Effect of a nociceptive trigeminal inhibitory splint on electromyographic activity in jaw closing muscles during sleep

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SUMMARY The nociceptive trigeminal inhibitory (NTI) splint has been claimed to decrease the electromyographic (EMG) activity of jaw-closing muscles and relieve symptoms of various types of temporomandibular disorders (TMD) and bruxism. The present study was designed to address the question about EMG-changes during sleep. Ten patients (age: 23–39 years) with a self-report of tooth-grinding during sleep were recruited. Patients were examined at baseline and after each treatment period with the use of the Research Diagnostic Criteria for TMD. A portable EMG-device was used to record EMG-activity from the masseter muscle during sleep. The patients received two 2-week splint treatments in a randomized cross-over fashion; an NTI splint and a standard flat occlusal splint (OS). EMG data were analysed according to published criteria. Using a 10% of maximum clenching EMG-activity cut-off threshold to determine the number of EMG-events h^{-1} of sleep, the NTI splint was associated with a significant reduction ($9{\cdot}2 \pm 3{\cdot}2$ events h^{-1}) compared with baseline EMG ($19{\cdot}3 \pm 4{\cdot}0$; ANOVA: $P = 0{\cdot}004$, Tukey *post hoc*: $P = 0{\cdot}006$), whereas there were no differences between the OS ($16{\cdot}2 \pm 4{\cdot}7$) and baseline EMG ($19{\cdot}2 \pm 4{\cdot}1$; $P = 0{\cdot}716$). There were no effects of either NTI or OS on clinical outcome measures (ANOVAS: $P > 0{\cdot}194$). This short-term study indicated a strong inhibitory effect on EMG-activity in jaw closing muscles during sleep of the NTI, but not the OS. However, the EMG-activity was not directly related to clinical outcome. Further studies will be needed to determine long-term effects and possible side effects of the NTI splint.

KEYWORDS: temporomandibular disorders, electromyography, bruxism

Introduction

Oral splints have often caused an emotional debate about their effectiveness on signs and symptoms from the temporomandibular system. Recent systematic reviews conclude that oral splints may be of some benefit but that the number needed to treat (NNT), i.e. the number of patients who needs to receive a certain treatment in order for one patient to achieve a 50% pain relief or more (1), is in the range of 4–6. However, the exact mechanisms of action are not well understood (2).

Several studies have examined the electromyographic (EMG) activity in masticatory muscles in response to occlusal splint treatment (3–6). Generally, some patients seem to increase their EMG activity recorded during the night, whereas others decrease the EMG activity. It is a common belief that the patho-physiological model of temporomandibular disorder (TMD) pain involves muscle hyperactivity, although this has never been proven directly (for a review see Ref. 7). Recently, a new type of oral splint has been developed with some resemblance to previous splint designs covering only the incisors and the new member of the oral splint family has been termed 'nociceptive trigeminal inhibitory tension suppression system' (NTI). The proposed mechanism of action is that overloading of the periodontal ligament will cause

activation of nociceptive afferents, which by reflex pathways will inhibit the jaw-closing muscles and subsequently reduce the muscle tension (8). The NTI splint has gained substantial popularity amongst clinicians treating TMD pain, although the evidence in favour is limited. For example, a recent study was unable to demonstrate a significant difference between the NTI and a conventional oral splint in treatment efficacy (9).

The aim of the present investigator-blinded randomized cross-over study was to test the effect of the NTI splint compared with a conventional flat occlusal stabilization splint (OS) on the EMG activity during sleep. Secondly, to test if changes in EMG activity would be associated with short-term changes in the signs and symptoms of TMD pain. The specific hypothesis was that the NTI splint would be associated with a decrease in EMG activity, which would be related to an improvement in TMD pain problems.

Materials and methods

Participants

Ten patients (three men and seven women, aged: 23–39 years) with a self-report of tooth-grinding during sleep, supported by complaints from bed-partners, reports of muscle soreness on awakening and signs of tooth wear were recruited (10). Patients were examined by a blinded investigator at baseline and after 1 week of treatment with both splint-types with the use of the RDC/TMD form (11), and the patients were asked to rate their pain (if present) on a 0–10 visual analogue scale (VAS). From the RDC/TMD examination, data on number of painful muscles to palpation and maximum unassisted jaw-opening capacity were retrieved in addition to characteristic pain intensity (CPI) (11). Patients using any other medication than mild analgesics were excluded from the study. The patients scored their spontaneous pain and use of mild analgesics every day. The study was approved by the local ethics committee in Aarhus County and written informed consent was obtained from all participants before entering the study and the study was performed according to the Helsinki Declaration.

Study design

The study was performed as an investigator-blinded randomized, controlled cross-over study with EMG-recordings performed during sleep in two treatment periods, one with an NTI splint and one with a standard occlusal splint. For each participant, the study lasted 7 or 8 weeks, including a 'wash-out' period. After 1 week of baseline EMG-recording from the masseter muscles during sleep, the patients received either a NTI-splint providing occlusion only on the front teeth or a standard flat OS (12), in randomized order. The patients wore the splint during sleep for 1 week without EMG-recording followed by 1 week with the splint and EMG recording (NTI + EMG/OS + EMG). After 1–2 weeks of 'wash-out', another baseline EMG recording without splint was done followed by 2 weeks with the other type of splint, one without and one with EMG recordings. A total of 4 weeks EMG data were obtained for each patient and subsequently analysed according to published criteria (13).

Sleep EMG recordings

A portable EMG device* was used to record the EMG activity from the masseter muscle during sleep. The device consisted of a display, two recording channels, a keyboard, wires for connection of electrodes, and cables for data transmission to a home computer. Surface Ag/AgCl electrodes with a diameter of 2·25 cm were used. The connectivity between electrodes and skin was optimized by the use of an electrode gel. The EMG signals were recorded, amplified, filtered (filter 20–500 Hz), and rectified with analogue signal processing (sampling rate: 2 kHz, stored in 500 ms bins). The data were stored in the memory of the device and calculations of the EMG area under the curve (AUC) and root mean square value for each time interval of 500 ms were performed automatically. The data were transferred to the home computer for analyses of the EMG activities during sleep. A customized software system (SW)* installed on the patient's home computer processed all the EMG data from each night systematically. The EMG data were sent by e-mail to the research team every morning in order to further analyse and store the data and to eliminate possible problems with the device. All data were checked for problems and errors due to bad recordings. If a problem was found, the patient was contacted in order to solve the problem and ask them to add one night to the phase in order to get sufficient data in each phase. The number of EMG-events h^{-1} sleep,

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EMG duration h^{-1} sleep and EMG AUC h^{-1} sleep were obtained from the all-night EMG-recordings using the Jaws version 1.51[†] software for three levels of cut-off thresholds; 3%, 10% and 20% of maximum voluntary contraction (MVC) for each night. The first night was used for adaptation to the device and to observe, whether the patient exhibited any bruxism-like activity. The first and last 15 min (falling asleep and waking up) of each night's recordings were excluded from analysis.

NTI and occlusal splints

The two splints were made for the patients by a different investigator than the one who performed the RDC/TMD examinations. The RDC/TMD examinations were performed on the day of delivering the first splint to the patient (baseline) and after 2 weeks of use of both splints (NTI and OS). The NTI-splint was made according to the guidelines supplied by the manufacturer. In brief, a pre-fabricated splint was used and individually fitted to cover the upper incisors and to create a one-point contact with the lower incisors. The OS were standard flat occlusal stabilization splints with many evenly distributed occlusal contacts and group contact on laterotrusion and protrusion (12).

Statistics

The data are presented as mean value ± s.e.m. The EMG-derived data were analysed with a two-way analysis of variance (ANOVA) for repeated measures. One factor was *treatment* (baseline 1, NTI, baseline 2, OS); the other factor was *EMG threshold level* (3%, 10% and 20% of MVC). Additional ANOVAS looking at *time* (consecutive EMG recordings in each phase) were performed in order to evaluate changes in EMG over time within each phase. The VAS pain scores, the CPI, the number of painful muscles to palpation, and maximum unassisted jaw-opening-capacity were analysed with one-way ANOVAS. Tukey *post hoc* tests were used to adjust for multiple comparisons. The clinical measures were tested for associations with the EMG data at baseline with the use of Pearson product moment correlations. Significance was accepted at a 5% level.

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Results

Patient characteristics at baseline

Five of the 10 included bruxism patients perceived pain at baseline. The mean (±s.e.m.) spontaneous pain at baseline was $1 \cdot 6 \pm 0 \cdot 6$ on a 0–10 VAS. Four of 10 patients received a diagnosis of myofascial TMD and one patient had a unilateral disc displacement with reduction according to the RDC/TMD (11). The mean (±s.e.m.) maximum unassisted jaw opening capacity of the patients were $50 \cdot 2 \pm 2 \cdot 2$ mm.

EMG outcome

The number of EMG events h^{-1} sleep, EMG duration h^{-1} sleep and EMG area h^{-1} sleep were all significantly influenced by EMG threshold level (ANO-VAS: $F > 21 \cdot 816$; $P < 0 \cdot 001$) with highest values detected at the 3% level (Tukey: $P < 0 \cdot 001$) (Fig. 1a–c).

The number of EMG events h^{-1} sleep, EMG duration h^{-1} sleep and EMG AUC h^{-1} sleep were also significantly dependent on treatment (ANOVAS: $F > 6 \cdot 081$; $P < 0 \cdot 003$) and with significant interactions between treatment and EMG threshold level (ANOVAS: $F > 2 \cdot 783$; $P < 0 \cdot 02$). Post hoc tests demonstrated significant decreases during the use of the NTI splint when compared with baseline (base 1: Tukey: $P < 0 \cdot 05$) at all levels of EMG threshold (Fig. 1a and b) except for EMG area h^{-1} at the 10% and 20% level (Fig. 1c). There were no differences between the baseline EMG values for any of the EMG measures (base 1 versus base 2: Tukey: $P > 0 \cdot 281$) and the OS did not differ from the baseline values (base 2: Tukey: $P > 0 \cdot 237$).

The additional ANOVAS did not indicate any significant changes over time in any of the phases (ANOVA: $F < 0 \cdot 479$; $P > 0 \cdot 633$).

Clinical outcome

No differences between treatments (baseline, NTI, OS) were detected in VAS pain scores (range at baseline 0–5 on a 0–10 VAS) (ANOVA: $F = 0 \cdot 065$; $P = 0 \cdot 937$), CPI (ANOVA: $F = 1 \cdot 836$; $P = 0 \cdot 194$), number of painful muscles to palpation (ANOVA: $F = 0 \cdot 035$; $P = 0 \cdot 996$), or maximum unassisted jaw-opening capacity (ANOVA: $F = 1 \cdot 316$; $P = 0 \cdot 293$) (Fig. 2).
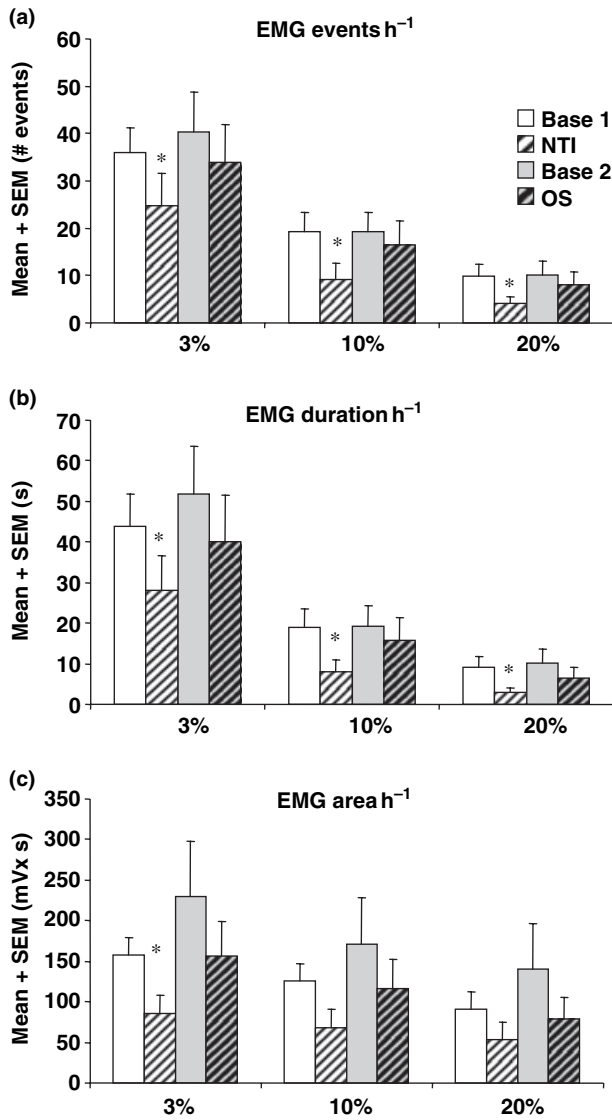
Fig. 1. The mean (+s.e.m.) electromyographic (EMG) activity in masseter muscles during different phases of the study. Base 1 (first baseline period), Base 2, NTI (period with treatment with nociceptive trigeminal inhibitory), OS (period with treatment with occlusal splint). (a) The number of events h^{-1} of sleep with EMG levels exceeding three different cut-off levels (3%, 10% and 20% of maximum voluntary contraction, MVC). (b) The duration (s h^{-1} of sleep) of EMG levels exceeding the three cut-off levels. (c) The area under the EMG curve exceeding the three cut-off levels. *$P < 0.045$. $n = 10$.

Correlation EMG and clinical data

No significant correlations were present between EMG data (events h^{-1}; 3%, 10% and 20% cut-off) and VAS pain, CPI, number of painful muscles to palpation, or maximum unassisted jaw-opening capacity (Pearson's correlation: $P > 0.197$).
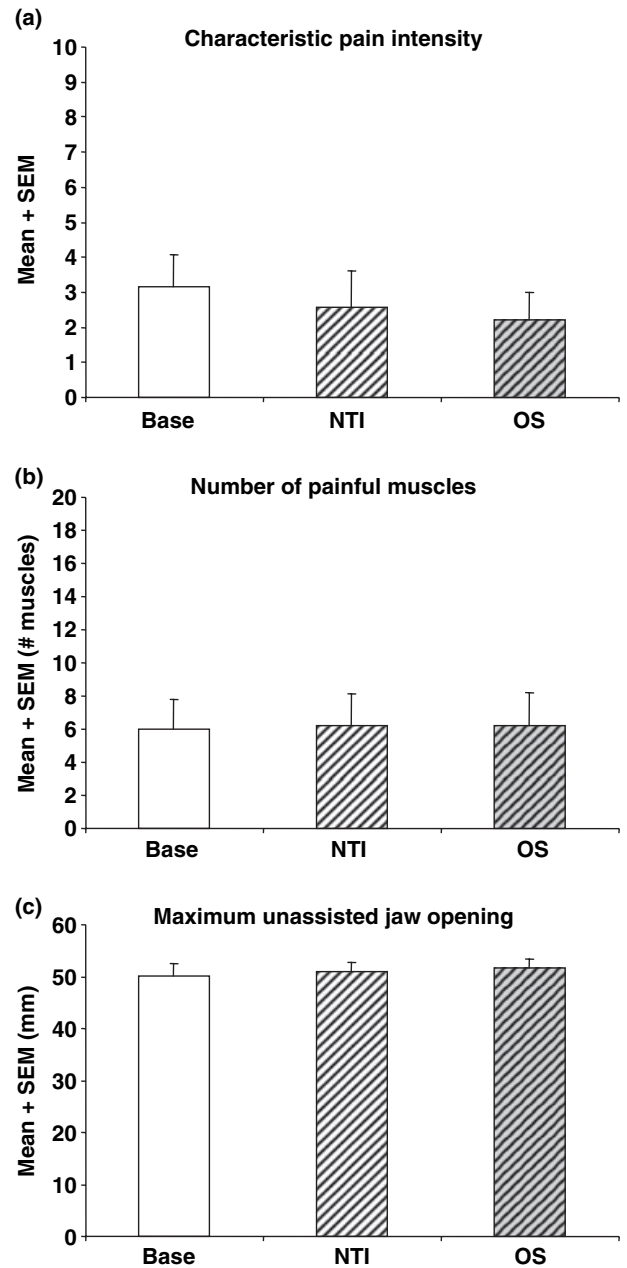

Fig. 2. The mean (+s.e.m.) of the clinical outcome measures during three phases: (a) characteristic pain intensity, (b) number of muscles painful to palpation, (c) maximum unassisted jaw opening. Base (Baseline), NTI (nociceptive trigeminal inhibitory splint treatment phase) and OS (treatment with occlusal splint). No differences between treatments or between baseline and splint treatment phases were detected. $n = 10$. As the two baseline periods are not different, the means of the two periods are shown.

Discussion

The major finding in this study was the robust reduction in EMG activity in the masseter muscles during

treatment with the NTI splint. Conversely, in this study the OS did not cause a significant reduction in EMG activity. The reduction in masseter muscle-activity during NTI treatment was not associated with a short-term reduction in TMD signs or symptoms of the patients.

Methodological issues

Only recently, the effects on EMG-activity of occlusal splint treatment during more than just a few nights of sleep have been examined (14). The effect of a stabilization splint and a palatal splint was examined using a portable EMG system for five periods of three nights at different stages of a treatment period (14). They found that immediately after insertion of the splints, the EMG activity was reduced for both splint types, but not later in the treatment period (14). The measurement of EMG activity during weeks instead of two or three nights reduces the effect of random variation in bruxism activity. Still, the possible effect of a splint after 2 weeks of treatment, like in the present study, must be considered a relatively short-term effect.

The lack of reduction in EMG activity during treatment with the OS in the present study should be taken with caution due to the small sample size, although the cross-over design of the study strengthens the power by allowing paired comparisons between treatments. A *post hoc* calculation has been performed, which indicates that a sample size of 24 would have been needed to demonstrate a difference between OS and baseline. Furthermore, the limited amount of symptoms at baseline may also have hampered the attempt to demonstrate possible short-term reductions in clinical symptoms during treatment with NTI or OS. Only five patients experienced pain at baseline, as they were recruited based on self-reported criteria for bruxism and not pain. However, this may represent a majority of patients with bruxism habits as pain and bruxism are not strongly linked (15). Furthermore, the limited sample size and the fairly short treatment periods (2 weeks) with each splint-type may add to the difficulty of demonstration of a potential symptom-relief. However, the primary aim of this study was to study possible reductions in EMG activity during treatment with the two different types of splints and only secondarily to evaluate short-term changes in clinical symptoms. Hence, further studies will be needed to examine the relationship between possible long-term

reductions in EMG activity and TMD signs and symptoms.

A concern could be raised about the ability to distinguish between clenching activity and EMG activity caused by facial mimics, i.e. the specificity of the EMG analysis to detect clenching events (16). However, because the differences in EMG events h^{-1} between treatments (baseline, NTI, OS) were statistically significant for all cut-off thresholds (3%, 10% and 20%), it can be argued that facial mimics may not markedly have contaminated the results. In most of the recently published related papers, a cut-off threshold of 10% is used (6). Of course, polysomnographic studies with video- and audio-recordings could have eliminated non-bruxism-related EMG activity from the analysis but we argue that ambulatory recordings inside the patients' home environment has some advantages over the sleep laboratory setup with regard to costs and maybe even sleep quality.

EMG changes and effects of oral splints

The ability of an OS to reduce EMG activity has been examined in several studies with conflicting results (3, 5, 6, 17), although the most frequent finding is that OS causes such a reduction. In the present study, the NTI splint alone caused a significant reduction in EMG activity during sleep, although a trend for reduction was found for the OS. This may be due to a higher variability in EMG responses to treatment with OS compared with the NTI. However, the important question that remains to be answered is whether or not a reduction in masticatory muscle EMG activity is directly related to reductions in clinical pain or discomfort. Two main theories regarding this subject prevail: the vicious cycle model (18) and the pain-adaptation model (19, 20). The vicious cycle model, in short, entails that muscle pain causes muscle hyperactivity, which in turn causes more muscle pain and so on (18). The vicious cycle model gives a clear rationale for treatment of myofacial TMD with a splint which reduces muscle activity, thereby 'breaking' the vicious cycle. However, this model was questioned by Lund *et al.* (19), who instead proposed the pain-adaptation model and suggested that muscle pain actually reduces the ability of the muscle to contract, i.e. the painful muscle decreases its function in order to avoid further injury and pain. The pain-adaptation model is supported by several experimental muscle pain studies (7) and

does not lend support to inhibition of muscle activity as a primary treatment goal for pain relief. Therefore, the possible pain- or discomfort-relieving effect of an NTI or OS as has been demonstrated in some but not all studies (8, 9) suggests a possible involvement of other mechanisms, as no studies have so far demonstrated a direct relationship between reduction in EMG activity and changes in TMD symptoms. The two published studies, which have compared the clinical outcome of treatment with NTI splints and standard OS have not found the effects of the NTI to be superior to the OS (8, 9). On the contrary, a tendency towards a poorer clinical effect of the NTI splint has been found and furthermore, a concern about a risk for changes in occlusion after treatment with an NTI splint has been raised (8).

Possible harmful effects are important to consider when making an evidence-based treatment strategy. The evidence-based evaluation of the clinical efficacy of a given treatment often involves calculation of the NNT (1). The NNT for treatment of TMD with OS has been reported to be between 4 and 6 (2). This means that between 4 and 6 patients need to be treated for one patient to receive a 50% pain relief. No NNT estimates are yet available for the NTI splint. However, the important question arises, whether the OS or the NTI splint may also be potentially harmful? In one study, 10 patients were treated with an NTI splint, and one of these patients was reported to have an impaired occlusion at the 6-month follow-up (8). In the present study, no systematic data collection on comfort, discomfort, snoring and sleep quality related to wearing the two splints were collected. Some of the patients spontaneously reported the OS to be more comfortable and some preferred the NTI splint. However, there is a possibility that the use of a splint may, for example, improve airway patency during sleep due to a posterior rotation of the mandible. Thus, in future randomized controlled trials, it is essential to collect data on pain, discomfort, sleep quality etc. and to calculate both the NNT and the number needed to harm in order for clinicians to be able to perform evidence-based decisions.

Conclusion

In conclusion, a strong and lasting inhibition of EMG activity in masseter muscles during sleep was caused by wearing the NTI splint but not the OS. However, this was not directly related to the short-term clinical outcome measures. Further studies with systematic examination of long-term effects on TMD symptoms and potential harmful effects are needed.

Disclaimer

The authors have no financial or commercial interest in the NTI splint.

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