Effect of a Jig on EMG Activity in Different Orofacial Pain Conditions

Celine Bodere, DDS, PhD[a]/Alain Woda, DDS, PhD[b]

Purpose: The bite stop (jig) is commonly used in clinical practice. It has been recommended as a simple means to routinely record or provide centric relation closure and, more recently, to reduce migraines and tension-type headaches. However, the reason for the jig effect has yet to be explained. This study tested the hypothesis that it works through a decrease in masticatory muscle activity. **Materials and Methods:** The effect of a jig placed on the maxillary anterior teeth was investigated by recording the electromyographic (EMG) activity of the superficial masseter and anterior temporal muscles at postural position and when swallowing on the jig. EMG recordings were obtained from 2 groups of pain patients (myofascial and neuropathic) and from 2 groups of pain-free patients (disc derangement and controls) unaware of the role of dental occlusion treatments. **Results:** EMG activity in postural position was higher in pain groups than in pain-free groups. The jig strongly but temporarily decreased the postural EMG activity for masseter muscles in all groups except for the neuropathic group and for temporal muscles in the myofascial group. The EMG activity when swallowing with the jig was reduced in control, disc derangement, and myofascial groups; however, EMG "hyperactivity" in the neuropathic pain group seemed to be locked. **Conclusions:** The decrease of postural EMG activity, especially in the myofascial group, was short lasting and cannot be considered as evidence to support the hypothesis of a long-term muscle relaxation jig effect. However, the results may uphold certain short-term clinical approaches. *Int J Prosthodont 2008;21:253–258.*

The anterior bite stop (jig) was initially proposed by Lucia.[1] It consists of either a leaf gauge or other anterior appliance. Its use has been proposed for occlusal adjustment, recording of the craniomandibular relationship,[2,3] treatment of some forms of temporomandibular disorders (TMD), and more recently, for migraine associated with tension-type headaches.[4] The underlying concept is to deprogram the memorized pattern of muscle activity by preventing tooth contacts at the time of swallowing. It is presumed to work through a relaxation effect, leading to a reduction in the muscle activity at postural position.[4,5]

[a]Associate Professor, Faculté d'Odontologie, Brest, France; Service d'Exploration Fonctionnelle Neurologique, Centre Hospitalier et Universitaire, Brest, France.

[b]Professor, Dental Faculty, University of Clermont-Ferrand, France.

Correspondence to: *Dr Celine Bodere, Faculté d'Odontologie, Rue Camille Desmoulins, 29200 Brest, France. Fax: 33 298 016 932. E-mail: cel.bodere@wanadoo.fr*

Many studies have tested this hypothesis by recording electromyographic (EMG) activity in the course of a bite on either a jig or a splint. All such studies showed less activity when clenching on a jig[6–8] or splint[9–15] than during an intercuspal bite. What is expected from jig application is a decrease in the postural EMG activity of elevator muscles triggered by the first contact on a jig. The lack of effect on postural activity reported in asymptomatic subjects[15] was as expected, since, in these subjects, the muscles were presumed not to be tense. Therefore, it is not known whether in symptomatic patients the application of a jig would decrease the postural EMG activity of masticatory muscles.

According to this study, the term "postural mandibular position" will be used to describe the maxillomandibular relation as it occurs in the "habitual mandibular position"[16] at rest in dorsal decubitus. A possible jig effect at the postural position was sought by recording the EMG activity of superficial masseter and anterior temporal muscles before, during, and after placing a jig. Four groups of subjects were investigated: 2 pain groups comprising patients with neuropathic or myofascial orofacial pain and 2 pain-free groups comprising patients with disc derangement and asymptomatic subjects.

Materials and Methods

This work was approved by the ethics committee of the University Hospital of Bretagne Occidentale, and all subjects gave informed consent after receiving a full explanation of the goals of the experiment. The 112 subjects in this study had already been part of a previous study.[17] The control subjects were students or staff members recruited within the dental or medical schools. Eighty outpatients were recruited from the orofacial pain clinic of the hospital. The patients were included in the groups at the first consultation at the pain clinic; they had not received occlusal treatment before and were unaware of the presumed jig effect. During the first visit, a single examiner (a dental surgeon) performed the clinical and physiologic examinations using a standardized form. This form encompassed palpation of the masticatory and neck muscles and temporomandibular joint (TMJ), assessment of spontaneous and triggered pain, and EMG recordings. The assessment of pain was performed with a visual analogue scale. Subjects with dental appliances, bruxism subjects, subjects taking drugs other than minor analgesics, subjects presenting with systemic pathologies, and subjects suffering from generalized diffuse muscle and/or articular pain were excluded from the experiment.

Patient Groups

Four groups of patients were investigated. The myofascial pain group consisted of subjects suffering from pain in the masticatory muscles (n = 33; age: 27.4 ± 6.9 years; 84% women). The criteria for inclusion were those of Okeson.[18] Patients with spontaneous or triggered TMJ pain were excluded. The disc derangement group consisted of patients (n = 27; age: 22.4 ± 7.9 years; 92% female) who did not complain either of spontaneous or triggered myofascial or TMJ pain. Disc derangements were with (n = 17) or without (n = 10) disc displacement reduction.[17] The neuropathic pain group consisted of patients (n = 20; age: 43.7 ± 15 years; 81% women) suffering from posttraumatic pain, postzosterian pain, trigeminal neuralgia, or idiopathic orofacial pain, the pathophysiology of which is now considered as neuropathic.[19–25] The control group consisted of healthy subjects free from signs and symptoms of TMD. The controls were matched with the myofascial and disc derangement group patients on the basis of sex and age (n = 32; age: 27.1 ± 4 years; 80% women).

The 4 groups were not matched for dental status, but all subjects had a full complement of natural anterior and premolar teeth and at least 1 molar pair. The relationship between the maxilla and mandible was judged unsatisfactory in 0%, 52%, 67%, and 26% of the subjects in the control, myofascial, disc derangement, and neuropathic groups, respectively. Criteria for unsatisfactory occlusion are described elsewhere.[16]

During the first visit, a jig was constructed as small as possible with acrylic resin to fit the maxillary central incisors,[5,26] make 1 median contact on its palatal side, and induce approximately 1 mm of posterior disocclusion during swallowing.

EMG Activity

A second operator (a neurologist) who was unaware of the groups' status as determined by the first investigator took EMG recordings 1 day after the first visit. Subjects were relaxed in dorsal decubitus on an examination table, with arms along the body and back raised at 60 degrees. Patients were asked to close their eyes. Surrounding noises were controlled. Bipolar surface electrodes were placed on the left and right superficial masseter and left and right anterior temporal muscles. Electrode impedances were lower than 5 kΩ. The raw EMG signal (Nicolet Vicking polygraph) was filtered and amplified (band pass: 20 Hz to 1 kHz, sensitivity: 10 to 100 μV, sampling frequency: 2 kHz).

The EMG activity was sampled for an 18-minute recording period. EMG was measured at postural position at 1 minute and 2 minutes. At 3 minutes, the subject was asked to perform an empty swallow without a jig. After deglutition, the signal was recorded at 3.5, 4, 5, 7, and 10 minutes, and the jig was placed on the maxillary incisors. At 11 minutes, another empty swallow was initiated on the jig. Recording at postural position then lasted for 7 more minutes with the jig in place, and the values were observed at 11.5, 12, 15, and 18 minutes (Figs 1 and 2). Each time, the mean EMG amplitude was picked up during periods of 10 seconds and measured peak to peak with an algorithm after manual positioning of the cursors. The validity of the measurements was assessed by calculating the coefficient of variation (CV = 0.07) and the between-subject reproducibility ($P < .001$).

Trace measurements and data analyses were performed in blind conditions. After testing the normality of the distributions, the mean EMG values in μV (± standard error of the mean) were compared and analyzed. General linear models for a 2-way analysis of variance (ANOVA) with repeated measures were used to test the group effects as the independent factor and the time effects as the repeated factor (Statistica software, Statsoft). A Student-Newman-Keuls test was carried out as a post hoc test. Paired or unpaired

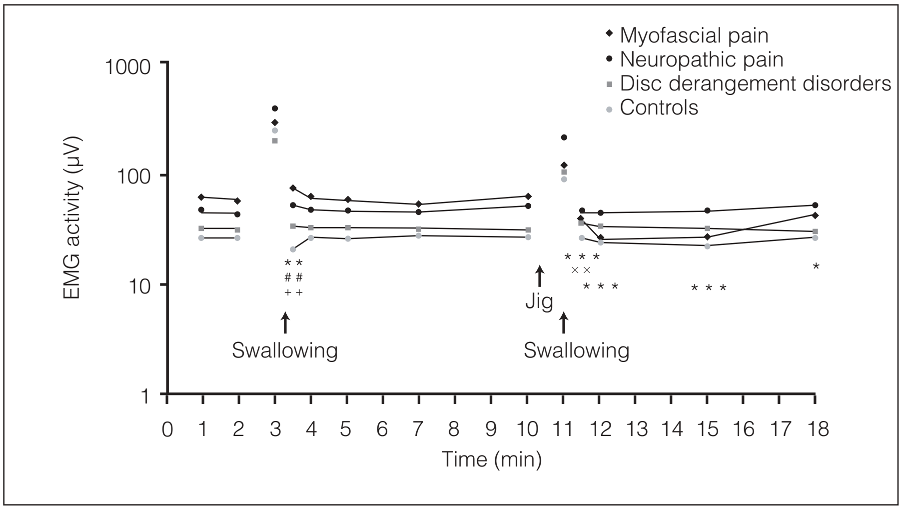

Fig 1 EMG activity of the anterior temporalis muscle. Paired Student *t* tests were used to compare EMG values for the 4 groups. For the first swallow, values at 3.5, 4, 5, and 7 minutes were compared to values at 2 minutes. For the second swallow, values at 11.5, 12, 15, and 18 minutes were compared to values at 10 minutes. $^{*}P < .05$, $^{**}P < .01$, $^{***}P < .001$ for myofascial group; $^{\#\#}P < .01$ for neuropathic group; $^{xx}P < .01$ for disc derangement group; $^{++}P > .01$ for control group.

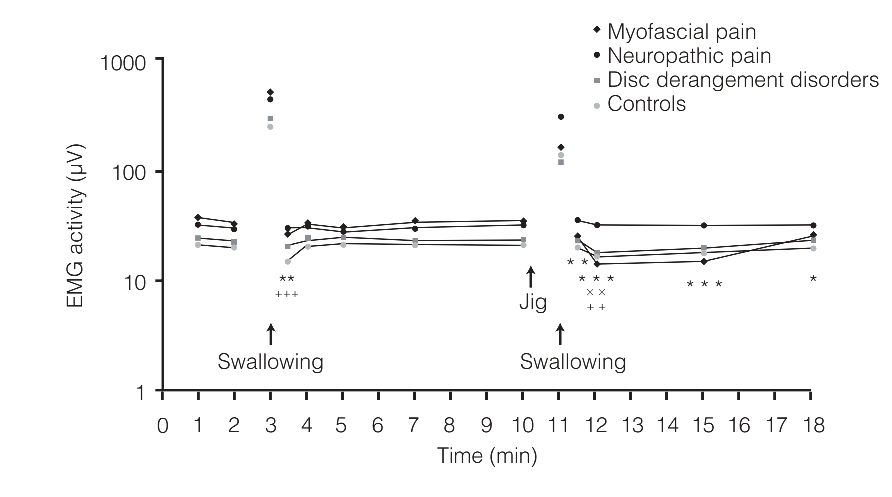

Fig 2 EMG activity of the superficial masseter muscle. Paired Student *t* tests were used to compare EMG values for the 4 groups. For the first swallow, values at 3.5, 4, 5, and 7 minutes were compared to values at 2 minutes. For the second swallow, values at 11.5, 12, 15, and 18 minutes were compared to values at 10 minutes. $^{*}P < .05$, $^{**}P < .01$, $^{***}P < .001$ for myofascial group; $^{xx}P < .01$ for disc derangement group; $^{++}P < .01$, $^{+++}P < .001$ for control group.

Student *t* tests were also used when necessary. The significance level was set at $P < .05$.

Results

There was no difference in EMG activity at postural position between painful and pain-free sides for both muscles in all groups (paired Student *t* test) at each recording time with or without a jig. Left and right side values were therefore pooled.

EMG Activity at Postural Position

Two-way ANOVA (with time as a repeated factor) applied to the temporal and masseter muscles indicated a significant difference between groups ($F_{3,108} = 17.9$ for temporal muscles and $F_{3,108} = 6.8$ for masseter muscles; $P < .001$) and between time points ($F_{10,1080} = 11.2$ for temporal muscles and $F_{10,1080} = 11.2$ for masseter muscles; $P < .001$) (Figs 1 and 2, Table 1). The Student-Newman-Keuls test indicated that both the temporal and masseter values of the 2 pain groups differed from the values of the 2 pain-free groups. The effect of time on the EMG values depended on the group under consideration ($F_{30,1080} = 10.0$ for temporal muscles and $F_{30,1080} = 5.28$ for masseter muscles; $P < .001$). The comparisons made before and after swallowing, with or without a jig, are shown in Figs 1 and 2. The myofascial group was the most influenced by the jig. Values of the myofascial group were not significantly higher than those of the neuropathic group without a jig at 1 minute and at 2, 3.5, 4,5, 7, and 10 minutes (*t* test) but were significantly lower at 12 and 15 minutes ($P < .01$ for temporal muscles and $P < .001$ for masseter muscles). Importantly, the myofascial group values were significantly higher than those of controls without a jig at 1 minutes and 2, 3.5, 4, 5, 7, and 10 minutes ($P < .001$ for temporal muscles and $P < .01$ for masseter muscles) although they reached the same level after the first contact on the jig.

Table 1 EMG Activity of the Muscles (μV ± SEM)

Group	Measurement time (min)												
	1.0	2.0	3.0	3.5	4.0	5.0	7.0	10.0	11.0	11.5	12.0	15.0	18.0
Temporal muscle													
MF	63.1 ± 6.6	55.5 ± 6.3	288.5 ± 43.4	63.3 ± 6.6	60.3 ± 5.7	55.5 ± 6.3	55.5 ± 6.3	56.9 ± 5.5	112.1 ± 9.9	40.5 ± 2.2	25.5 ± 2.2	27.9 ± 2.1	42.9 ± 3.9
NP	45.0 ± 5.9	40.8 ± 5.6	349.8 ± 65.4	47.4 ± 5.4	44.5 ± 5.1	44.1 ± 5.9	42.8 ± 5.9	44.5 ± 5.1	193.9 ± 40.8	44.3 ± 5.4	40.0 ± 5.4	42 ± 5.4	47.1 ± 5.4
DD	33.1 ± 3.1	31.5 ± 2.9	194.6 ± 23.5	32.1 ± 3.0	29.7 ± 2.5	31.6 ± 2.9	31.1 ± 2.5	29.7 ± 2.5	107.3 ± 13.2	38.6 ± 3.4	31.5 ± 3.4	30.5 ± 3.4	29.7 ± 3.4
CT	26.0 ± 1.0	25.1 ± 1.0	257.9 ± 30.2	20.5 ± 1.0	25.1 ± 1.0	25.0 ± 1.0	27.5 ± 1.0	26.0 ± 1.0	117.5 ± 11.0	25.5 ± 1.2	23.9 ± 0.9	21.5 ± 1.5	24.5 ± 1.4
Masseter muscle													
MF	40.2 ± 5.0	32.5 ± 4.2	475.6 ± 84.5	27.8 ± 3.6	33.5 ± 4.9	32.5 ± 5.0	33.7 ± 5.2	35.2 ± 4.2	161.9 ± 15.3	27.0 ± 1.5	16.1 ± 1.4	16.1 ± 1.4	26.1 ± 1.4
NP	33.5 ± 4.1	30.1 ± 3.7	437.0 ± 98.1	28.9 ± 3.1	32.6 ± 3.9	31.1 ± 3.8	30.9 ± 4.1	32.6 ± 3.1	303.4 ± 84.5	35.5 ± 5.3	33.3 ± 5.3	32.1 ± 5.3	31.8 ± 5.3
DD	24.8 ± 2.7	23.7 ± 2.8	291.7 ± 45.2	21.8 ± 2.3	24.2 ± 2.0	24.7 ± 2.5	23.5 ± 2.6	24.2 ± 2.0	132.2 ± 12.4	23.5 ± 2.3	18.2 ± 2.3	20.5 ± 2.3	24.7 ± 2.3
CT	22.5 ± 1.2	21.1 ± 1.0	257.9 ± 36.3	16.0 ± 1.0	21.0 ± 1.0	22.0 ± 1.0	21.8 ± 1.0	21.5 ± 1.0	138.9 ± 12.3	20.5 ± 1.8	17.0 ± 1.1	18.5 ± 1.1	20.5 ± 1.7

MF = myofascial group; NP = neuropathic group; DD = disc derangement group; CT = control group.

EMG Activity During Swallowing

Two-way ANOVA showed a significant group effect in the temporal and masseter muscles ($F_{3,214} = 5.5$ and $F_{216} = 6.4$; $P < .01$) and a significant jig effect [$F_{1,214} = 38.8$ and F_{216} 12.1; $P < .001$]. No interaction was found between these 2 factors for either temporal or masseter muscles (Figs 1 and 2, Table1). The neuropathic group differed from all other groups ($.001 < P < .05$; Student-Newman-Keuls), with higher EMG activity than the other groups when swallowing on the jig. There was no significant difference between the myofascial, disc derangement, and control groups.

Discussion

Jig Effects

Patients with chronic orofacial pain displayed higher tonic EMG activity.[17] In this study, the jig induced a transient decrease of the EMG activity, which was greatest in myofascial pain patients. This effect, observed for the first time, was remarkable since resting EMG was the highest in this group before jig placement and the lowest after it. However, this reduction did not continue, and EMG activity kept rising until the end of the recordings. The only study that recorded postural activity just after placement of an interocclusal appliance examined only asymptomatic subjects, and similarly to what was observed in the control group of this study, reported no significant effect.[15] Other studies observed the postponed action of an interocclusal appliance on EMG activity during a clenching task[10,11,13] and showed variable results, which are difficult to interpret. It is, however, interesting to note that Scopel et al[14] compared the EMG activity in a masticatory myofascial group against 2 control groups (occlusal disturbances and asymptomatic healthy subjects) after placement of a splint for several weeks. In the myofascial group, the EMG activity reached the same values as in the control group after splint therapy lasting 4 to 9 weeks until total pain relief. It must be noted, however, that the postponed action induced by a long splint therapy is probably not the result of the same mechanisms as an immediate jig effect.

The neuropathic pain group, which displayed a relatively high resting activity, showed no change after jig placement. Although likely of central origin in both pain groups,[17] EMG "hyperactivity" seen in neuropathic pain patients seems to be centrally locked with no possibility of modulation of the central motor program, as opposed to the jig-induced modulation observed in myofascial pain patients. These results support the hypothesis that the pathophysiologies of the 2 pain groups differ in some respects. In addition, with the jig in place, EMG activity was reduced for both muscles and in all groups except for the neuropathic group. Values in the disc derangement group were similar to those of controls, suggesting there was almost no involvement of masticatory muscles in this pain-free group.

Whereas most of the methods were different, the results are not at odds with those described in the sparse literature on the topic. The only EMG studies that described the jig effect were limited to clenching and bruxism tasks.[6–8] Other researchers used a hard splint during voluntary contraction in healthy subjects.[9,12,15] The placement of each occlusal appliance provoked a significant decrease in elevator muscle activity during clenching. A recent short-term study recorded the EMG activity in TMD patients self-reporting tooth grinding during sleep.[27] The authors compared the effect of a so-called patented jig NTI-Tension Suppression System to a flat occlusal splint on the EMG events in the course of sleep. They observed a reduction of the EMG activity during the bruxism periods with the jig only. In accordance with these previous works, the present study showed that EMG activity during swallowing with the jig in place was reduced in all groups except for the neuropathic group compared to swallowing without the jig.

Clinical Issues

The results showed that the jig device, applied as in this study, influenced EMG activity to a different extent in the 4 groups. The transient decrease in EMG postural activity in myofascial pain patients cannot be considered as evidence to support the hypothesis of a long-term muscle relaxation jig effect. However, this transient EMG decrease and the reduction of EMG activity when swallowing with a jig could support certain short-lasting clinical approaches, such as the recording of centric relation closures and the reduction of a premature occlusal contact.

Limitations of the Study

This study has several limitations. First, EMG recordings of masticatory muscle activity at postural position depend on many factors, including the position of the electrodes and the activity of nearby muscles.[28] Since the coefficient of variation found in the recordings of EMG activity at postural position in the controls was small,[17] these factors could not have increased the variability during the recordings. However, contamination from the facial muscles cannot be totally excluded, at least for the temporal muscle.

Further, the neuropathic group, and to a lesser extent the disc derangement group, were not totally homogeneous. The neuropathic group was also not matched in age or in dental status with the controls because of the low prevalence of most pathologic neuropathic conditions under the age of 35.[29]

It must also be noted that this study did not use a placebo jig (eg, with no palatal side). This would have allowed for a more powerful crossover design. Therefore, the differences seen in this study before and after jig placement could be the result of a nonspecific effect. This prohibits any interpretations about the mechanism of the jig effect. However, the observed differences between groups were controlled and are thus considered as valuable data. In addition, it must be noted that the reduction of muscle activity found in patients cannot be the consequence of a learned "avoidance behavior" because patients were naive about occlusal treatment.

The relationship between the maxilla and mandible was significantly different between groups; however, it is unlikely that this had a strong impact on EMG or pain since no association between these factors could be seen across the 4 groups.

Another major limitation of this study is the risk of misinterpretation of the results. This is especially important to minimize the outcomes in regard to non-scientific dental groups that are promoting the application of various devices to treat TMD patients. The evidence suggests that the jig effect is very brief in postural position, lasting only a few minutes, and cannot be considered favorable for treating patients with TMD or neuropathic pain. The only evidence that this study and others[6–9,12,15,27] confirmed is that the jig reduced the EMG activity of masticatory muscles during swallowing, clenching, and bruxism tasks. In accordance with these thoughts, the use of a stabilization splint with TMD patients showed better results than the use of a jig. In the study by Magnusson et al,[30] which did not use a placebo jig, the jig effect was consistent with a placebo effect.

Finally, 2 side effects of a jig worn regularly during sleep over a period of several weeks must be pointed out: *(1)* the posterior disocclusion could aggravate a joint dysfunction because of the absence of posterior contact, and *(2)* the lateral dental sectors in disocclusion could grow out, causing a secondary anterior open bite.

Conclusion

The use of a jig strongly but temporarily decreased the masticatory muscle tone in myofascial pain patients. In addition, using a jig decreased the masticatory muscle activity during swallowing in control, disc derangement, and myofascial pain groups but not in the neuropathic pain group. These results support the common use of the jig in dental clinical practice as a tool for the recording of centric relation closures or for occlusal adjustment; however, the jig cannot be considered favorable for treating patients with neuropathic pain or temporomandibular disorders.

Acknowledgments

The authors are grateful to Prof Abjean, who inspired this work, and to Ms C. John for the language correction. We also thank all of the staff of the Department of Neurological Exploration of the University Hospital of Brest, especially Dr Tea. We are obliged to the head of the department, Dr L'Heveder, for supporting this work.

References

1. Lucia VO. Jig-method [in German]. Quintessenz Zahntech 1991;17:701–714.
2. Carroll WJ, Woelfel JB, Huffman RW. Simple application of anterior jig or leaf gauge in routine clinical practice. J Prosthet Dent 1988;59:611–617.
3. Urstein M, Fitzig S, Moskona D, Cardash HS. A clinical evaluation of materials used in registering interjaw relationships. J Prosthet Dent 1991;65:372–377.
4. Shankland WE. Nociceptive trigeminal inhibition–tension suppression system: A method of preventing migraine and tension headaches. Compend Contin Educ Dent 2002;23:105–108.
5. Karl PJ, Foley TF. The use of a deprogramming appliance to obtain centric relation records. Angle Orthod 1999;69:117–124.
6. Becker I, Tarantola G, Zambrano J, Spitzer S, Oquendo D. Effect of a prefabricated anterior bite stop on electromyographic activity of masticatory muscles. J Prosthet Dent 1999;82:22–26.
7. Hickman DM, Cramer R, Stauber WT. The effect of four jaw relations on electromyographic activity in human masticatory muscles. Arch Oral Biol 1993;38:261–264.
8. Santosa RE, Azizi M, Whittle T, Wanigaratrne K, Klineberg IJ. The influence of the leaf gauge and anterior jig on jaw muscle electromyography and condylar head displacement: A pilot study. Aust Dent J 2006;51:33–41.
9. Al Quran FA, Lyons MF. The immediate effect of hard and soft splints on the EMG activity of the masseter and temporalis muscles. J Oral Rehabil 1999;26:559–563.
10. Greco PM, Vanarsdall RL Jr, Levrini M, Read R. An evaluation of anterior temporal and masseter muscle activity in appliance therapy. Angle Orthod 1999;69:141–146.
11. Hersek N, Uzun G, Cindas A Canay S, Kutsal YG. Effect of anterior repositioning splints on the electromyographic activities of masseter and anterior temporalis muscles. Cranio 1998;16:11–16.
12. Jimenez ID. Electromyography of masticatory muscles in three jaw registration positions. Am J Orthod Dentofacial Orthop 1989; 95:282–288.
13. Landulpho AB, E Silva WA, E Silva FA, Vitti M. The effect of the occlusal splints on the treatment of a temporomandibular disorders—A computerized electromyographic study of masseter an anterior temporalis muscles. Electromyogr Clin Neurophysiol 2002;3: 187–191.
14. Scopel V, Alves da Costa GS, Urias D. An electromyographic study of masseter and anterior temporalis muscles in extra-articular myogenous TMJ pain patients compared to an asymptomatic and normal population. Cranio 2005;23:194–203.
15. Sgobbi de Faria CR, Berzin F. Electromyography study of the temporal, masseter and suprahyoid muscles in the mandibular rest position. J Oral Rehabil 1998;25:776–780.
16. Woda A, Pionchon P, Palla S. Regulation of mandibular postures: Mechanisms and clinical implications. Crit Rev Oral Biol Med 2001;12:166–178.
17. Bodere C, Tea SH, Giroux-Metges MA, Woda A. Activity of masticatory muscles in subjects with different orofacial pain conditions. Pain 2005;116:33–41.
18. Okeson JP. Orofacial Pain: Guidelines for Assessment, Diagnosis, and Management. Chicago: Quintessence, 1996.
19. Gremeau-Richard C, Woda A, Navez ML, et al. Topical clonazepam in stomatodynia: A randomized placebo-controlled study. Pain 2004;108:51–57.
20. Hagelberg N, Forssell H, Aalto S, et al. Altered dopamine D2 receptor binding in atypical facial pain. Pain 2003;106:43–48.
21. Hagelberg N, Forssell H, Rinne JO, et al. Striatal dopamine D1 and D2 receptors in burning mouth syndrome. Pain 2003;101:149–154.
22. Lauria G. Small fibre neuropathies. Curr Opin Neurol 2005; 18:591–597.
23. Scala A, Checchi L, Montevecchi M, Marini I, Giamberardino MA. Update on burning mouth syndrome: Overview and patient management. Crit Rev Oral Biol Med 2003;14:275–291.
24. Woda A, Pionchon P. A unified concept of idiopathic orofacial pain: Pathophysiologic features. J Orofac Pain 2000;14:196–212.
25. Woda A, Tubert-Jeannin S, Bouhassira D, et al. Towards a new taxonomy of idiopathic orofacial pain. Pain 2005;116:396–406.
26 Abjean J, Bodin C. Lateral condylar movement: Recording and transfer from the physiologic closing path [in French]. Rev Odontostomatol (Paris) 1989;18:111–127.
27. Baad-Hansen L, Jadidi F, Castrillon E, Thomsen PB, Svensson P. Effect of a nociceptive trigeminal inhibitory splint on electromyographic activity in jaw closing muscles during sleep. J Oral Rehabil 2007;34:105–111.
28. Lund JP, Widmer CG. Evaluation of the use of surface electromyography in the diagnosis, documentation, and treatment of dental patients. J Craniomandib Disord 1989;3:125–137.
29. LeResche L. Epidemiology of orofacial pain. In: Lund JP, Lavigne G, Dubner R, Sessle BJ (eds). Orofacial Pain. From Basic Science to Clinical Management. Chicago: Quintessence, 2001:15–25.
30. Magnusson T, Adiels AM, Nilsson HL, Helkimo M. Treatment effect on signs and symptoms of temporomandibular disorders—Comparison between stabilisation splint and a new type of splint (NTI). Swed Dent J 2004;28:11–20.