Comparing the occlusal contact area of individual teeth during low-level clenching

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(Received June 9, 2016; Accepted October 8, 2016)

Abstract: The aim of this study was to investigate the occlusal contact area (OCA) in individual teeth during low-level tooth clenching in 24 healthy participants. Before measurements were made, the 100% maximum voluntary contraction (MVC) was determined. At baseline, all subjects were instructed to close their mouth and touch the opposing teeth with minimal force. Occlusal contact was recorded during three jaw motor tasks (baseline, 20% MVC, and 40% MVC) using a blue silicone material. OCA thickness was determined from images and defined on five levels: level 1 (0-149 µm), level 2 (0-89 µm), level 3 (0-49 µm), level 4 (0-29 µm), and level 5 (0-4 µm). Premolar and molar OCAs increased significantly from baseline to 20% MVC and 40% MVC. The OCA of each anterior tooth did not change significantly with increasing clenching intensity at all levels. Our findings suggest that premolar and molar OCAs may be altered by low-intensity clenching, affecting the teeth and periodontal tissues.

Keywords: occlusal contact area; clenching; bite registration; tooth; periodontal tissues.

Introduction

It is essential to evaluate occlusal contact area (OCA) and occlusal contact points in patients before prosthodontic treatment. OCAs or occlusal points have been compared using occlusal articulating paper (1,2), occlusal strips or silk (3,4), alginate impressions (5-7), black silicone bite registration material (8), pressure-sensitive film (9,10), the T-Scan system (11,12), and silicone material. These studies demonstrated a positive correlation between OCAs or occlusal points and tooth clenching intensity. Gurdsapsri et al. compared OCAs at different levels of maximum voluntary tooth contraction (MVC) (10%, 30%, 70%, and 100%) controlled by electromyography (EMG) visual feedback, and observed a significant increase in the OCA with higher tooth clenching intensity (13). A previous study showed that occlusal contacts differed with low-intensity tooth clenching (14). However, the OCA has not yet been measured in individual teeth. Lavigne et al. defined the clinical diagnostic criteria for sleep bruxism based on macroscopic observations including tooth wear, but they did not evaluate occlusal contact (15). Alkan et al. suggested that clarification of the relationship between occlusal contact and bruxism would require the development of clinical diagnostic criteria for bruxism from occlusal contact (16). Determining the OCA of individual teeth during low-intensity tooth clenching may increase the accuracy of clinical diagnostic criteria for awake or sleep-related bruxism. In addition, Tamaki et al. defined patients with a sense of a malocclusion but no objective findings as having “occlusal discomfort syndrome” (17). However, no clinical diagnostic criteria
have been described for evaluating occlusal condition in patients with occlusal discomfort syndrome.

To determine clinical diagnostic criteria for bruxism or occlusal discomfort syndrome, the relationships between OCA and occlusal contact points in individual teeth need to be established in normal subjects. In the present study, we investigated whether differences exist in OCAs and occlusal contact points between incisor, premolar, and molar teeth in normal subjects during low-intensity tooth clenching.

**Materials and Methods**

**Subjects**

Twenty-four healthy participants (12 men, 12 women, mean age ± standard error of mean 24.3 ± 2.0 years) with complete dentition except for third molars and no known neurological disorders participated in this study. Abnormal stomatognathic function or bruxism were ruled out by evaluation of dental history using standard questionnaires and self-reports, as well as an oral examination using the research diagnostic criteria for temporomandibular disorder (18). All participants gave informed consent before the study began. Approval was obtained from the Institutional Ethics Committee (EC05-015) and the study was conducted in accordance with the guidelines of the Declaration of Helsinki.

**Experimental procedure**

Subjects sat upright and relaxed in a dental chair while measurements were taken and the head was supported by a headrest. Before measurements were taken, subjects performed a maximum clench to determine the 100% MVC. For the main experiment, subjects were asked to perform three low-intensity tooth clenching tasks (baseline, 20% MVC, and 40% MVC) as previously described (14) for 1 min. To avoid muscle fatigue, a 2 min interval was set between each task. Three jaw motor tasks were performed twice in randomized order. Visual feedback was used to determine 20% and 40% MVC. For baseline measurements, all subjects were instructed to close their mouths and touch the opposing teeth with minimal force. In all measurements, masseter muscle activity was recorded from all subjects.

**EMG acquisition and data analysis**

Disposable bipolar surface EMG electrodes (NM319Y, Nihon Kohden, Tokyo, Japan) were used to record surface EMG activity of the left masseter muscle (LM) and right masseter muscle (RM) in all subjects. The electrodes were positioned parallel to the main direction of the muscle fibers over the lower anterior part of the main muscle belly. This position was determined by palpation about 3 cm superior and anterior to the mandibular angle (19). The electrodes were positioned 10 mm apart along the central part of the muscle, midway between the anterior and posterior borders and superior and inferior borders of the LM and RM, approximately 2 cm lateral to the eyebrow. Visual feedback of muscle activity via the EMG signal was amplified 500 to 5,000 times and sampled at 1,500 Hz using a muscle balance monitor (GC, Tokyo, Japan). Masseter muscle EMG was recorded during all measurements with a time constant of 0.03 s, sensitivity of 0.5 mV/diV, and a sampling frequency of 1 kHz using a multitelemeter system (WEB-5000, Nihon Kohden). EMG signals were analyzed off-line after being transferred to wave analysis software (Powerlab, AD Instruments, Sydney, Australia). EMG activity was initially quantified during each task by calculating the root mean square (RMS) EMG amplitude in each 10 s epoch from both masseter EMG channels in all subjects. The relative ratios in each jaw motor tasks were also calculated from RMS EMG amplitudes of masseter EMG activity.

**Occlusal contact acquisition and data analysis**

An occlusal contact record was made during three jaw motor tasks (baseline, 20% MVC, and 40% MVC) using a blue silicone material (Blue Silicone, GC, Tokyo, Japan). The blue silicone material was prepared from an automatic mixing cartridge of silicone materials and injected onto the surfaces of mandibular teeth. The subjects were asked to close their teeth slowly into the maximum intercuspal position and to clench vertically with each jaw motor task for 1 min.

The OCA was calculated by an occlusal analysis device (BITEEYE BE-I, GC, Tokyo, Japan) based on the silicone registration materials. Silicone recording materials thicker than 5 mm were unable to calculate the OCA using this occlusal analysis device, therefore silicone recording materials were trimmed to less than 5 mm thick to maintain exact transmittance. As previously described (14), the OCA thicknesses were defined as level 1 (<150 µm: 0-149 µm), level 2 (<90 µm: 0-89 µm), level 3 (<50 µm: 0-49 µm), level 4 (<30 µm: 0-29 µm), and level 5 (<5 µm: 0-4 µm) in the image to calculate the OCA. The OCA in each tooth was divided using a maxillary tooth image on the computer screen and calculated from three jaw motor tasks according to the five thickness levels. The mean OCA values of each tooth were calculated for each jaw motor task from five different thickness levels of silicone registration material.
Statistical analysis

One-way analysis of variance was used to analyze the RMS EMG amplitude and the relative ratio of masseter EMG activity. The effects of the task on the OCA of individual teeth were analyzed by the Kruskal Wallis test with multiple comparisons (Mann-Whitney correction) for each level of silicone registration material thickness. $P$ values < 0.05 were considered statistically significant.

Results

EMG measurements

RMS EMG amplitude values at baseline, 20% MVC, and 40% MVC were: 0.014 ± 0.002, 0.037 ± 0.029, and 0.071 ± 0.069, respectively, and the relative ratios of masseter EMG activity were 11.1% ± 7.3%, 21.4% ± 8.7%, and 37.5% ± 13.6%, respectively. RMS EMG amplitudes and relative ratios of masseter EMG activity were significantly higher during 40% MVC than those of 20% MVC ($P < 0.05$) and baseline ($P < 0.001$). RMS EMG amplitudes and relative ratios of masseter EMG activity were significantly higher during 20% MVC than at baseline ($P < 0.001$).

Occlusal contact area

At level 1, OCAs at baseline, 20% MVC, and 40% MVC were: left side second molar (L7) (10.1 ± 5.4 mm², 12.6 ± 5.3 mm², and 5.1 ± 0.4 mm²); left side first molar (L6) (7.7 ± 4.6 mm², 10.1 ± 4.5 mm², and 10.5 ± 4.7 mm²); left side second premolar (L5) (2.7 ± 2.1 mm², 3.2 ± 2.0 mm², and 3.4 ± 1.9 mm²); left side first premolar (L4) (2.1 ± 2.0 mm², 2.8 ± 2.3 mm², and 2.9 ± 2.2 mm²); left side canine (L3) (0.8 ± 1.2 mm², 0.9 ± 1.4 mm², and 1.1 ± 1.4 mm²); left side lateral incisor (L2) (0.6 ± 0.6 mm², 0.7 ± 0.5 mm², and 0.8 ± 0.9 mm²); left side central incisor (L1) (0.6 ± 0.6 mm², 0.5 ± 0.5 mm², and 0.7 ± 0.6 mm²); right side central incisor (R1) (0.5 ± 0.6 mm², 0.6 ± 0.6 mm², and 0.6 ± 0.6 mm²); right side lateral incisor (R2) (0.5 ± 0.7 mm², 0.6 ± 0.6 mm², and 0.8 ± 1.0 mm²); right side canine (R3) (0.4 ± 0.5 mm², 0.6 ± 0.7 mm², and 0.7 ± 0.7 mm²); right side first premolar (R4) (1.8 ± 1.5 mm², 2.1 ± 1.4 mm², and 2.4 ± 1.5 mm²); right side second premolar (R5) (2.8 ± 2.0 mm², 3.3 ± 1.9 mm², and 3.5 ± 1.8 mm²); right side first molar (R6) (7.7 ± 3.9 mm², 10.4 ± 3.8 mm², and 11.0 ± 3.9 mm²); and right side second molar (R7) (8.3 ± 5.3 mm², 10.4 ± 4.5 mm², and 11.3 ± 4.8 mm²). There was a significant increase from baseline to 40% MVC in the OCA in R6 at level 1 ($P < 0.05$) (Fig. 1).

At level 2, OCAs at baseline, 20% MVC and 40% MVC were: L7 (6.8 ± 3.9 mm², 9.4 ± 4.0 mm², and 9.6 ± 3.8 mm²); L6 (5.2 ± 3.6 mm², 7.4 ± 3.6 mm², and 7.9 ± 3.9 mm²); L5 (1.8 ± 1.5 mm², 2.4 ± 1.6 mm², and 2.6 ± 1.5 mm²); L4 (1.4 ± 1.4 mm², 1.9 ± 1.8 mm², and 2.0 ± 1.8 mm²); L3 (0.5 ± 0.9 mm², 0.6 ± 1.1 mm², and 0.7 ± 1.1 mm²); L2 (0.4 ± 0.4 mm², 0.5 ± 0.4 mm², and 0.5 ± 0.7 mm²); L1 (0.2 ± 0.3 mm², 0.2 ± 0.2 mm², and 0.4 ± 0.3 mm²); R1 (0.3 ± 0.4 mm², 0.4 ± 0.4 mm², and 0.4 ± 0.4 mm²); R2 (0.3 ± 0.3 mm², 0.4 ± 0.4 mm², and 0.6 ± 0.8 mm²); R3 (0.2 ± 0.3 mm², 0.4 ± 0.6 mm², and 0.5 ± 0.6 mm²); R4 (1.2 ± 1.1 mm², 1.5 ± 1.0 mm², and 1.7 ± 1.1 mm²); R5 (1.8 ± 1.5 mm², 2.4 ± 1.6 mm², and 2.7 ± 1.5 mm²); R6 (4.6 ± 3.1 mm², 7.5 ± 3.2 mm², and 7.8 ± 3.0 mm²); and R7 (5.5 ± 4.1 mm², 7.6 ± 3.8 mm², and 8.2 ± 3.9 mm²). At level 2, the OCA in R6 increased significantly from baseline to 20% MVC, and bilateral molars significantly increased from baseline to 40% MVC ($P < 0.05$) (Fig. 2).

At level 3, OCAs at baseline, 20% MVC, and 40% MVC were: L7 (5.1 ± 2.9 mm², 7.9 ± 3.3 mm², and 8.2 ± 3.3 mm²); L6 (3.8 ± 2.9 mm², 6.0 ± 3.2 mm², and 6.6 ± 3.4 mm²); L5 (1.4 ± 1.2 mm², 2.0 ± 1.3 mm², and 2.2 ± 1.3 mm²); L4 (1.0 ± 1.2 mm², 1.6 ± 1.6 mm², and 1.7 ± 1.5 mm²); L3 (0.3 ± 0.6 mm², 0.4 ± 0.9 mm², and 0.5 ± 0.9 mm²); L2 (0.3 ± 0.3 mm², 0.4 ± 0.3 mm², and 0.4 ± 0.6 mm²); L1 (0.1 ± 0.2 mm², 0.1 ± 0.1 mm², and 0.2 ± 0.2 mm²); R1 (0.2 ± 0.3 mm², 0.2 ± 0.3 mm², and 0.3 ± 0.3 mm²); R2 (0.2 ± 0.3 mm², 0.3 ± 0.3 mm², and 0.5 ± 0.8 mm²); R3 (0.1 ± 0.3 mm², 0.4 ± 0.5 mm², and 0.4 ± 0.5 mm²); R4 (0.9 ± 0.9 mm², 1.2 ± 0.8 mm², and 1.4 ± 0.9 mm²); R5 (1.3 ± 1.3 mm², 2.0 ± 1.4 mm², and 2.3 ± 1.4 mm²); R6 (3.5 ± 2.7 mm², 6.1 ± 2.6 mm², and 6.6 ± 2.7 mm²); R7 (4.2 ± 3.5 mm², 6.3 ± 3.5 mm², and 7.0 ± 3.5 mm²). At level 3, the OCAs in bilateral molars increased significantly from baseline to 20% MVC, and those in R5 and bilateral molars increased significantly from baseline to 40% MVC ($P < 0.05$) (Fig. 3).

At level 4, OCAs at baseline, 20% MVC, and 40% MVC were: L7 (2.6 ± 1.9 mm², 5.5 ± 2.3 mm², and 6.0 ± 2.5 mm²); L6 (2.1 ± 1.8 mm², 3.9 ± 2.1 mm², and 4.7 ± 2.4 mm²); L5 (0.8 ± 0.7 mm², 1.4 ± 1.0 mm², and 1.6 ± 1.0 mm²); L4 (0.6 ± 0.7 mm², 1.1 ± 1.2 mm², and 1.1 ± 1.1 mm²); L3 (0.2 ± 0.3 mm², 0.2 ± 0.5 mm², and 0.3 ± 0.6 mm²); L2 (0.1 ± 0.2 mm², 0.2 ± 0.2 mm², and 0.3 ± 0.5 mm²); L1 (0.05 ± 0.09 mm², 0.05 ± 0.1 mm², and 0.08 ± 0.1 mm²); R1 (0.09 ± 0.1 mm², 0.1 ± 0.2 mm², and 0.1 ± 0.2 mm²); R2 (0.1 ± 0.1 mm², 0.1 ± 0.2 mm², and 0.3 ± 0.7 mm²); R3 (0.1 ± 0.1 mm², 0.2 ± 0.3 mm², and 0.2 ± 0.3 mm²); R4 (0.5 ± 0.7 mm², 0.8 ± 0.6 mm², and 1.0 ± 0.8 mm²); R5 (0.7 ± 0.9 mm², 1.4 ± 1.2 mm², and 1.6 ± 1.2 mm²); R6 (2.1 ± 2.0 mm², 4.2 ± 2.0 mm², and 4.9 ± 2.1 mm²); R7 (2.4 ± 2.2 mm², 4.4 ± 2.5 mm², and 5.0 ± 2.6 mm²). At level 4, the OCA increased significantly in the
R5 and bilateral molars from baseline to 20% MVC, and in bilateral second premolars and molars from baseline to 40% MVC ($P < 0.05$) (Fig. 4).

At level 5, OCAs at baseline, 20% MVC, and 40% MVC were: L7 (0.5 ± 0.5 mm$^2$, 1.9 ± 1.1 mm$^2$, and 2.6 ± 1.2 mm$^2$); L6 (0.4 ± 0.7 mm$^2$, 1.4 ± 0.8 mm$^2$, and 2.1 ± 1.0 mm$^2$); L5 (0.1 ± 0.2 mm$^2$, 0.4 ± 0.3 mm$^2$, and 0.6 ± 0.4 mm$^2$); L4 (0.1 ± 0.1 mm$^2$, 0.3 ± 0.3 mm$^2$, and 0.4 ± 0.3 mm$^2$); L3 (0.07 ± 0.1 mm$^2$, 0.07 ± 0.1 mm$^2$, and 0.1 ± 0.1 mm$^2$); L2 (0.06 ± 0.1 mm$^2$, 0.1 ± 0.1 mm$^2$, and 0.1 ± 0.3 mm$^2$); L1 (0.01 ± 0.03 mm$^2$, 0.01 ± 0.04 mm$^2$, and 0.02 ± 0.03 mm$^2$); R1 (0.01 ± 0.04 mm$^2$, 0.05 ± 0.1 mm$^2$, and 0.05 ± 0.1 mm$^2$); R2 (0.04 ± 0.09 mm$^2$, 0.07 ± 0.1 mm$^2$, and 0.2 ± 0.6 mm$^2$); R3 (0.02 ± 0.07 mm$^2$, 0.07 ± 0.1 mm$^2$, and 0.1 ± 0.1 mm$^2$); R4 (0.1 ± 0.2 mm$^2$, 0.3 ± 0.3 mm$^2$, and 0.4 ± 0.4 mm$^2$); R5 (0.1 ± 0.2 mm$^2$, 0.5 ± 0.4 mm$^2$, and 0.7 ± 0.5 mm$^2$); R6 (0.4 ± 0.6 mm$^2$, 1.5 ± 0.7 mm$^2$, and 2.1 ± 1.0 mm$^2$); R7 (0.6 ± 0.8 mm$^2$, 1.6 ± 0.9 mm$^2$, and 2.4 ± 1.2 mm$^2$). At level 5, the OCAs in bilateral premolars and molars increased significantly from baseline to 20% MVC and 40% MVC ($P < 0.05$) (Fig. 5). There were no significant differences in OCAs from 20% MVC to 40% MVC at all levels (Figs. 1-5). There were also no significant differences in OCAs of
incisors and canines from baseline to 20% and 40% MVC at all levels.

Discussion
In the present study, premolar and molar OCAs significantly increased at each level from baseline to 20 and 40% MVC, but no significant differences were observed from 20 to 40% MVC. Clenching intensity did not significantly change the OCA of anterior teeth at all detection levels.

Guadsapsri et al. discussed the mechanism by which changes in clenching intensity increase the OCA (13). Some studies showed that the mandible functions as a class III lever and tension vectors produced by isometric contraction of the jaw-closing muscles lie between the mandibular condyle and the dental arch (20-22). Mansour et al. suggested that the bite force between the molar teeth increases progressively in a non-linear but monotonic manner as the bite point moves posteriorly (23). In addition, Kikuchi et al. showed that the relative occlusal force ratio of the second molar increased with increasing clenching levels, while that of the canine decreased (24). These results suggest that contact distribution is altered in humans by shifts in regional occlusal loads during clenching. Non-rigidity of the bone and periodontal ligament allow minor tooth movement during forceful clenching. The greater the forces between antagonistic teeth, the greater are the tooth movements in the periodontal space, meaning closer intercuspatation and reduced space between antagonistic teeth (13). This may explain the significant increase in OCA in the posterior region.

Hidaka et al. compared the OCAs in upper teeth using a pressure-sensitive film (Dental Prescale, Fuji Film Co., Tokyo, Japan) with a thickness of 97 µm and showed that OCAs increased significantly with increasing clenching intensity. In their study, the OCAs at 30% MVC were: second molar, 3.3-4.6 mm²; first molar: 1.2-2 mm², second premolar: 0.3-0.5 mm², and first premolar: 0.2-0.4 mm² (9). The pressure-sensitive film had a similar thickness to level 2 in this study. Comparing OCAs between 30% MVC using the pressure-sensitive film and 20% MVC using a blue silicone material in a previous study, the OCA during 20% MVC using a blue silicone material at level 2 (<90 µm: 0-89 µm) was much larger than 0% MVC using the pressure-sensitive film (9). The bite recording material differed between these studies; therefore the reported differences in OCA may be explained by different characteristics of the material. On the other hand, Guardiansapsri et al. compared OCAs in the anterior part, right premolar, left premolar, right molar, and left molar using 50 µm black silicone material, and found that the OCAs in bilateral premolars and molars increased significantly with increasing clenching intensity. At 10% MVC, the OCAs were: second molar, 3.7-3.8 mm²; first molar, 3.7-4.0 mm²; second premolar, 1.4-1.6 mm²; and first premolar, 1.7-1.8 mm² (13). The detection level of 50 µm using black silicon material in their study was similar to level 3 in the present study. The OCAs were almost the same in these two studies (13). Our findings suggest that OCA measurement using silicon material is reproducible when different types of silicon material are used for bite registration.

The OCA thickness was defined as one of five levels and a negative correlation was found between OCA and thickness level. At level 5, tooth contact area significantly increased in all molar and premolar teeth with increasing clenching intensity, but this was not observed at level 1. In the future, use of the digital workflow technique will be more common and precise digital standardization from level 1 to level 3 will be required for occlusal analysis. The digital workflow for measuring OCAs must be technologically improved in the future. A limitation of this study was that the duration of each jaw motor task was set to 1 min because of the cure time of the silicone. The influence of jaw motor task duration on OCA measurements during bite registration should be investigated in the future.

In conclusion, the results of the present study suggest that low-intensity clenching may change OCAs in premolar and molar areas. Contact areas were affected by the tooth type and periodontal tissues. To develop clinical diagnostic criteria for bruxism or occlusal discomfort syndrome, the OCAs of individual teeth need to be investigated in patients with bruxism or occlusal discomfort syndrome and need to be compared with OCAs of normal subjects.

Acknowledgments
This study was supported by a Grant-in-Aid for Young Scientists (B 26861655) from the Ministry of Education, Culture, Sports, Science and Technology of Japan, and a Grant-in-Aid for Scientific Research (C 25463027 and C 26462959) from the Japan Society for the Promotion of Science.

Conflict of interest
The authors have no potential conflict of interest to declare.

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