

The effect of light premature occlusal contact on tooth pain threshold in humans

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SUMMARY The purpose of this study was to assess the influence of an experimentally induced light premature occlusal contact on tooth sensation. This assessment involved monitoring the electric tooth pain threshold (TPT) at multiple times before and after altering the occlusal contact. This alteration was produced by placing high inlays, which were measured with a custom made measuring device in maximum intercuspation. Data were collected on 10 teeth in 5 subjects, all whom were male and between the ages of 24 and 30 years. The contact area of the high inlays varied from 75 μm to 193 μm . The TPT

changes in these teeth ranged from -43 to +21% of their baseline level. Seven of these teeth showed a significant decrease ($P < 0.05$) in the TPT, one showed an increase and two did not show a significant change. Two of the teeth with a decreased TPT had cold water triggered occlusal pain, and 2 teeth had only occlusal pain. After several inlay adjustments to eliminate interference in maximum intercuspation, all tested teeth returned to their TPT baseline level and all symptoms disappeared. These results suggested that a light premature occlusal contact may change tooth sensation.

Introduction

Dentine hypersensitivity is caused by many aetiological factors, of which occlusion is one (Dowell & Addy, 1983). Occlusal abnormalities may induce an exposure of dentine as a result of attrition, and an excessive occlusal force may cause the genesis of a wedge-shaped defect at the cervical region of tooth (Spranger, 1995). Increasing stimulation to the dental pulp through exposing dentinal tubules in those lesions can induce dentine hypersensitivity (Yoshiyama *et al.*, 1990). Also, evidence from animal experiments has suggested that pulpal inflammatory reactions may also play a part in dentine hypersensitivity (Närhi *et al.*, 1994).

It has been reported in animal experiments that excessive occlusal force could cause inflammatory reactions and disturbance of blood circulation in periodontal and pulpal tissues (Kvinnslund & Heyeraas,

1992; Kvinnslund *et al.*, 1992) and that this can sensitize pulpal nerve (Edwall & Scott, 1971; Närhi, 1985; Olgart, 1985). Because there is a close relationship of blood circulation between periodontal and pulpal tissues (Seltzer & Bender, 1984), traumatic inflammation of periodontal tissues may extend easily to pulpal tissues. Clinical notions hold that dentine hypersensitivity may also be induced by very small (e.g. $< 200 \mu\text{m}$) premature occlusal contacts without obvious periodontal symptoms that could be relieved with occlusal adjustment (Ikeda *et al.*, 1987). Thus, it was supposed that even a light premature contact could induce dentine hypersensitivity.

Nevertheless, the effect of light premature occlusal contact on tooth pain threshold has not been examined. The purpose of this study was to test the hypothesis that a single experimentally induced light premature occlusal contact would not decrease the tooth pain threshold (TPT).

Table 1. Results of each experiment

Experiment No.	Subject	Tooth	Height (µm)	TPT change (max value %)	Symptom of tooth
1	No. 1	35	93 ± 2.7	Increase (+21)**	–
2	No. 1	35 ^s	149 ± 4.7	Decrease (–24)**	CWTP and OP
3	No. 2	35	123 ± 2.5	Decrease (–42)**	CWTP and OP
4	No. 3	45	122 ± 1.3	No change	–
5	No. 4	16	169 ± 3.5	Decrease (–16)**	–
6	No. 4	46	169 ± 3.5	No change	–
7	No. 4	16 ^s	193 ± 3.0	Decrease (–15)**	OP
8	No. 4	46 ^s	193 ± 3.0	Decrease (–14)**	OP
9	No. 5	16	75 ± 1.9	Decrease (–16)*	–
10	No. 5	46	75 ± 1.9	Decrease (–12)**	–

^s, second experiment; CWTP, cold water triggered pain; OP, occlusal pain.

*, $P < 0.05$; **, $P < 0.01$ (Mann–Whitney U-test)

Materials and methods

Subjects were five male volunteers who were between the ages of 24 and 30 years (mean ± SD: 27.0 ± 2.2) in good health, and who had no history of temporomandibular dysfunction. This study was conducted within the institutional standards regarding consent and protection of human subjects (i.e. if a subject developed symptoms of TMD and moderate or severe periodontal symptom, the experiment must stop immediately). Also, subjects were checked for any tooth symptoms and TMD (i.e. jaw muscle and joint pain, limitation of movement and joint noise) at every measurement.

Seven asymptomatic vital posterior teeth were selected that had Class 1 type amalgam fillings or inlays. Two experimental teeth were maxillary molars and five were mandibular (3 premolars and 2 molars). Three experimental teeth, in subjects 1 and 4 were used twice, using 2 different inlays over a 3-month interval. The total number of experiments was 10 (Table 1).

The outline of the experimental procedure was as follows:

- (i) Baseline TPT was determined for each experimental tooth using electrical stimulation before placement of the premature occlusal contact.
- (ii) A premature occluding inlay was placed on the experimental or opposing teeth and the height of each inlay in maximum intercuspation was measured.
- (iii) The TPT was monitored at multiple times after placement of the premature occlusal contacts and the occlusal adjustment.

The experimental premature occlusal contacts were introduced in two ways. Three experimental teeth that had Class 1 type inlays were prepared for new inlay cavities within the old inlay; this was done with continuous use of water coolant and a sharp high speed bur to minimize any substantial thermal irritation to the dental tissues. In these teeth no dental tissues were cut in the preparation. The high inlay was placed in this inlay cavity by Supper Bond C & B*. The other experimental teeth were given premature contacts by setting a high inlay on the opposing teeth. The level of occlusal interference was set to beyond the limits that the subject was able to masticate. Before inlay setting, it was adjusted to try and eliminate interferences of lateral movements.

The measurement of the height of each inlay in maximum intercuspation was carried out using an IP checker which is a custom made high-resolution measuring device (Hasegawa *et al.*, 1981). The IP checker measures the distance between the upper and lower targets (Fig. 1). It consists of 1.0 or 1.2 mm steel balls and their holder which are bonded to buccal surfaces of the tested teeth by Supper Bond C & B. The IP checker utilizes a Sony Magnesensor Set B2[†]. A calibration study of this device indicated a discrimination capacity of 1 µm, and an error of linearity of less than 1% between –125 µm and +800 µm. The measuring principle of the height of the inlay is shown in Fig. 2. The measurement was done in maximum

* Sun Medical Co., Kyoto, Japan.

[†] Sony Precision Co., Tokyo, Japan.

Fig. 1. IP checker was fixed onto the maxillary anterior teeth using the stent glued with cyanoachrylate. The upper and lower targets, consisting of 1.0 or 1.2 mm steel balls and their holder were bonded by Supper Bond C&B (Sun Medical Co.)

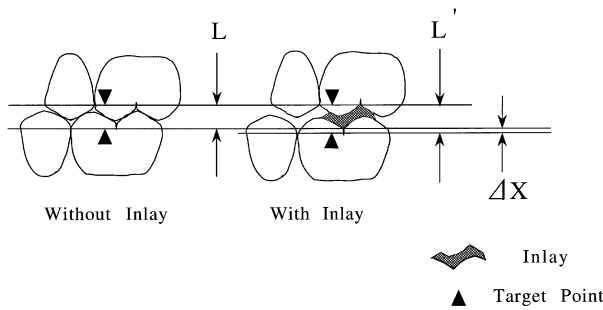
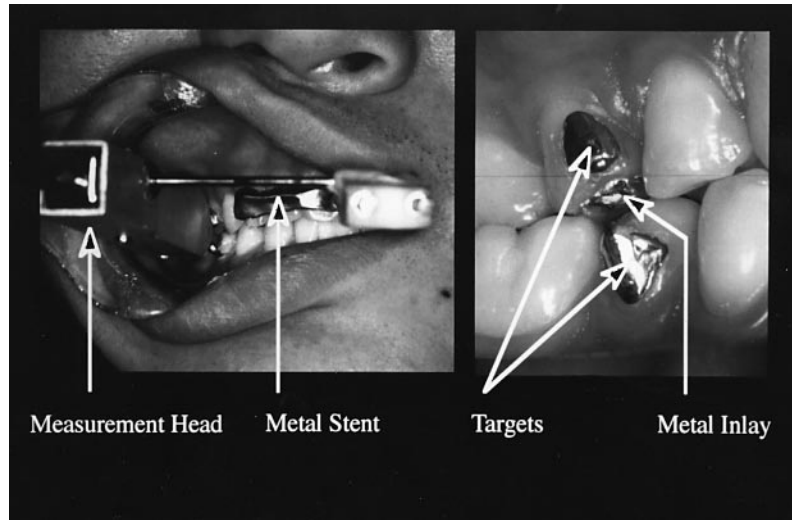


Fig. 2. The principle for measuring the height of the occlusal inlay. The inlay height in maximum intercuspation was calculated by the following expression. $\Delta X = L' - L$

intercuspation with light clenching. The mean value of five measurements indicated the inlay height.

All TPT assessments in this study were performed using a universal stimulation control module VSC6 and voltage stimulation isolator IS/V[‡]. Monopolar negative square pulse stimulation at 50 Hz and 0.5 ms duration was applied to the experimental teeth under rubber-dam isolation. To make clear the sensation at pain threshold level, adequate series impedance were selected between 0.3 and 10 MΩ for each experimental tooth. The Class 1 amalgam fillings, or the inlays of the experimental teeth were used as the electrode to stabilize the stimulating condition, and an indifferent electrode was held in the subject's right hand. The strength of stimulation was kept from the subjects during TPT measurement. Five measurements were made per day. A baseline TPT was determined from

[‡] Medelec Ltd, U.K.

the measurements gathered before application of the premature occlusal contact.

Results

Data available from 10 teeth in 5 subjects revealed TPT change from -42% to +21% of the baseline value (Table 1). The interference in occlusal contacts ranged from 75 to 193 μm. The standard deviation of inlay height was less than ± 5 μm (Table 1). The TPT of each experiment was divided into two groups: (1) before the application of the premature occlusal contact (baseline), and (2) during the application of the premature occlusal contact and the first occlusal adjustment; the Mann-Whitney U-test was used for statistical analysis for two groups. Eight of the teeth, 4 maxillary and 4 mandibular teeth, showed a significant decrease in TPT. One tooth showed a significant increase in TPT. Two teeth, both mandibular premolars, did not show a significant change in TPT (Table 1; Figs 3, 4 & 5). After several occlusal adjustments, TPT approached the baseline and all experimental teeth recovered to the baseline level during experimental period.

No subjects had any symptom of TMD or any moderate or severe periodontal symptom during the experimental period. Two of the teeth with a decreased TPT had pain triggered by cold water and occlusal pain, and 2 teeth had only occlusal pain (Table 1). All tooth symptoms were relieved after the occlusal adjustment.

The averaged TPT values within 2-day periods of each experiment, were gathered from all the experiments for comparison with the baseline, using the Wilcoxon

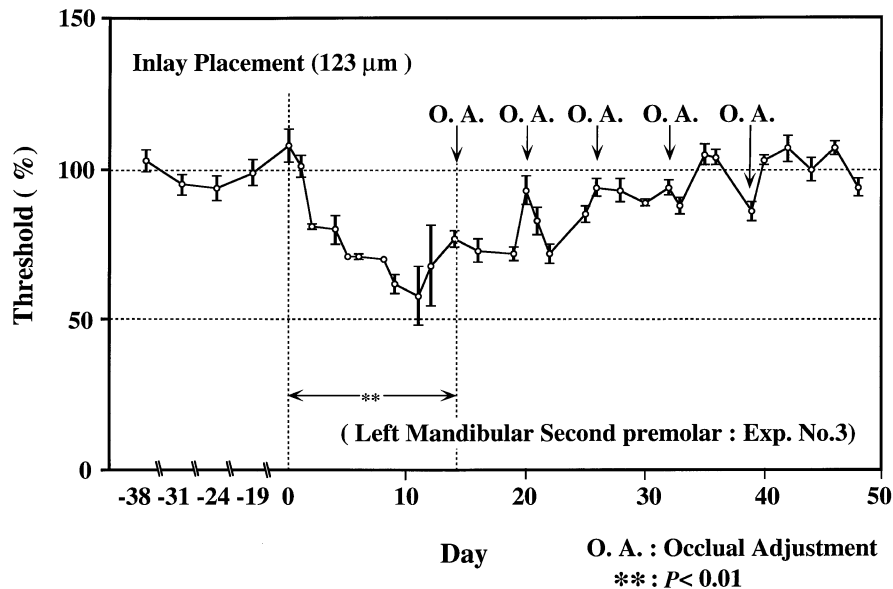


Fig. 3. An example of a decreased TPT. Following placement of the 123 µm-high inlay, TPT of a left mandibular second premolar (experiment 3) was decreasing significantly (**: $P < 0.01$). After 11 days, TPT decreased to -42% of the baseline level. TPT recovered to the baseline, after two visits for occlusal adjustment.

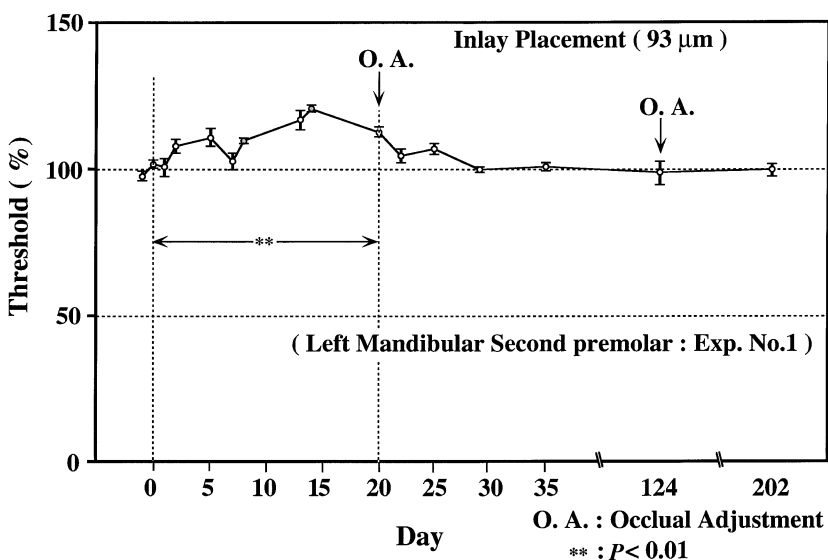


Fig. 4. An example of increased TPT. Following placement of a 93 µm - high inlay, TPT of a left mandibular second premolar (experiment 1) increasing significantly. After 14 days, TPT increased to + 21% over the baseline value. TPT recovered to the control level after two visits for occlusal adjustment.

Test. This analysis was done for 4 periods (up to the 8th days), because the minimum period from inlay setting to the first occlusal adjustment was 9 days. TPT data of the first and second sections could note a significant deviation ($P > 0.05$) from the baseline (Table 2).

In the experiments where two different heights were applied to the same tooth. The results showed the following: One mandibular second premolar had both an increase in the TPT (experiment 1, 93 µm) and a decrease (experiment 2, 149 µm) along with cold water triggered pain and occlusal pain. One maxillary first molar had a decrease in both cases (experiment 5, 169 µm and experiment 7, 193 µm). Finally, one

mandibular molar showed no significant change in the TPT (experiment 6, 169 µm) and a decrease (experiment 8, 193 µm).

Discussion

Since a sample of 5 men is very limited, the result of this study cannot be extrapolated to a border population. However, we think that the design of this study could clarify the possibility that a light premature contact effected tooth pain sensation. Although a tooth on the opposite side of the same arch should be used as control, the TPT elicited by electrical stimulation has been

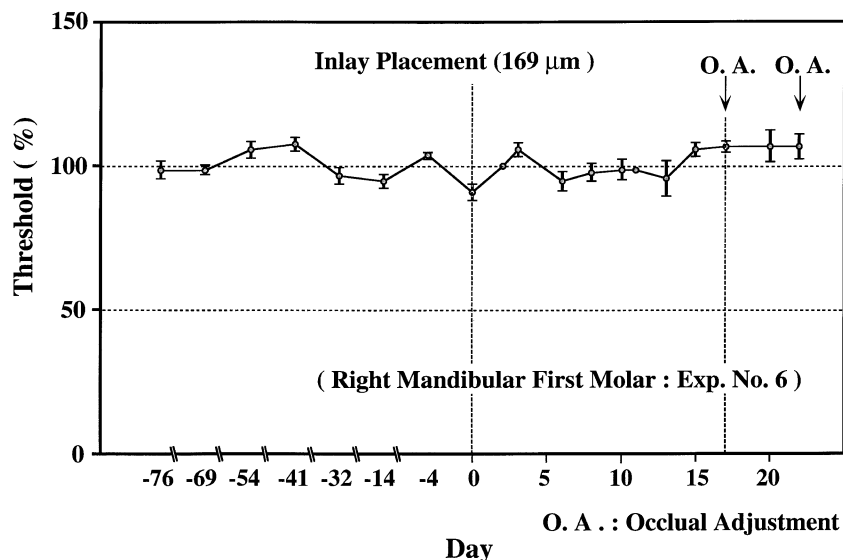


Fig. 5. An example of no change in TPT. After placement of a 169 μm-high inlay, TPT of a right mandibular first molar (experiment 6) did not change significantly.

Table 2. Baseline versus TPT averaged values by each 2-day period

	Period (day numbers)			
	1-2	3-4	5-6	7-8
<i>P</i> -factor	0.0499	0.0355	0.0966	0.1677

Wilcoxon Test

reported to be an accurate and reproducible method for pain measurement (Anderson, Hannam & Matthews, 1970). Monopolar electrical tooth stimulation at the threshold level has very little possibility of eliciting a response from non-pulpal nerve fibers (Matthews, Horiuchi & Greenwood, 1974). Deviation of occlusal measuring was less than 5 μm, but it changed day by day owing to the inclination, migration and attrition of tooth. Thus, the occlusal height of this study represents a qualitative index of premature contact on the inlay placement day.

All subjects were young males who had not been TMD patients, and the premature contacts were small and adjusted to exclude an interference of lateral jaw movements at inlay placement. These might be the reason that they did not have any symptoms of TMD in this study. The experimental teeth that placed less than 100 μm interference did not have any tooth symptoms, so this level of interference might not induce an obvious occlusal trauma in normal periodontal tissue.

It has been reported in animal experiments that excessive occlusal force could produce trauma, inflammatory reaction and circulatory disturbances in

periodontal and pulpal tissues (Kvinnslund & Heyeraas, 1992; Kvinnslund *et al.*, 1992). Also the effect of orthodontic force on the human tooth has been reported in the past. Orthodontic force may cause trauma and inflammatory reaction in periodontal tissues (Rygh *et al.*, 1986), cell damage and circulatory disturbances (Nixon *et al.*, 1993), inflammatory changes and growth of microvessels (Derringer, Jagers & Linden, 1996). Although orthodontic force is a continuous force and occlusal force is an intermittent force, the pathohistological pattern of premature occlusal contact in animal experiments resembles that of orthodontic force in human experiments. The latter have suggested that traumatic occlusal force may induce circulatory disturbances and inflammatory reactions in human pulpal tissue. It has been reported that some cases of dentine hypersensitivity induced by exposure of dentine may remain after the tubules are effectively closed, and that this may be due to pulpal inflammation and consequent sensitization of the intradental nerves (Närhi *et al.*, 1994). As the pulpal inflammatory reaction of light initial contact in this experiment may be milder than that of premature contact in animal experiments, we suggest that some pulpal changes may have occurred where there is hypersensitivity caused by light premature contact. Also it was reported that excessive occlusal force may cause the genesis of a wedge-shaped defect at the cervical region of tooth (Spranger, 1995). We think that some cases of dentine hypersensitivity might be induced by and in combination with both effect of premature occlusal contact.

Most pulpal nerve fibers consist of A-delta and C-

fibres that mediate pain information (Olgart, 1985; Johnsen, 1985). Recent evidence from animal experiments show that intradental A type nerve fibres mediate the sensitivity of dentine and are activated by fluid movements of dentinal tubules (Närhi *et al.*, 1994). Because the threshold of A-delta fibres is about one fourth or one fifth as much as that of C-fibres, A-delta fibre firing thresholds determine the TPT induced by electrical stimulation (Närhi, 1985; Olgart, 1985). A-delta fibres are sensitized by pain inducing substances and by the mechanical distortion of dental pulp tissue due to a disorder of pulpal blood circulation (Närhi, 1985; Olgart, 1985). On the other hand, decreased blood flow of pulp tissue, or increasing pressure surrounding would easily depress the activity of pulpal A delta fibres (Edwall & Scott, 1971; Närhi, 1985; Olgart, 1985). We hypothesize, therefore, that TPT change by premature contact might be dependent on the pulpal inflammatory reactions and/or the change of pulpal blood flow.

The change of TPT appeared quickly after altering occlusal contact, and was prolonged for a few days (Table 2). As this study is an acute experiment, this result cannot be extrapolated to a chronic case. However a chronic dentine hypersensitivity owing to an occlusal overload has been reported (Ikeda *et al.*, 1987). It could also be the result of functional changes in the connections of the intradental nerves in central nervous system (Sessele, 1987).

It was reported by Yamada (1983) that the maxillary alveolus was more susceptible to traumatic occlusal forces than the mandibular alveolus due to differences in their structure. This could explain why all maxillary experimental teeth had decreased TPT values and the TPT values of two mandibular teeth did not change significantly. Because an increase of TPT does not induce the symptoms of dentine hypersensitivity, we cannot notice this phenomenon at the chair-side. The experiments with two different heights applied to the same teeth showed different results in each tooth. When a right second premolar was altered by the high inlay of 93 μm (experiment 1), it showed an increase in TPT without any tooth symptoms. The same tooth increased by 149 μm (experiment 2), indicated a decrease in TPT along with cold water triggered pain and occlusal pain. This indicated that the periodontal and pulpal damage of increase in TPT was milder than where it was decreased. A left mandibular molar showed no significant change in the TPT (experiment 6, 169 μm) and a decrease (experiment 8, 193 μm) with an occlusal

pain. A left maxillary first molar which was opposite tooth of experiment 6, 7 had a decrease in both cases (experiment 5, 169 μm and experiment 7, 193 μm). So, the direction and degree of TPT change may depend on the flexibility of each tooth towards the excessive occlusal force.

In summary, a light premature occlusal contact (less than 200 μm in height) was able to induce dentine hypersensitivity both objectively (TPT) and subjectively (reported symptoms). The results of this experiment provide objective data on the magnitude of this threshold change. Removal of a high occlusal contact was associated with a return to TPT baseline. We conclude that a light premature contact is one of the aetiologic factors of dentine hypersensitivity, and occlusal adjustment is the most effective treatment for it.

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