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Sensory and motor changes of the human jaw muscles during induced orthodontic pain

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SUMMARY The aim of this study was to evaluate the short-term effects of orthodontic pain on the pressure pain threshold (PPT) of the masseter and anterior temporalis muscles, and on their electromyographic (EMG) activity during clenching and chewing. Orthodontic pain was induced in 14 healthy subjects (mean age 26.6, SEM 1.1) by placing orthodontic separators. The subjects were randomly assigned to an experimental and to a control session in a double-blind crossover study.

PPT was significantly reduced (Student's *t*-test; $P < 0.001$) after experimental sessions for both the masseter and the anterior temporalis muscles, whereas no significant differences were found during control sessions ($P > 0.05$). EMG activity during clenching and chewing was significantly reduced ($0.001 \leq P < 0.05$) after experimental sessions for both masseter and anterior temporalis muscles, whereas no significant differences were found during control sessions ($P > 0.05$).

The decrease of PPT found in this study can be related to the occurrence of muscle pain and headache reported by patients during orthodontic or other dental treatment. The decrease of EMG activity of the jaw muscles associated with orthodontic pain is consistent with the pain adaptation model and should be considered as a potential factor for loss of occlusal anchorage during orthodontic treatment.

Introduction

Orthodontic therapy is frequently associated with the occurrence of dental pain. This pain begins a few hours after application of the force, reaches a peak after 24 hours, and lasts for approximately 5 days (Ngan *et al.*, 1989; Scheurer *et al.*, 1996). In clinical practice, following the application of orthodontic forces, a number of patients report the occurrence of headaches and pain in other facial sites (Scheurer *et al.*, 1996). These complaints appear to occur more frequently in patients wearing headgear, intermaxillary elastics, or orthodontic separators.

Previous studies (Ohrbach and Gale, 1989a,b; Reid *et al.*, 1994) suggest that pressure algometry is a valid tool for the measurement of orofacial pain. A decreased pressure pain threshold (PPT) has been found in headache sufferers (Schoenen *et al.*, 1991; Bovim, 1992), and in subjects with both myogenous and arthrogenous temporomandibular disorders (TMD; Ohrbach and Gale, 1989b; Chung *et al.*, 1993).

Patients undergoing orthodontic treatment also report an impairment of chewing and biting ability. Noteworthy, a decrease of EMG functional activity of the masseter muscle following archwire adjustments has recently been described (Goldreich *et al.*, 1994).

The present study was designed in order to investigate the sensory and motor effects of orthodontic pain on the human jaw muscles. This pain was induced by means of orthodontic separators. Sensory and motor effects were assessed by means of pressure algometry and surface electromyography.

Two brief communications of this study have been published (Martina *et al.*, 1997; Michelotti *et al.*, 1997).

Subjects and methods

Subjects

Fourteen young adults (six men and eight women) recruited from the staff and graduate students of the Dental School of Naples, participated in this study. The mean age ± SEM of the subjects was 26.6 ± 1.1 years. All subjects gave informed consent to the procedures**,** which were approved by the local ethical committee. They had a complete natural dentition (with the exception of third molars), and were asymptomatic for craniomandibular disorders and orofacial pain (absence of jaw dysfunction, headaches, and muscle pain or soreness). Additional exclusion criteria were: (i) current orofacial inflammatory conditions; (ii) dental or orthodontic treatment during 3 months prior to the study; (iii) intake of medications or other treatment at the time of the study; and (iv) neurological or metabolic disorders.

Experimental design

The study was carried out with a double-blind crossover design. Each subject was assigned to the experimental or control session, with a balanced block randomization.

Assessments included the following variables: the PPT of the masseter and anterior temporalis muscles, the masseteric and temporal EMG activity during clenching and chewing, and the subjective evaluation of the pain experienced during chewing on a visual analogue scale (VAS).

In the experimental session, baseline recordings of PPT, EMG activity, and VAS scores were assessed in a sequential order. After these assessments, eight orthodontic separators (S45L100, GAC Inc., New York, USA) were placed in each subject at the mesial and distal contacts of all the first molars in order to induce orthodontic pain. The subjects returned after 24 hours and the separators were removed. Such an interval is expected to cause a peak in the level of pain experienced (Ngan *et al*., 1989; Scheurer *et al*., 1996). Immediately after the removal of separators PPT, EMG, and VAS were again assessed.

In the control session, PPT, EMG and VAS were measured following the same procedure as that of the experimental session, but the separators were removed 10 minutes after their placement.

After a 1-week wash-out period, each subject returned again and separators were applied in a reverse order (i.e. subjects who participated in the experimental session first, underwent the control session, and *vice versa*). Following this design, each subject served as his own control.

Algometric measurements

PPT was evaluated by means of an electronic algometer (Somedic AB, Farsta, Sweden). The instrument consists of a gun-shaped handle with a pressure-sensitive strain gauge at the tip. The tip is coated with soft rubber and has a surface of 1 cm^2. The algometer display shows pressure (in kPa) and the rate of pressure increase (kPa/second). In this study, a rate of approximately 20 kPa/second was chosen (Schoenen *et al*., 1991). Good reliability and validity of algometric measurements in both musculoskeletal pain syndromes and asymptomatic subjects have been previously reported (Ohrbach and Gale, 1989a,b). The PPT was determined as the point at which the pressure stimulus applied to the skin changed from a sensation of pressure to pain. The subjects indicated the PPT by pressing a push-button, which froze the current pressure value on the digital display.

Before starting, the subjects were carefully instructed concerning the whole procedure and a few test measurements were performed on their hand. The subjects sat in a dental chair, and were asked to relax and keep the teeth apart during the recordings (McMillan and Lawson, 1994). While assessing the PPT, the subject's head was supported by counter-pressure from the opposite hand of the operator. During the measurements, the algometer was held perpendicular to the skin. Algometric measurements were performed on two sites located on the right masseter and on the right anterior temporalis (Figure 1). For the masseter muscle, the following sites were chosen: M1 was located over the most prominent part of the muscle, as determined by palpation during

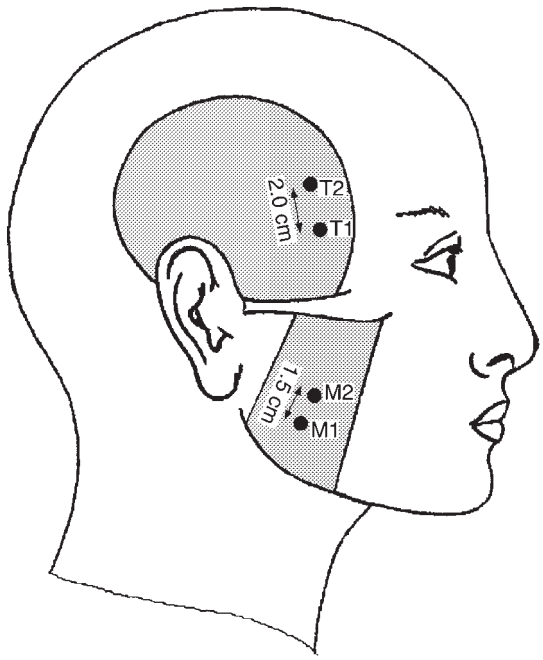

Figure 1 Schematic representation of the algometric sites located on masseter and anterior temporalis muscles. The same sites were used for placement of the electrodes.

voluntary contraction; M2 was located 1.5 cm superiorly to M1, along the main direction of the muscle fibres. For the temporalis muscle the following sites were selected: T1, a line between the upper orbital margin to the upper point of the outer ear, 2 cm behind the anterior border of the muscle. This border was determined from palpation during forceful voluntary contraction. T2 was located 2 cm superiorly to T1, along the main direction of the muscle fibres. To ensure precise relocation of these sites in each session, a transparent pliable plastic template was aligned to the ear, to the labial margin, and to the eye, and the location of the sites was marked.

The sites were measured in a sequential order M1–T2 with approximately a 5-second interval between measurements. The distal phalanx of the third finger of the ipsilateral hand was chosen as a control non-cranial site. Four PPT measurements were made at each recording site with a 2-minute rest interval between trials. Since the first PPT assessment has been shown as being highly variable (Wolff and Jarvik, 1964; Fagius and Wahren, 1981), it was discarded and each PPT was defined by the mean of the successive three trials.

Assessment of muscle activity

Activity of the masseter and anterior temporalis was recorded unilaterally (right side) in each session by means of disposable surface electrodes (Bio-Research, Milwaukee WI, USA). The electrodes were circular with a diameter of approximately 10 mm. The skin over the muscles was rubbed vigorously with ether and the electrodes were then placed on the sites chosen for the algometric measurements. The ground electrode was fixed to the ipsilateral mastoid process. Relocation of the electrodes was obtained by means of the plastic template previously described. EMG activity was monitored with commercially available equipment (Bio-Pak, Bio-Research, Milwaukee WI, USA), and was amplified (×5000) and filtered between 25 and 1500 Hz. Analogue-to-digital conversion was undertaken at a rate of 9600 samples per second, the EMG signals were then averaged, and stored on floppy disks for subsequent analysis. EMG assessments included maximal voluntary contraction (MVC) in the intercuspal position and unilateral chewing activity in the muscles under study. To evaluate MVC, the subjects were asked four times to clench as hard as possible. The duration of each clenching was 1–2 seconds with a rest interval of 1 minute between trials. The peak amplitude of the averaged, rectified electromyogram was used as an indication of the maximal clenching activity. After clenching the subjects were allowed a 5-minute rest before starting to chew. The subjects were asked to chew a standard gum (Seugim, Perfetti, Istanbul, Turkey) on the right side. Thirty seconds after chewing started, EMG recordings with a duration of 5 seconds were taken.

In the subsequent analysis of each EMG recording, the peak amplitudes of five successive chewing strokes were averaged as a single estimate of EMG chewing activity (Michler *et al.*, 1988).

Visual analogue scale (VAS)

During chewing, perceived pain was rated after 30 seconds on a 100-mm VAS. This scale is widely used for measuring pain, and has been described as being sensitive and reliable (Huskisson, 1974). The left end-point of the scale indicated 'no pain at all' and the right end-point corresponded to 'worst pain I can now imagine'. The subjects were carefully instructed to rate their pain on VAS in a reliable manner. The subjects were additionally asked whether the insertion of separators induced changes of the frequency of dental contacts (i.e. clenching/grinding of the teeth) and were allowed to choose one of three possible answers (frequency increased, unchanged, or decreased).

Statistics

All data collected were preliminarily analysed using the Kolmogorov–Smirnov test. Because this test failed to show normality for VAS scores, they were analysed by means of non-parametric Wilcoxon–Pratt matched pairs test. Since the hypothesis that the muscle activity and PPT were normally distributed could not be rejected, subsequent analysis of EMG and PPT was performed by means of a paired Student's *t*-test.

Changes in VAS, EMG, and PPT were expressed as a percentage of baseline values. All the data were analysed using commercial statistical software (Statgraphics Plus 2.0, Manugistics Inc., Rockville, Maryland, USA) and statistical significance was accepted at $P < 0.05$ (two-tailed). The results are presented as the mean (M) and the standard error of the mean ± SEM.

Results

Subjective findings

All the subjects investigated reported the occurrence of dental pain while wearing the separators during the experimental sessions. This justified subsequent evaluation of sensory and motor changes associated with such pain.

After 24 hours, the amount of pain perceived during chewing, as assessed on VAS, was 35 ± 10 mm after the experimental sessions, whereas pain reported after the control sessions was almost negligible (0.3 ± 0.6 mm). These scores were significantly different (Wilcoxon test; $P < 0.0001$).

After experimental sessions, 10 subjects (71 per cent) reported a decrease of the frequency of tooth contacts, whereas four subjects (29 per cent) reported an increase; no change in the frequency of tooth contacts was reported after control sessions by any of the subjects investigated (100 per cent).

Pressure pain thresholds

Preliminary analysis of the PPT at the recording sites was carried out; the PPT was significantly higher at the temporal than at the masseteric sites ($0.001 < P \leq 0.01$). Within-muscle comparison of sites (site M1 versus M2 and T1 versus T2) did not reveal significant differences ($P > 0.05$), consequently the data from the two sites from each muscle were averaged.

Algometric measurements assessed during experimental and control sessions are given in Table 1. The PPT was significantly reduced ($P < 0.001$) after experimental sessions for both

Table 1 Mean values ± SEM of the pressure pain thresholds (PPT) for experimental and control sessions.

PPT (kPa)	Anterior temporalis			Masseter		
	Baseline	After 24 h	*P*	Baseline	After 24 h	*P*
Control session	267.0 ± 35.1	256.7 ± 30.1	NS	201.8 ± 20.1	203.1 ± 18.8	NS
Experimental session	286.7 ± 35.6	233.3 ± 25.4	***	193.7 ± 19.7	160.6 ± 14.7	***

P, level of statistical significance (two-tailed); NS, not significantly different, ***$P < 0.001$.

Table 2 Mean values ± SEM of the EMG activity during maximal clenching and chewing for experimental and control sessions.

	Anterior temporalis			Masseter		
	Baseline	After 24 h	*P*	Baseline	After 24 h	*P*
Maximal clenching activity (μV)						
Control session	262.0 ± 20.0	264.0 ± 19.9	NS	348.3 ± 31.2	362.3 ± 33.9	NS
Experimental session	283.0 ± 21.0	220.0 ± 18.5	**	368.5 ± 32.4	260.5 ± 38.7	**
Chewing activity (μV)						
Control session	117.0 ± 8.8	112.1 ± 7.4	NS	140.8 ± 12.9	156.0 ± 12.8	NS
Experimental session	117.4 ± 6.5	97.2 ± 6.6	**	160.3 ± 16.6	121.6 ± 14.0	*

P, level of statistical significance (two-tailed); NS, not significantly different, *$P < 0.05$, **$P < 0.01$.

masseter (–15.5 ± 2.6 per cent) and anterior temporalis muscles (–17.4 ± 2.2 per cent). By contrast, no significant differences ($P > 0.05$) were found during control sessions (masseter: +2.2 ± 2.7 per cent, anterior temporalis: –1.9 ± 3.6 per cent). Pain thresholds at the third finger did not show significant changes ($P > 0.05$) both after control and experimental sessions (+5.5 ± 5.1 and –6.6 ± 4.6 per cent, respectively).

Muscle activity

Masseteric and temporal electromyographic activity assessed during experimental and control sessions is summarized in Table 2.

EMG activity during clenching and chewing was significantly reduced ($0.001 \leq P < 0.05$) after experimental sessions for both the masseter and the anterior temporalis muscles (Table 2). The mean decrease of maximal EMG activity was –30.5 ± 7.5 per cent for the masseter muscle and –23.1 ± 7.8 per cent for the temporalis muscle. The mean decrease of chewing activity was –20.6 ± 6.05 per cent for the masseter muscle and –15.5 ± 5.7 per cent for the temporalis muscle. No significant differences ($P > 0.05$) in functional activities were found after control sessions for both muscles investigated (clenching: +5.4 ± 4.9 per cent for the masseter muscle and +1.6 ± 3.8 per cent for the temporalis muscle; chewing: +8.3 ± 4.6 per cent for the masseter muscle and –2.6 ± 3.5 per cent for the temporalis muscle). Changes of PPT and EMG activity did not differ between males and females (Student's *t*-test: $P > 0.05$).

Discussion

In agreement with previous reports (Ngan *et al.*, 1989), the findings in this study suggest that the application of orthodontic separators over a 24-hour period induces pain in the teeth. Although this pain occurred in all the subjects investigated during the experimental sessions, the amount was highly variable, as some subjects reported unbearable pain, whereas others reported only slight to moderate pain. This variability was also reflected in the VAS scores assessed during gum chewing and may be related to local anatomical (e.g. amplitude of interdental spaces) and central-psychological factors that can affect pain perception (Gamsa, 1994).

The mechanisms of pain resulting from orthodontic forces are not yet fully understood. The pain perceived on the teeth has been related to the levels of prostaglandin and substance P in the periodontal ligament, and is probably associated with an inflammatory response (Kamogashira *et al.*, 1988; Marklund *et al.*, 1994). The application of orthodontic forces may also injure the periodontal nerve terminals. This suggestion is supported by the observation that orthodontic forces can affect force thresholds and discharge frequencies of the periodontal receptors (Loescher *et al.*, 1993), and that the distribution of nerve growth factor-receptors is

altered during experimental tooth movement (Saito *et al.*, 1993).

The EMG findings of the present study show a significant decrease of temporal and masseteric muscle activity during maximal clenching and chewing. These results are in agreement with Goldreich *et al*. (1994), who reported a reduction of masseteric EMG activity after orthodontic archwire adjustment and support 'the pain adaptation model' (Lund *et al*., 1991). In the orofacial region, this model explains why pain arising from temporomandibular joints, teeth, and other non-muscular tissues can affect the motor control of the jaw muscles. Briefly, the nociceptive afferents from cutaneous, somatic, and visceral tissues converge in the central nervous system (CNS), and interact with alpha motor neurons along reciprocal pathways. The motor effects of nociceptive inputs are described as 'a decrease in the motor neurons' output, when the muscle is acting as an agonist, and an increase in the output, when it acts as an antagonist'. Thus, the reduction of masseteric and temporal motor output found in the present study can be explained by the interaction between periodontal nociceptive afferents and inhibitory interneurons supplying the jaw-closing motor neurons.

In this study, a small but highly significant decrease of PPTs was found after the experimental sessions for both the masseter and anterior temporalis muscles. This sensitization of the jaw muscles after inducing pain in the teeth may be speculatively ascribed to several peripheral and central mechanisms.

With respect to peripheral mechanisms, it is interesting to note that the occurrence of pain, headache, and tenderness of the human jaw muscles has been reported after the insertion of occlusal interferences (for review, see Christensen and Rassouli, 1995). These clinical signs and symptoms have been related to an abnormal motor behaviour of the jaw musculature (Funakoshi *et al.*, 1976; Sheikholeslam and Riise, 1983). Muscle hyperactivity can result in mechanical or chemical tissue trauma and, consequently, in a peripheral sensitization of muscle nociceptors (Hargreaves *et al.*, 1995). Since the orthodontic separators used in the present study may interfere with the intercuspal position, they could trigger muscle over-use or hyperactivity. To our knowledge, PPT of the jaw muscles after insertion of occlusal interferences has not previously been evaluated, but Clark *et al*. (1989, 1991) and Bakke *et al.* (1996) suggest that prolonged overloading of the masticatory system does not affect the pressure pain thresholds of the jaw elevator muscles in healthy subjects. In the present study, the subjects investigated were asked whether insertion of the separators had induced changes in the frequency of tooth contacts. It is noteworthy that the majority of them (10 subjects) reported a decrease in the frequency of tooth contacts during the experimental period, whereas only four subjects reported an increase. Based on the subjective and EMG findings, in this investigation, the hypothesis that the insertion of orthodontic separators induced muscle hyperactivity and consequently a peripheral sensitization of the jaw muscles, is not supported. Nevertheless, a definitive answer to this hypothesis requires further investigation. This research should evaluate long-term masticatory activity by means of portable EMG recorders (e.g. Gallo and Palla, 1995).

Algometric measurements reflect the combined effects of peripheral nociception and central pain processing. Therefore, a lowering of pain thresholds may also be related to a central mechanism.

There is increasing knowledge (Woolf, 1991; Coderre *et al*., 1993; Woolf *et al*., 1994) of changes that take place in the CNS as a result of peripheral injury. These changes include central hyper-excitability of the second order neurons (spinal cord or brain stem) and altered CNS processing of noxious input (Roberts and Foglesong, 1988). In the orofacial region, wide dynamic range and nociceptive specific neurons in the trigeminal nucleus caudalis receive extensive convergent inputs from both superficial and deep tissues, and can be modulated by nerve injuries, as well as by inflammatory conditions (Sessle and Greenwood, 1976; Sessle and Hu, 1991). There is some evidence (Ohrbach and Gale, 1989b; Hu *et al*., 1992; Reid *et al*., 1994) that neuroplastic changes and neuro-anatomic convergence of trigeminal noxious input may account, at least in part, for the local tenderness, spread, and referral of pain, often found in

temporomandibular joint dysfunction and related pain conditions. Brain stem mechanisms similar to those described above, can also account for the decrease of pressure pain thresholds of the jaw muscles found in the present study as the result of the periodontal noxious input. In the subjects investigated, sensitivity to mechanical pressure was also assessed at the finger site, which was selected as a control non-cranial site. Despite the fact that PPT was significantly lowered in the masseter and the temporalis muscles, no significant changes in PPT were found in the control site. Therefore, the hypothesis that higher brain centres are involved in mediating the sensitization of the jaw muscles found in the present study, is not supported.

Conclusions

In conclusion, the findings suggest that short-term occurrence of orthodontic pain is associated with motor and sensory changes of the masticatory muscles, represented by a decrease of the motor output and of the pressure pain thresholds of the jaw-closing muscles. These changes are probably mediated in the brain stem and may reflect a protective mechanism against further damage to an injured part of the masticatory system. Speculatively, sensory changes similar to those found in this study can help to explain the occurrence or the worsening of muscle pain occasionally reported by patients during orthodontic or other dental treatment. On the other hand, motor changes should be considered as a potential factor for loss of occlusal anchorage during orthodontic treatment (Melsen and Bosch, 1997). These hypotheses should be tested in long-term studies, and should address further research on sensory and motor changes of the human jaw muscles arising from heterotopic pain in the orofacial region.

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