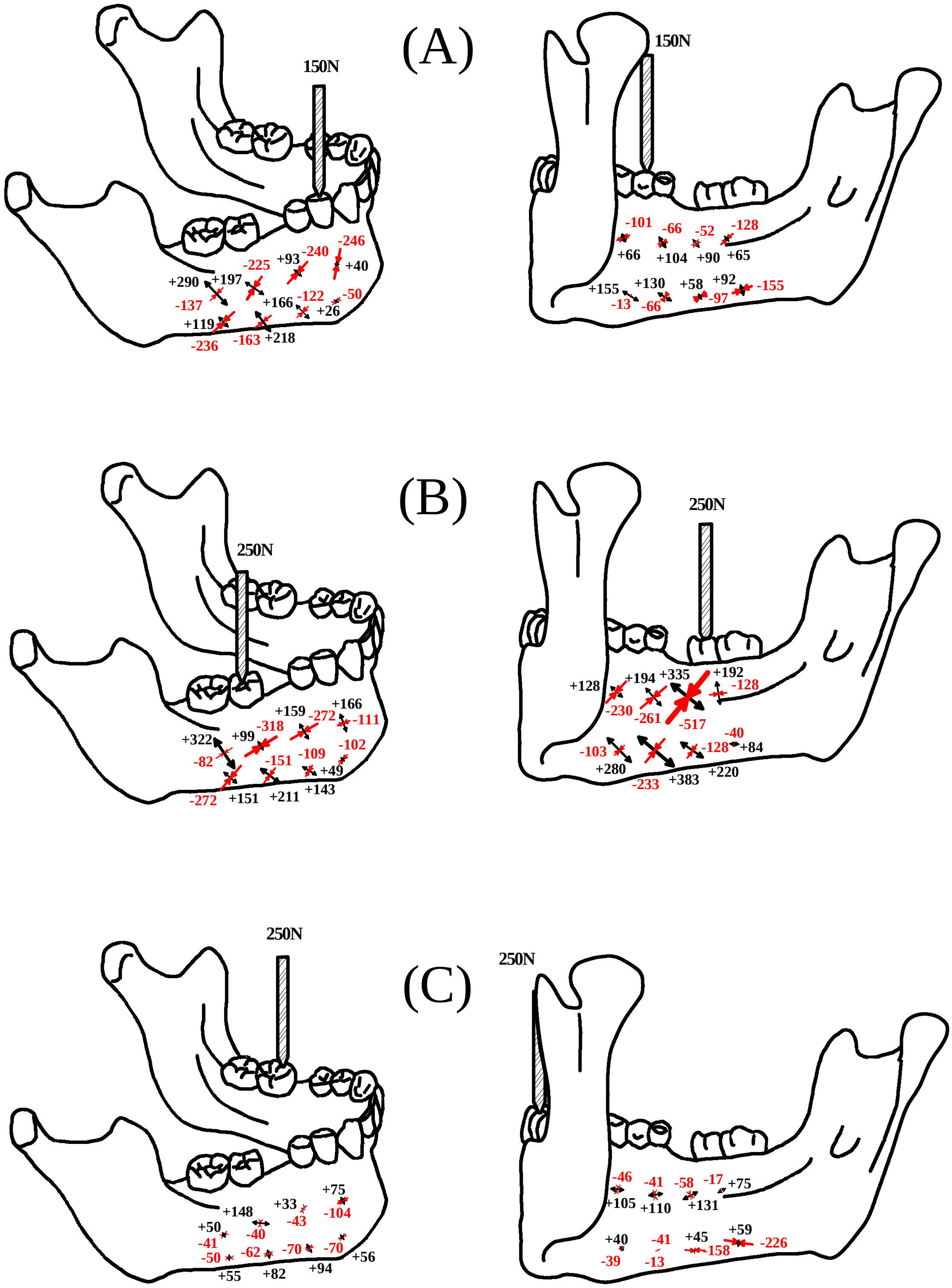

Fig. 3 Strain distribution on the mandible when loaded before FPD therapy. Positive value denotes tensile strain. Negative value denotes compressive strain. (unit: x10^{-6} ε). (A): Loading of 150N was applied onto the buccal cusp of 1st premolar. Greater strain was noted on buccal than lingual side. (B): Loading of 250N was applied onto the central fossa of the 2nd molar. Direction of tensile strain on the buccal cortex was almost at right angle to that on the lingual cortex. Likewise, orientation of compressive strain was approximately at right angle to that on the lingual cortex. These strain distribution patterns indicate torsion of the mandible. (C): Loading of 250 N was applied to the buccal cusp of left 2nd molar (Balancing side). Relatively small strain level was measured on both buccal and lingual cortex.

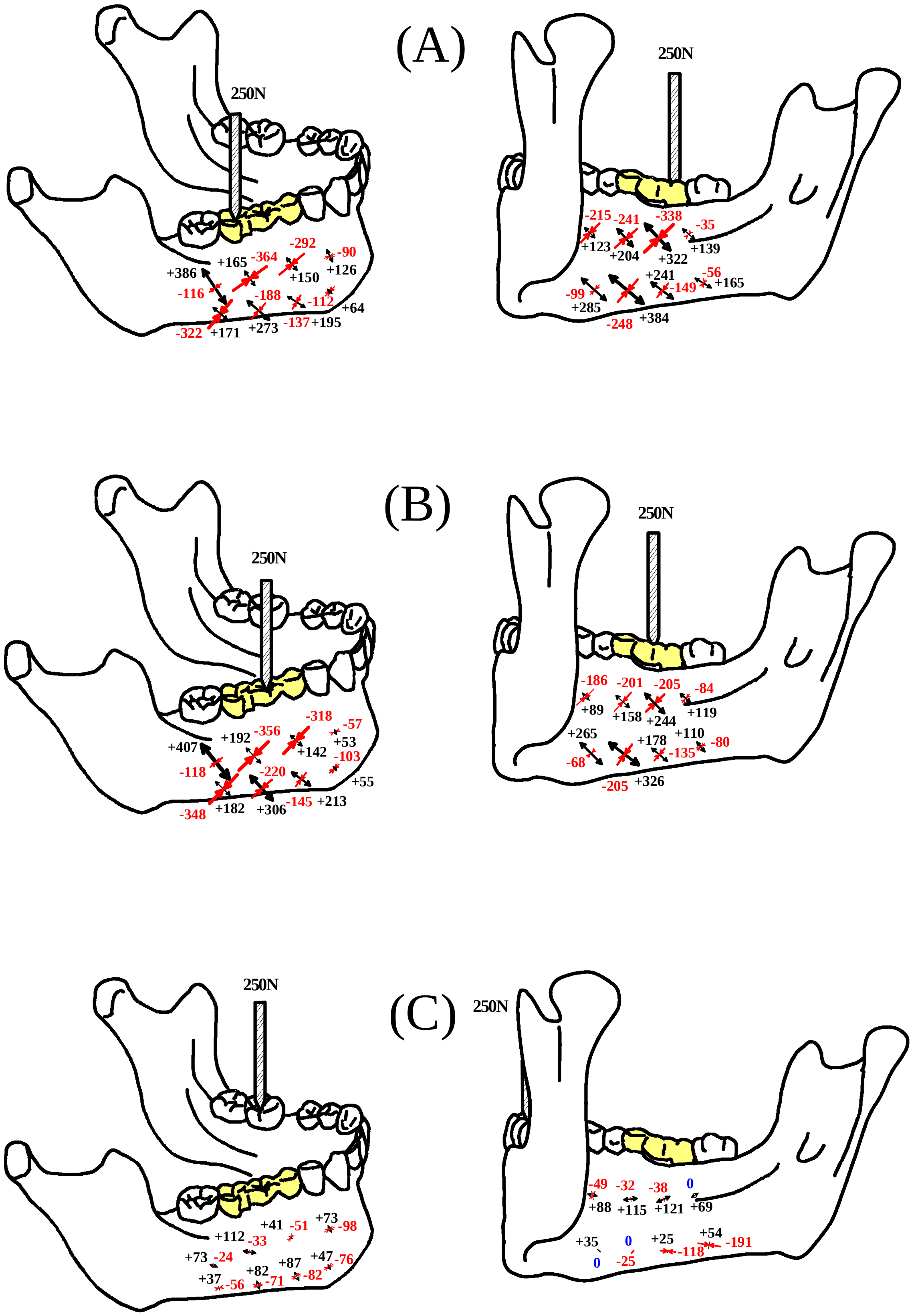

Fig. 4 Strain distribution on the mandible when loaded after FPD therapy. Positive value denotes tensile strain. Negative value denotes compressive strain. (unit: x10^{-6} ε). (A): Loading of 250N was applied to the posterior retainer. Large strain was noted on buccal alveolar bone posteriorly. (B): Loading of 250N was placed to the pontic. (C): Loading of 250N was applied to the buccal cusp of left 2nd molar (Balancing side). Strain distribution was almost identical to that of the mandible before FPD therapy (Fig. 3C)

was noted before and after the FPD therapy except for one mandible. In one case, strain near the posterior retainer decreased significantly and strain of the anterior gauge near the mandible border increased greatly. When loaded on the pontic, strain patterns were similar to those when loaded on the posterior retainer in direction. However, more evenly distributed strain pattern was observed compared to that when loaded on the posterior retainer (Fig. 4B). There was no difference in strain patterns when loaded on the left molar with and without the FPD (Fig. 4C).

DISCUSSION

It has been reported that physiological deformation of the mandible occurs during mastication.[3),4)] Such deformation takes place as a result of the combination of external force through teeth, contraction of masticatory muscles, and reaction force around temporomandibular joint. Among these three factors, teeth are thought to be an unstable factor because they can be affected by trauma, dental diseases, and subsequent dental treatments. One example is being edentulous. Biomechanical condition of edentulous mandibles is different from that of dentate mandibles. Thus, biomechanical environment in the oral cavity would not be uniform for a long-term. Alterations of biomechanical environment would lead to change in the deformation pattern of the mandible in some degree. Because bone deformation is considered to be an essential element for maintaining bone modeling and remodeling activity, alteration in deformation pattern would influence on bone mechanical properties in the long run. A recent study reported that differences exist in mechanical properties, such as Young's modulus and anisotropy, between dentate and edentulous mandibles.[11)]

In modern dentistry, a conventional 3-unit FPD therapy is regarded as a reliable treatment modality for one missing tooth. The FPD therapy is to restore a lost anatomical crown, thereby recovering masticatory efficiency and preventing tooth movements, such as tilting and extrusion of an opposing tooth. In a 3-unit FPD, because two independent teeth are rigidly connected each other and two roots support three anatomical crowns, it is reasonable to think that deformation pattern of the mandible during function would be different from that without an FPD. In clinic, SOH is occasionally found in association with a 3-unit FPD. Although precise etiology is unknown, biomechanical effect of an FPD is postulated to be an etiologic factor.[9)] For these reasons, in this study, biomechanical effect of FPD therapy was investigated in terms of mandible deformation.

The shape of the mandible is not simple and inner architecture is complicated due to variations in cortical thickness and material properties.[12)] Such variations in inner architecture affect deformation pattern and make it difficult to predict. In such situation, multiple rosette strain gauges should be used to assess the direction and magnitude of principal strain. One limitation of strain gauge technique is that only surface strain can be measured. However, because human cadaveric mandibles were used in this study, measured strain patterns certainly reflect the accurate deformation of the mandible, and thus, give us the correct overall picture of deformation. Besides, it is not the objective of this study to analyze deformation of the inner architecture in detail but compare the strain pattern before and after the FPD therapy. To simulate biting, in the present experiment, the area of the mandible to which medial pterygoid, masseter, and temporalis muscles attach was semifixed with silicone of 1mm in thickness. This semi-fixation of rami was considered to be enough to allow the mandible deform without restriction during loading because dimensional change in posterior teeth in function has been reported about 500μm.[13)]

The results of strain patterns of the mandible before the FPD therapy pointed out that torsion is predominant deformation pattern in working side. When loaded, strain pattern of working side showed tensile strain in posterosuperior direction on buccal cortex. On lingual cortex of working side, compressive strain was found in posterosuperior direction. That is, quality of strain was opposite (tension vs. compression) in an identical direction between buccal and lingual cotices. This strain pattern indicates that working side of the mandible was subjected to torsional force: inversion of alveolus and teeth. This finding is consistent with that of Daegling and Hylander, where *in vitro* study was performed to analyze torsion of the human mandible,[14] and Korioth et al. where FEM simulation of human mandible was performed.[15] When strain patterns are compared before and after the FPD therapies, it is clear that the FPD placement does not affect the strain patterns on the mandible when loaded on the teeth not involved in the FPD. However, the FPD therapy had influence on strain magnitude on the alveolar bone when loaded on a posterior retainer. This is because, as the result of connecting two teeth firmly, loading was distributed and transmitted to both retainers. However, the effect of connecting two teeth seemed to be limited locally because the direction of principal strain was the same before and after the FPD therapy. Accordingly, it can be concluded that the 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 the strain gauges were attached away from the retainers. It could be possible that the FPD placement alters stress-strain distribution of peridental structure. Another research design is needed to elucidate the possible biomechanical effect of the FPD upon local peridental micro structures. Interestingly, the strain patterns when the pontic was loaded are similar to those when loading was on the posterior retainer. Normally, the FPD therapy is recommended for more efficient mastication and to prevent collapse in occlusion from occurring. This data further suggests that FPD therapy also contribute to normal bone physiology by providing appropriate mechanical stimuli for modeling and remodeling activity in the mandible.

In the present study, no statistical method was utilized to assess the strain distribution of the mandible. Despite the extensive literature search, there was no appropriate statistical method described to analyze the deformation of an object. Taking the fact that an individual mandible has its own dimension and material properties, it is of importance to get a trend of alteration in strain patterns before and after the FPD therapy. One of such trend obtained from this study is that the direction of principal strain stays same before and after the therapy. This may implies that overall deformation pattern of the mandible during function is determined by the individual shape of the mandible, not by the teeth factor.

In conclusion, we reject the hypothesis that the 3-unit FPD therapy would influence the strain patterns of the mandible during function. We found that the FPD treatment would not alter the overall deformation patterns of the andible during function, and that working side of the mandible is subjected to torsion during loading. Furthermore, as the result of the FPD therapy, the pontic contributes to the physiological bone deformation during loading as well as its conventional role in maintaining masticatory efficacy and occlusal stability.

REFERENCES:

1. Creugers NH, Kayser AF, van 't Hof MA. A meta-analysis of durability data on conventional fixed bridges. Community Dent Oral Epidemiol; 22:448-452, 1994.
2. Scurria MS, Bader JD, Shugars DA. Meta-analysis of fixed partial denture survival: prostheses and abutments. J Prosthet Dent; 79:459-464, 1998.
3. Hylander WL. Stress and strain in the mandibular symphysis of primates: a test of competing hypotheses. Am J Phys Anthropol; 64: 1-46, 1984.
4. Hylander WL, Crompton AW. Jaw movements and patterns of mandibular bone strain during mastication in the monkey Macaca fascicularis. Arch Oral Biol;31:841-848, 1986.
5. Rubin CT, Lanyon LE. Kappa Delta Award paper. Osteoregulatory nature of mechanical stimuli: function as a determinant for adaptive remodeling in bone. J Orthop Res;5:300-310, 1987.
6. Duncan RL, Turner CH. Mechanotransduction and the functional response of bone to mechanical strain. Calcif Tissue Int; 57:344-358, 1995.
7. Lorenzana ER, Hallmon WW. Subpontic osseous hyperplasia: a case report. Quintessence Int; 31:57-61, 2000.
8. Daniels WC. Subpontic osseous hyperplasia: a five-patient report. J Prosthodont; 6:137-143, 1997.
9. Wasson DJ, Rapley JW, Cronin RJ. Subpontic

osseous hyperplasia: a literature review. J Prosthet Dent;66:638-641, 1991.
10. Yamashita J, Shiozawa I, Takakuda K. A comparison of in vivo and in vitro strain with posterior fixed partial dentures. J Prosthet Dent;77:250-255, 1997.
11. Schwartz-Dabney CL, Dechow PC. Edentulation alters material properties of cortical bone in the human mandible. J Dent Res;81:613-617, 2002.
12. Kingsmill VJ, Boyde A. Variation in the apparent density of human mandibular bone with age and dental status. J Anat;192 (Pt 2):233-244, 1998.
13. Chen DC, Lai YL, Chi LY, Lee SY. Contributing factors of mandibular deformation during mouth opening. J Dent;28:583-588, 2000.
14. Daegling DJ, Hylander WL. Biomechanics of torsion in the human mandible. Am J Phys Anthropol;105:73-87, 1998.
15. Korioth TW, Romilly DP, Hannam AG. Three-dimensional finite element stress analysis of the dentate human mandible. Am J Phys Anthropol;88:69-96, 1992.