

# The Influence of Controlled Occlusal Overload on Peri-implant Tissue. Part 3: A Histologic Study in Monkeys

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*The influence of experimental occlusal overload on peri-implantitis in monkeys (Macaca fascicularis) has been examined to explain the pathology of the disease that develops in the tissue around osseointegrated implants. In the first article of this series, it was reported that bone resorption was not observed around implants when occlusal trauma was produced by a superstructure that was in supraocclusal contact with an excess occlusal height of approximately 100  $\mu\text{m}$ , provided there was no inflammation in the peri-implant tissue. In the second part of the study, experimental inflammation was created in the peri-implant tissue, and occlusal overload was produced by a superstructure with an excess occlusal height of 100  $\mu\text{m}$ . Notable bone resorption was observed around the implant with the passage of time. These results suggested that, in addition to the control of inflammation in peri-implant tissue, traumatic occlusion may play a role in bone breakdown around the implant. In the present study, while the peri-implant tissue was kept in an inflammation-free state, bone level changes around the implants were investigated when various levels of traumatic force were exerted. The supraoccluding prostheses were defined as excessively high by 100  $\mu\text{m}$ , 180  $\mu\text{m}$ , and 250  $\mu\text{m}$ , respectively. The heights were determined with an image analysis device, and the bone responses around the implants induced by the traumatic forces were investigated. The results showed that bone resorption around implants tended to increase with 180  $\mu\text{m}$  or more excessive height of the superstructure. This suggests that the threshold of excessive height of the superstructures at which peri-implant tissue breakdown may start is approximately 180  $\mu\text{m}$ . It is also suggested that there is a possibility of bone resorption around the implants caused by excess occlusal trauma, even when there is no inflammation in peri-implant tissue. (INT J ORAL MAXILLOFAC IMPLANTS 2000;15:425-431)*

**Key words:** animal study, bone resorption, comparative histology, endosseous dental implantation

A variety of implant systems have been developed, and advances continue to be made in areas such as the material used for implant bodies, implant placement technique, and new abutments, as well as

in basic studies. However, similar numbers of implant failures continue to be reported, and the pathology seen is in many aspects similar to that of periodontitis occurring around natural teeth. Although much research has been conducted with regard to oral bacteria, inflammation, occlusion, and superstructure,<sup>1-4</sup> the onset of peri-implantitis has not yet been fully explained.

Currently, it is believed that the disease is not the result of a single cause but is a multifactorial disease, with potential etiologic factors interrelating with one another. It has also been reported that overloading an oral implant may result in loss of osseointegration.<sup>5-8</sup> In clinical or radiographic studies in the monkey to investigate the influence of overloading on implants, Isidor has demonstrated that occlusal overload can result in loss of osseointegration.<sup>9-11</sup>

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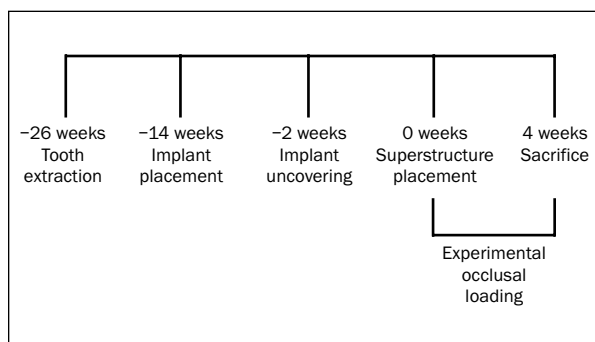
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**Fig 1** Timeline of the experiment.

In an initial study by the authors involving the monkey (*Macaca fascicularis*), plaque control was carried out around implants to keep the peri-implant tissue free of inflammation.<sup>12</sup>

To establish the same conditions in terms of excess occlusal height introduced by superstructures, an image analysis device was utilized to produce approximately 100  $\mu\text{m}$  of excess height. This excessively high superstructure was in place for 1 to 4 weeks so as to provide occlusal trauma, after which histopathologic examination was conducted. The results suggested that no bone resorption was caused by occlusal trauma around any implant. In a second study, experimental inflammation was induced using ligature wires after second-stage surgery, followed by occlusal trauma through the superstructure with an excess occlusal height of 100  $\mu\text{m}$ , as in the first study. As a result, as the duration of overloading caused by traumatic occlusion increased, notable bone resorption around the implants was observed. These results suggest that as a part of the development of peri-implantitis, bone breakdown around the implants was accelerated when traumatic occlusion was added to inflammation in the tissue around the implants.

In these previous studies, it was assumed that an excess height of 100  $\mu\text{m}$  would be the limit that could be withstood by the human periodontal ligament. However, there was no evidence of an exact amount of excessive height that could be withstood. In this study, based on the results of previous studies, occlusal trauma was produced over a range of excess occlusal heights in monkeys, while oral hygiene was maintained. Consequently, the purpose of the present study was to determine the excessive height limit before bone breakdown occurs around the implants, with a histopathologic examination conducted to investigate bone level changes around the implant.

## MATERIALS AND METHODS

Four monkeys (*Macaca fascicularis*), 5 to 6 years of age and 5.5 to 6.5 kg in weight (Japan Clea Co, Tokyo), were used. Each animal was raised in an experimental room held at a constant temperature and humidity ( $28 \pm 1^\circ\text{C}$ , 50% to 60%). The animals were given hard monkey food (Japan Clea Co) and sufficient tap water. Oral hygiene was conducted once a week under general anesthesia so that general and oral health would be maintained. The test site extended from the second premolar to the first molar in the right mandible. The implants used in the study were IMZ implants (provided by Friatec, Mannheim, Germany) designed for experimental investigation (2.8 mm diameter, 8 mm length).

The timeline for the experimental design is shown in Fig 1. For all procedures, intramuscular injections of 0.1 mL/kg atropine and 0.05 mL/kg ketamine hydrochloride (Sankyo, Tokyo, Japan) were given as preanesthetic medication. Fifteen minutes later, the animals were anesthetized with an intramuscular injection of 0.15 ketamine hydrochloride. Under general anesthesia, a local injection of 2% lidocaine with epinephrine was given in the surgical area, followed by extraction of the second premolar and the first molar in the right mandible. After a 3-month healing period, 2 experimental implants were placed in each monkey according to the standard protocol. After 3 months were allowed for the implants to be osseointegrated, the second surgery was conducted according to standard procedures. A 2-week healing period was allowed for the peri-implant tissue, after which an impression was made using impression abutments.

The superstructure was adjusted according to Miyata's method,<sup>13</sup> as in the previous studies, so that the excess occlusal height would be 100  $\mu\text{m}$ , 180  $\mu\text{m}$ , or 250  $\mu\text{m}$ , respectively, as measured with an image analysis device. According to the timeline, each monkey was subjected to occlusal trauma for 4 weeks. Clinical examinations of the peri-implant tissue or periodontal tissue around the implant and adjacent teeth were conducted twice, first at the time of prosthesis placement and again at the time traumatic occlusion was terminated (just before sacrifice). The clinical examination included probing pocket depth (PPD) or probing peri-implant sulcus depth (PPSD), bleeding on probing (BOP), and tooth or implant mobility (MO). Then, according to the guidelines of Meikai University for experimental animals, the animals were sacrificed, and after conventional perfusion fixation, non-demineralized ground sections were prepared. They were prepared with Col's hematoxylin-eosin staining, as in previous

**Table 1** Changes in Periodontal Probing Depth (mm) or Peri-implant Sulcus Depth (mm) Before and After Occlusal Loading

	Model A		Model B		Model C		Model D	
	Before	After	Before	After	Before	After	Before	After
Second premolar	1.0	1.1	1.0	1.0	1.0	1.2	1.0	1.0
First molar	1.1	1.3	1.0	1.2	1.0	1.1	1.2	1.2
Implant 1	1.0	1.0	1.1	1.1	1.0	2.0	1.1	2.5
Implant 2	1.0	1.0	1.0	1.0	1.0	1.4	1.1	3.2

Model A = control (no excess occlusal height); Model B = supraocclusion with excess height of 100  $\mu$ m; Model C = supraocclusion with excess height of 180  $\mu$ m; Model D = supraocclusion with excess height of 250  $\mu$ m.

studies. The images of sections that were input to an image analytical computer system were measured for the purpose of comparing the ratio of implant placement site width from buccal to lingual between human and experimental animals.

## RESULTS

### Clinical Examination

When the experimental period was completed, no inflammatory responses, such as redness or swelling, were observed in any animals. The results of the clinical examination are shown in Table 1. There were no changes in the control or 100- $\mu$ m excess height models. However, the 180- $\mu$ m and 250- $\mu$ m excess height models showed a tendency to develop greater depths on the PPSD examination compared with pre-occlusal loading conditions.

### Radiographic Examination

The radiographs were compared before loading and just before sacrifice. As for the control (Fig 2) and the 100- $\mu$ m model, no notable changes were observed (Fig 3). However, in the 180- $\mu$ m (Fig 4) and 250- $\mu$ m models (Fig 5) (especially in the 250- $\mu$ m excess height model), mesiodistal bone resorption was observed to almost half of the implant body.

### Histologic Examination

In the control model, no bone resorption, bone destruction, or epithelial downgrowth was observed around the implants. Most of the surface of each implant was well integrated with bone (Fig 6). In the model with 100  $\mu$ m of excess height, the implant was placed at the buccal side of the ridge crest, and as a result, the coronal one sixth of the implant body

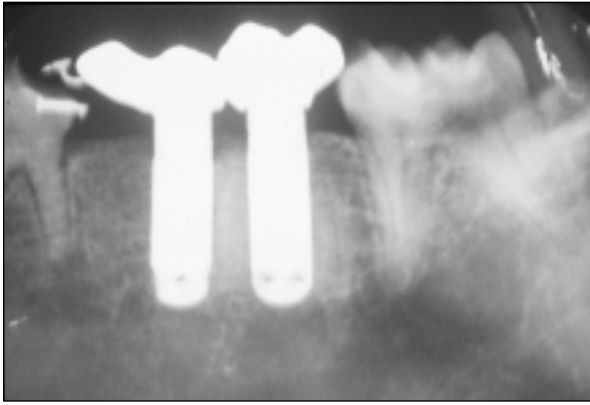
was exposed over the bone. Bone resorption and bone destruction around the implant was not found and well-established bone-to-implant contact was observed (Fig 7). In the model with 180  $\mu$ m of excess height, slight bone resorption from the buccal side of the alveolar bone to almost half of the implant was observed (Fig 8). In the final model (supraocclusion with 250  $\mu$ m of excess height), vertical bone resorption reaching the apex of the implant, as well as epithelial downgrowth, was found on both buccal and lingual aspects of the alveolar bone (Fig 9). Contact of the implant with bone was observed in only a small region of the apex and at the vent.

### Maximum Width of Placement Site from the Buccal to the Lingual Side

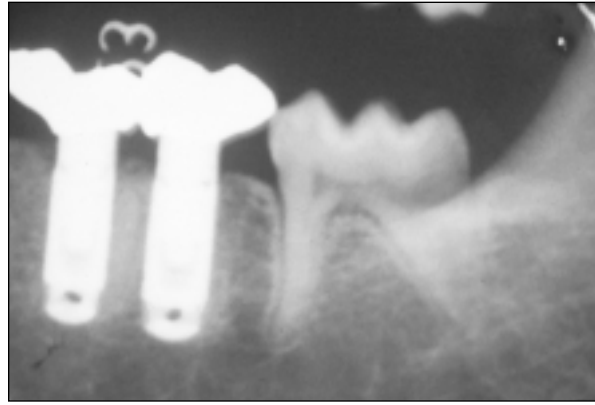
The results of this measurement are shown in Table 2. The average maximum width of the placement site was 7.55 mm. Also, the average implant share of maximum width of the placement site from the buccal to the lingual was 37.0%.

## DISCUSSION

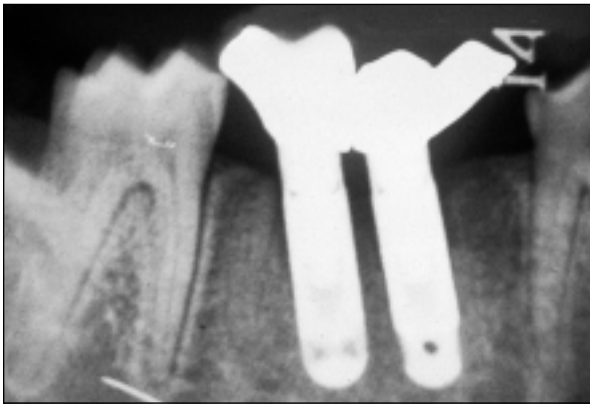
A number of studies have been conducted regarding peri-implantitis, and Adell<sup>14</sup> and Albrektsson et al<sup>15</sup> strongly suggested that occlusion could significantly affect the bone around implants. Also, various studies have been conducted on periodontitis involving natural teeth, bone remodeling around implants induced by occlusal forces,<sup>16</sup> and bone changes around implants in response to lateral forces.<sup>17</sup> Lindhe et al<sup>18</sup> reported that once inflammatory destruction begins in the peri-implant tissue, it progresses rapidly compared with that around natural teeth and is not likely to reach remission.



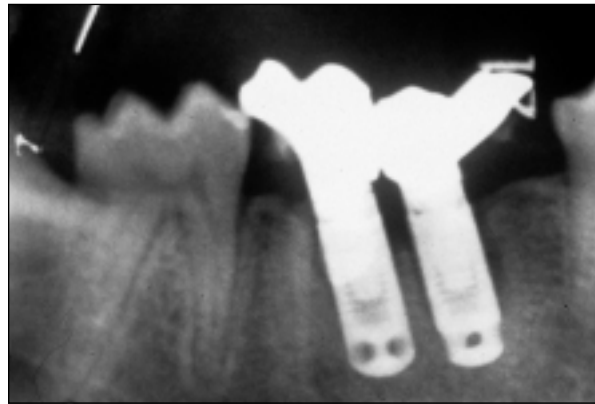
**Fig 2** Radiographic view of the control model taken just before sacrifice. No occlusal stress was added; no notable changes were observed.



**Fig 3** Radiographic view of model in which supraocclusion was produced by excess height of 100  $\mu\text{m}$ . Changes were not observed as resulted in the first article in this series.<sup>12</sup>



**Fig 4** Radiographic view of model with supraocclusion produced by excess height of 180  $\mu\text{m}$ . Bone resorption was seen at the mesiodistal of the marginal bone level on both implants.

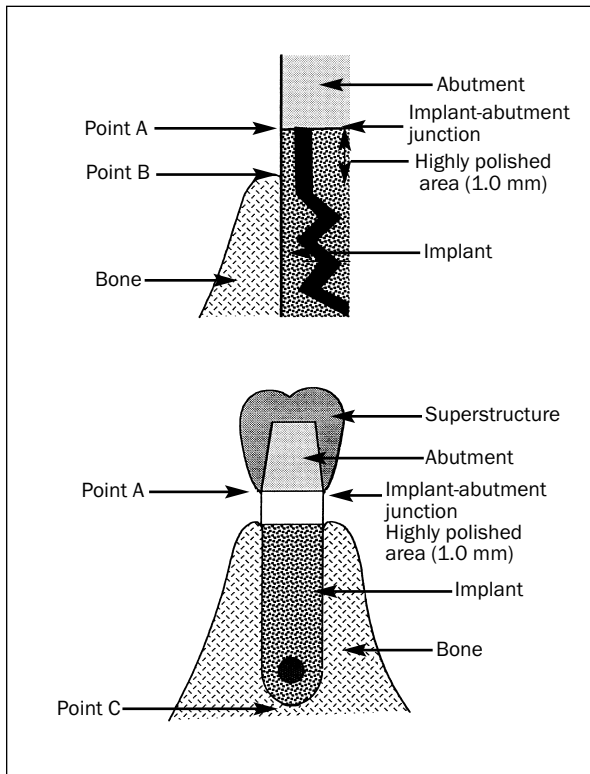


**Fig 5** Radiographic view of model with supraocclusion produced by excess height of 250  $\mu\text{m}$ . More bone resorption was seen than in the 180- $\mu\text{m}$  model at the mesiodistal of the marginal bone level and in the apical direction on both implants.

The relationship between periodontal pathogenic microorganisms around implants and inflammation in the tissue around the implants<sup>19</sup> has been confirmed. Consequently, peri-implantitis could result from infection caused by periodontal pathogenic microorganisms and traumatic force from occlusion. Experimental studies of implant overloading in monkeys have been reported. Ogiso et al<sup>20</sup> demonstrated that increasing the occlusal load of dense apatite implants placed in monkeys resulted in remodeling and thickening of the surrounding bone. Some reports have noted increased resistance to high occlusal load in the monkey models.<sup>21,22</sup>

In the present authors' first article, it was reported that, following traumatic occlusion produced by wearing a superstructure with an excess occlusal

height of 100  $\mu\text{m}$  for 4 weeks while oral hygiene was well maintained, no bone resorption or destruction was observed around the implants.<sup>12</sup> This suggested that if stress produced by occlusion remains within an acceptable range, it might not lead to tissue destruction around implants. In a second report,<sup>23</sup> experimental inflammation of peri-implant tissue was induced and superstructures were placed whose occlusal height was excessively high by 100  $\mu\text{m}$ , as in the first study. As a result, notable bone resorption was seen as time passed. This suggested that there could be a risk that traumatic occlusal load, under conditions of inflammation, can rapidly increase the destruction of peri-implant tissue around the implant. This finding coincides with the results of various previous studies in animals.<sup>9-11,24-26</sup>



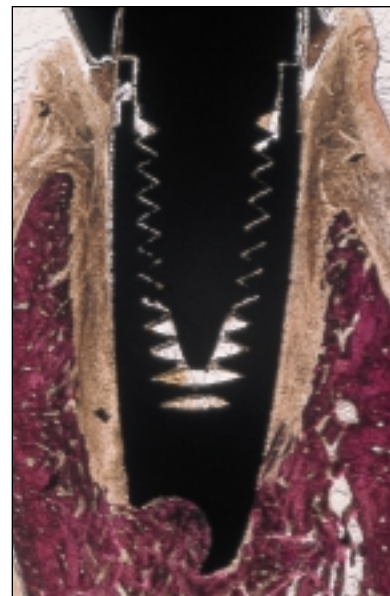
**Fig 6** (Left) The width at the implant placement site from buccal to lingual was measured at points A, B, and C using each section and analyzed by an image analytical computer. The maximum width at points A, B, and C was selected as the width at the implant placement site. (Below) Histopathologic view of control model (no occlusal loading). Most of the surface of each implant was well integrated with bone without obvious resorption around the implant (original magnification  $\times 10$ ).



**Fig 7** Histopathologic view of model in which supraocclusion was produced by excess height of 100  $\mu\text{m}$ . The implant was placed near the buccal side of the ridge crest; therefore, the coronal one sixth of the implant was exposed over the bone. However, most of the surface of each implant was well integrated with bone, without obvious resorption around the implant (original magnification  $\times 10$ ).



**Fig 8** Histopathologic view of model in which supraocclusion was produced by excess height of 180  $\mu\text{m}$ . Definite vertical bone resorption was seen from the marginal bone level to one half of the implant body on the buccal side. Bone destruction at the lingual side was not notable compared with the buccal side (original magnification  $\times 10$ ).



**Fig 9** Histopathologic view of model in which supraocclusion was produced by excess height of 250  $\mu\text{m}$ . Vertical bone resorption or destruction to almost the apex can be seen around the implant. Epithelial downgrowth can be seen in the space created by bone destruction (original magnification  $\times 10$ ).

**Table 2** Measurement Values of Maximum Width at Implant Placement Site from Buccal to Lingual and the Share of Implant in the Width

Implant model	Average of implants 1 and 2	
	Width (mm)	Share (%)
Model A	7.50	37.3
Model B	7.80	35.9
Model C	7.30	38.3
Model D	7.60	36.8
Average	7.55	37.0

Model A = control (no excess occlusal height); Model B = supraocclusion with excess height of 100  $\mu$ m; Model C = supraocclusion with excess height of 180  $\mu$ m; Model D = supraocclusion with excess height of 250  $\mu$ m.

However, investigation regarding the extent of excessive height with no inflammation in the peri-implant tissue and the magnitude of the load was performed. Therefore, in the present study, while peri-implant tissue health around the implants was maintained, the excess occlusal height of the superstructure was varied and response of the bone around the implants was investigated. Excess heights of 100  $\mu$ m, 180  $\mu$ m, and 250  $\mu$ m for the occluding structure were applied, and the response of the bone around the implants was histopathologically examined. When the excess was 180  $\mu$ m or more, bone resorption around the implants tended to be notable. This suggested that even though plaque was well controlled, when 180  $\mu$ m or more hyperocclusion was provided, bone resorption around the implants could be induced. This present study also demonstrated the tendency for peri-implant sulcus depth to increase over pre-occlusal loading in models with 180  $\mu$ m or more of excess height. This suggests that the destruction extended not only to bone, but also to the soft tissue around the implants.

Considering various factors such as oral environment, occlusal relations, occlusal forces, and the temporomandibular joint, this result in monkeys cannot be automatically applied to the human model. Also, most studies have been conducted in mandibles, where the magnitude or direction of the applied load is difficult to establish. Therefore, it is uncertain to what extent human occlusal relationships are reproduced. However, judging from the results of this present study, as well as those of the previous studies, for long-term maintenance and survival of implants it seems important to maintain

good plaque control in the peri-implant tissue around implants and not to create the equivalent of 100  $\mu$ m or more of excess occlusal height in superstructure fabrication. Also, it was significant that a baseline index of approximately 180  $\mu$ m was determined. This series of studies used implants that were 2.8 mm in diameter. In some studies involving monkeys, implants 3.5 mm or more in diameter were used.<sup>9-11</sup> Accordingly, the width at the placement site from buccal to lingual was measured in the present study for the purpose of comparing the ratio of implant width and buccal-to-lingual bone width between humans and experimental animals.

On average, 7.55 mm of width (range, 7.30 to 7.80 mm) was produced, and the implant share was 37.0%. According to a statistical report, measurements of the width in Japanese adult mandibles at the second molar site averaged 15.5 mm.<sup>27</sup> If the standard implant diameter is 3.75 mm, the ratio between implant width and buccal-to-lingual bone width was 24.0%. This result in monkeys shows nearly the same share level as in humans.

In conclusion, the results of the present study show that bone resorption around endosseous implants tends to increase with 180  $\mu$ m or more of excess height of a functioning superstructure. It also suggests that there is the possibility of bone resorption around implants caused by excess occlusal trauma, even when there is no inflammation in peri-implant tissue.

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