Effect of Contingent Electrical Stimulation on Masticatory Muscle Activity and Pain in Patients with a Myofascial Temporomandibular Disorder and Sleep Bruxism

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Aims: *To determine whether an intervention reduces oromotor activity and masticatory muscle pain in myofascial temporomandibular disorder (M/TMD) patients with high levels of masticatory muscle activity associated with sleep bruxism.* ***Methods:*** *Fourteen women with M/TMD and prior polysomnographic evidence consistent with sleep bruxism participated in a 10-week single-group pre-test/post-test mechanistic clinical trial. A 2-week period of baseline monitoring of individually biocalibrated electromyographic (EMG) events associated with sleep bruxism was followed by 6 weeks of EMG-event-contingent treatment via an innocuous electrical pulse to the skin overlying the temporalis muscle. Treatment was discontinued during 2-week follow-up monitoring. Each night before sleep, subjects recorded their average daily pain.* ***Results:*** *Mixed-model analysis of variance showed a reliable reduction of EMG events during contingent stimulation treatment periods, but frequency of EMG events returned to baseline levels during follow-up (linear term,* P = *.002; quadratic term,* P = *.001). In contrast, nightly pain reports failed to show any systematic changes during treatment (linear and quadratic trends, both* P > *.10).* ***Conclusion:*** *Spontaneous pain severity and nighttime oromotor activity vary independently over nights, even in M/TMD patients selected for relatively high levels of both characteristics.* J OROFAC PAIN 2013;27:21–31. doi: 10.11607/jop.1029

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The most prevalent subtype of temporomandibular disorders (TMD) is myofascial TMD (M/TMD), which is characterized by pain and dysfunction in the masticatory muscles.[1] Bruxism, a parafunctional activity characterized by grinding or clenching of the teeth, has been implicated in the onset and persistence of TMD by more than 50 years of theoretical[2,3] and empirical (see reviews[4–6]) studies. Bruxism may occur during waking or sleep periods.

Despite the widespread belief about the pathogenic role of bruxism and other parafunctional activity among dentists[7–9] and TMD patients,[10] data increasingly indicate that bruxism is unlikely to be either a necessary or sufficient condition for M/TMD. As the methods for assessing sleep bruxism have become more objective and less potentially biased, research studies are less likely to find a relationship to M/TMD; studies using self-report methods are most likely to conclude that there are elevated rates of sleep bruxism in TMD patients.[4,6] For example, a recent large case-control study found that patients with M/TMD were no more likely than controls to exhibit sleep bruxism during laboratory polysomnographic (PSG) studies.[11]

A similarly small proportion of both M/TMD cases and controls (ie, 10% and 11%, respectively) met research diagnostic criteria for sleep bruxism,[12] and cases were actually less likely than controls (60% vs 78%) to demonstrate multiple grinding noises during sleep. Thus, masticatory muscle hyperactivity, as reflected by bruxism and associated oromotor activity measured during sleep, is likely to be a poor overall explanation for persistent M/TMD pain.

Nevertheless, heterogeneity in mechanisms underlying TMD is commonly acknowledged and is inherent in the newest models of TMD pain onset and persistence.[13,14] The possibility remains that masticatory muscle hyperactivity is a pain maintenance factor in a subset of individuals with M/TMD. If high levels of oromotor activity are a contributing cause of pain in a subset of patients with both M/TMD and high levels of oromotor activity associated with bruxism, these patients should experience reduced masticatory muscle pain when exposed to an intervention that reduces oromotor hyperactivity.

To conduct a mechanistic clinical trial exploring relationships between oromotor hyperactivity and masticatory muscle pain, an intervention that reliably reduces hyperactivity is needed. As reviewed by others,[15,16] several promising treatments such as clonidine and mandibular-advancement devices have been associated with a number of adverse events, making them unsuitable for use with most TMD patients. More recently, Jadidi et al[17] reported a promising treatment for reduction of masticatory muscle motor hyperactivity. The Grindcare (Medotech A/S) device monitors temporalis muscle activity, identifying individually biocalibrated electromyographic (EMG) events associated with bruxism. During a treatment mode, it delivers an innocuous electrical pulse to the skin overlying the temporalis muscle contingent upon EMG activity exceeding individually biocalibrated levels associated with bruxism. In one study of 14 individuals with sleep bruxism,[17] treatment with Grindcare led to a 40% to 50% reduction in EMG events. Based on these preliminary findings, the current single-group pre-test/post-test study aimed to determine whether an intervention reduces oromotor activity and masticatory muscle pain in M/TMD patients with high levels of masticatory muscle activity associated with sleep bruxism.

Materials and Methods

Participants

Participants were women selected from among those with M/TMD who met the Research Diagnostic Criteria for TMD (RDC/TMD)[18] and who had participated in an earlier PSG study investigating risk factors for TMD.[11]

Since few M/TMD patients met stringent research diagnostic criteria for sleep bruxism,[12] lower levels of EMG activity consistent with sleep bruxism during the earlier study's PSG evaluation were permitted (ie, multiple grinding noises during sleep or rhythmic masticatory muscle activity characteristic of sleep grinding), but the initial review of their PSG record needed to show some characteristic sleep-grinding activity. Additionally, they had to report that a sleep partner told them that they ground their teeth at night (see Raphael et al[11] for details). Telephone screening of potentially eligible patients confirmed that *(a)* they reported that they experienced facial pain that averaged ≥ 4 on a 0-to-10 pain severity scale (0 = no pain, 10 = worst pain imaginable) over at least 4 of the last 7 days, *(b)* they experienced pain on waking in the morning, and *(c)* they were not pregnant or –if sexually active—were using birth control. The first two criteria were designed to identify participants who had pain of sufficient severity to allow for change and whose variation in pain could potentially be related to oromotor activity during sleep. At the first in-person study visit, the RDC examination was repeated[18] to confirm that participants still met criteria for M/TMD.

Of 19 women who began the study, five discontinued the study during a 2-week baseline EMG-monitoring phase, prior to treatment in a contingent-stimulation mode. Two discontinued because of concerns about using an "experimental device"; the other three discontinued because of participant burden involved in a daily study over 10 weeks. Fourteen women with M/TMD successfully completed the 10-week study. The study was approved by the New York University School of Medicine Institutional Review Board, and subjects completed a detailed informed-consent process prior to the beginning of the study.

EMG Event Monitoring and Treatment

The Grindcare device can be operated in two different modes: The first is EMG-monitoring only, in which an electrode is placed on the skin overlying the temporalis muscle (Fig 1) and signals are conducted over a 12-inch cable to a 15 × 55 ×40-mm unit that digitizes, analyzes, and stores extracted event data in an internal microchip. In brief, this is done by filtering the EMG signals between 250 and 600 Hz, sampling the data (10-bit, 2-kHz sampling rate), performing Fast Fourier Transformation (FFT), and finally comparing the root mean square (RMS)

outcome to a threshold value that is set to 20% of the maximum EMG during a clench at 60% of the maximum voluntary contraction (MVC). Setup of the threshold level is done every time the device is mounted before sleep. When the amplitude of the EMG signal has been above the threshold for more than 100 ms, an event is recorded in the log file.

An event can represent a voltage that exceeds threshold for up to 1 second; longer continuing events are counted as additional events. The total number of EMG events is then used to characterize each individual on each night. Through its biocalibration procedure, the Grindcare device is intended to detect bruxism-related EMG events, ie, those occurring when the patient actually grinds his/her teeth and there is activity in the jaw-closing musculature. Methodological studies have demonstrated that the signal recognition algorithm in the device differentiates among bruxism/clenching, relaxing, and grimacing activity.[17]

Treatment of EMG Events Through Contingent Stimulation

In its contingent-stimulation treatment mode, the Grindcare device delivers to the skin overlying the temporalis muscle a nonpainful electrical stimulus (a train of 92 biphasic pulses ramping up to the nightly determined maximum over 450 ms) when the subject's level of temporalis muscle EMG activity exceeds the amplitude determined during nightly biocalibration. The intensity (1 to 7 mA) of this stimulus, also set each night before sleep, is at a level that the subject finds to be just reliably detectable. The electrical stimulus has been described as a mild "tap." Just as during the EMG-monitoring–only phase, the device records the number of EMG events for which a contingent stimulus has been delivered (for details, see Jadidi et al[19]).

Design

The study employed a single-group pre-test/post-test design consisting of a 2-week baseline period, 6 weeks of event-contingent Grindcare device treatment, and a 2-week follow-up period. EMG events were recorded nightly throughout the 10-week study via the device, and contingent stimulation ("treatment") was delivered nightly between weeks 3 and 8. Subjects were instructed to place a phone call to the study's answering machine nightly before sleep to report their average daily pain. If a subject failed to place a message, the study coordinator contacted the subject during the next day to record her pain score. In addition to nightly pain reports, the study evaluated current levels of spontaneous pain and pain elicited during in-person RDC-specified muscle palpation examinations conducted at weeks 1 (baseline), 5 (mid-active treatment), 8 (end of active treatment), and 10 (end of 2-week follow-up) (Table 1).

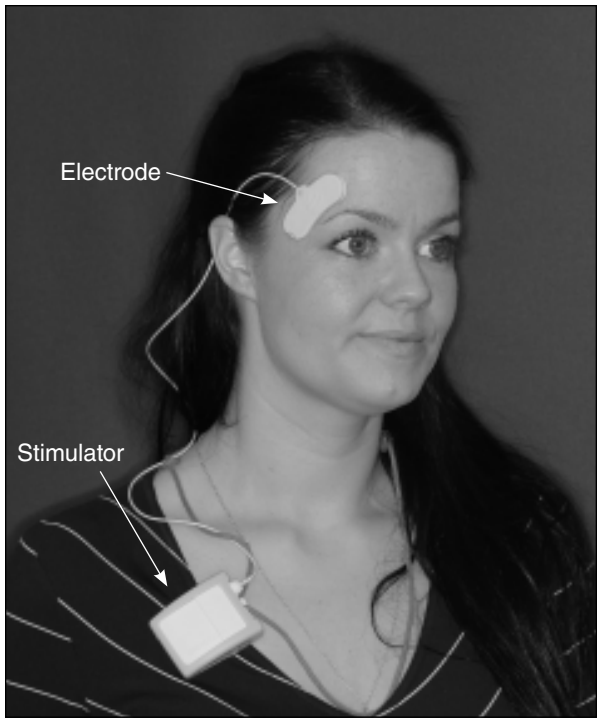

Fig 1 Depiction of Grindcare unit electrode and stimulator.

Subjects were asked to maintain their current pain-related medications or treatments throughout the course of the 10 weeks of the study in order to avoid confounding changes in pain with changes in co-interventions.

Measures

EMG Events. To assess the effect of the Grindcare device on the number of nightly EMG events, the number of EMG events was standardized as a rate per minute of sleep (E/m) per night.

Pain. The primary measure of spontaneous pain was average pain over the current day, as reported during the subject's nightly call-in. The subject was instructed to score her pain on a 0- to-10 scale, where 0 = no pain and 10 = pain as bad as it could be. Secondary measures of pain were derived during in-person study visits at weeks 1, 5, 8, and 10. *Elicited pain on examination* was defined as the number of painful tender-point (TP) sites found using RDC-examination[18] muscle palpation specifications. Values could range between 0 and 20 TPs, but must

Table 1 Overview of Study Design and Assessments (Measures/Treatment by Week)

	Study week									
	Baseline		Active treatment						Follow-up	
	1	2	3	4	5	6	7	8	9	10
In-person visit no.	1	2			3			4		5
EMG	x	x	x	x	x	x	x	x	x	x
Contingent stimulation	x	x	x	x	x	x				
Self-report sx	x	x	x	x	x	x	x	x	x	x
RDC exam	x				x			x		x

Each study night, EMG events per minute (E/m) and average daily pain were recorded.

have been at least 3 at the initial visit in order to meet RDC criteria. *Spontaneous pain intensity at time of examination* was used to determine consistency between findings by utilizing nightly pain and immediate spontaneous pain at time of examination. Current spontaneous pain was recorded on a scale ranging from 0 (no pain) to 10 (pain as bad as it could be).

Perceived Symptom Change. Finally, the study evaluated (at week 10) the extent to which subjects felt that their symptoms changed since they began to be treated with the Grindcare device. The following dimensions were rated: overall health, facial pain, mood, sleep quality, grinding and clenching at night, and grinding and clenching during the day. Scale values for each symptom dimension ranged from 1 (much worse) to 7 (much better). The scale value of 4 represented "the same/no change."

Statistical Analysis

Linear mixed-model analysis (IBM SPSS VER 20) was used to model linear and quadratic treatment-related reductions in E/m. While conceptually parallel to ANOVA, this analysis provides correct probability levels in consideration of the dependencies that arise from repeated observations of the same individuals. A linear model would evaluate the hypothesis that treatment produced a reduction in E/m during treatment relative to baseline, without a pattern of return to baseline activity levels during the follow-up period. In contrast, a quadratic model would evaluate the hypothesis that treatment produced a reduction in E/m during treatment that returned to baseline activity levels during the follow-up period.

Similar analyses modeled changes in spontaneous pain (ie, nightly pain ratings and current pain reported at in-person sessions) and elicited pain (ie, as measured by number of painful tender points on palpation). A final set of analyses extended those described above, simultaneously modeling the linear and quadratic effects of time and E/m, in order to test whether changes in E/m could account for any changes in pain over the course of the trial.

For measures of perceived symptom change, the study tested whether 95% confidence intervals (CI) for mean change excluded a value of 4, indicating "no change."

Sample size was determined to be 14 in order to detect a 0.7 standard deviation (SD) difference between pre- and posttreatment means with type 1 and 2 error rates of 5% and 20%, respectively (G*Power [v. 3.0.10][20]). A sample of this size proved sufficient to detect significant reductions in the number of EMG events before and after treatment in a similarly designed prior study of the Grindcare device.[17]

Since the current investigation was driven primarily by an interest in understanding possible bruxism-related mechanisms of M/TMD rather than efficacy of the Grindcare device, the usually recommended intent-to-treat analysis is inapplicable. Only those who were protocol adherent could inform study endpoints regarding mechanism. Thus, dropouts during the baseline period (ie, 2; see Protocol Adherence below) were excluded from primary analyses, and new subjects were recruited to complete the cohort.

Results

Protocol Adherence

Two subjects complied fully with the 10-week protocol, wearing the Grindcare device in monitoring or treatment mode and completing nightly call-in pain diaries on all 70 nights. Another seven subjects adhered to the protocol for 60 or more nights. The subject who was the least adherent to the protocol provided data for 48 of the 70 nights. Had all

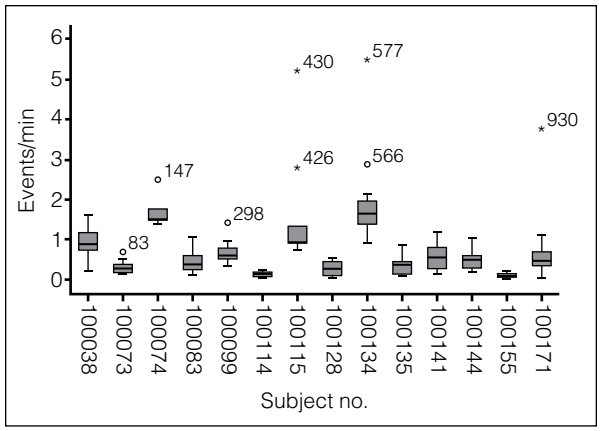

Fig 2 Individual subject variability in EMG events per minute of sleep over study nights. Boxplots show, by subject, the median number of events/min *(line within box)*, the middle 75% of scores *(upper and lower box limits)*, and outliers *(filled circles)*. Some subjects produced highly variable levels of response, which necessitated subsequent z-transformation and ranking.

subjects adhered to the protocol on all study nights, 980 nights of EMG events and pain would have been available for analysis. Excluding nights without adherence yielded 871 nights for analysis, for an average adherence across all subjects and nights of 88.9%.

Among 14 completers, self-reported average pain over the past 2 weeks was 3.9 (SD = 2.1; range, 1 to 8) during the first in-person visit. Mean age was 34.9 years (SD = 11.5; range, 24 to 64). Most self-identified their race as white (71.43%).

At the time of study completion, PSG records from the earlier study[11] from which subjects were drawn had been fully scored for sleep bruxism. Of the 14 completers, four met stringent research diagnostic criteria for sleep bruxism[12] and an additional six showed levels of sleep bruxism characterized as "moderate" in other research.[21] Three of the other four completers engaged in at least two nightly periods of grinding noise. Thus, the large majority of the 14 completers had engaged in moderate-to-severe bruxism in prior PSG studies.

Eight of the 14 subjects used over-the-counter anti-inflammatory medication on an as-needed basis throughout the study; consistent with instructions to participants, all other medication use remained constant during the study.

EMG Events per Minute of Sleep (E/m)

Event counts during the 14 baseline nights averaged less than 1 E/m (mean = 0.66, SD = 0.76). As suggested by an SD that is larger than the mean, data were skewed (skewness ratio = 1907.6), and the median number of E/m was 0.45 E/m. Figure 2 displays EMG events per minute. It shows that individual activity ranged widely, from a median of 0.1 to 1.5 E/m. While nightly values were symmetrically distributed within 11 individuals, those from another four produced skewness ratios between 3 and 5, indicating a few nights with high levels of E/m, and in two cases more than 5.

To minimize the influence of these anomalies, data were standardized within subjects across their ≤ 70 nights to have a mean of 0 and an SD of 1. Although this transformation removed differences in level of E/m between individuals, it did not fully correct skewness (skewness ratio = 9.0). Therefore, data were further transformed to ranks. In this way, the mean rank of E/m across all nights and subjects was 436 (50th percentile across all 871 nights) within a rectangular distribution. Scores > 436 for any treatment period indicate a majority of ranks above each individual's average, and scores < 436 represent a clustering of ranks below the average. All subsequent analyses of E/m utilized the rank of these z-score transformed scores, RzE/m. While parallel analyses that used only the rank transformation (or the z-transformation) produced a similar pattern of results, dual transformations yielded the greatest precision.

Changes in RzE/m

If treatment was an effective intervention for EMG activity, one would expect to see high average ranks before treatment, reduced ranks during treatment, and depending on the duration of effect, either reduced or reverting ranks during follow-up. Figure 3 shows mean (± SE) RzE/m scores over each of the 10 treatment weeks. As expected, initial scores appeared above the median rank, indicating more events during baseline. Scores then decreased during treatment and again rose toward initial levels during follow-up. Mixed-model analyses confirmed that these changes were unlikely to have occurred by chance, producing a best-fitting model with significant linear and quadratic terms. This model, with an initial rank of 488.3 (SE = 21.4), determined that grinding events decreased 35.4 ranks per week as a

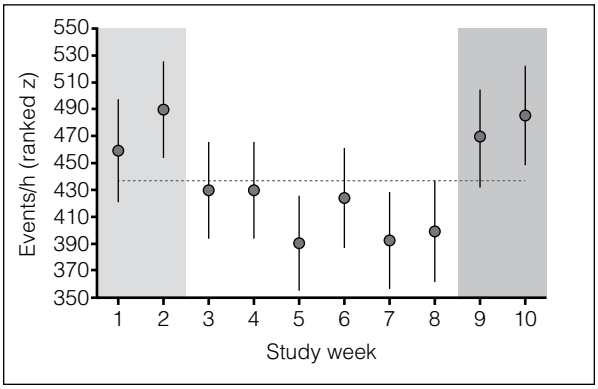

Fig 3 Mean (SEM) ranked Z-transformed EMG events per hour (RzE/h) broken down by treatment period and study week. Chart shows mean level of the transformed measure of events/min *(filled circles)* and SE *(vertical lines)* as a function of study week. Relative to baseline and follow-up periods *(shaded areas)*, event rates fell below the mean level *(indicated by the broken horizontal line)* during treatment.

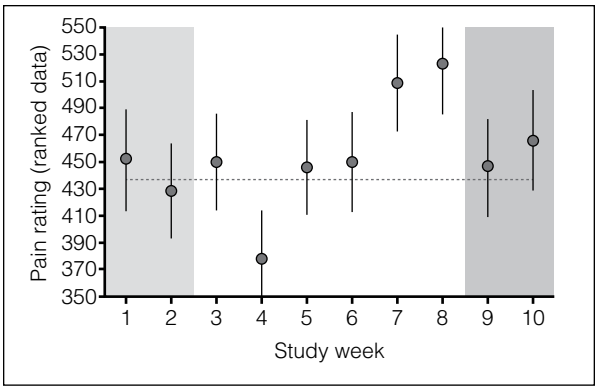

Fig 4 Mean (SEM) ranked nightly pain reports broken down by treatment period and study week. Chart shows mean level of the transformed measure of nightly pain *(filled circles)* and SE *(vertical lines)* as a function of study week. Pain ratings were as likely to be above the mean level *(indicated by the broken horizontal line)* during baseline and follow-up periods *(shaded areas)* as during treatment.

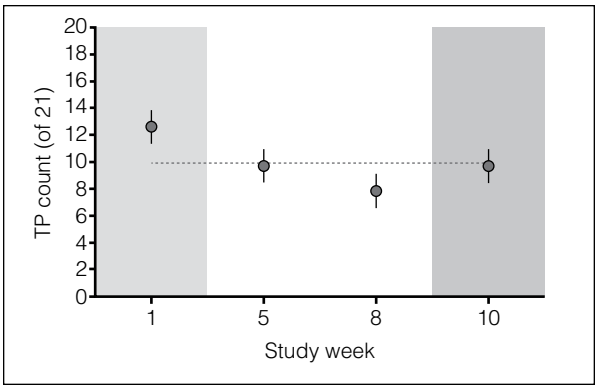

Fig 5 Mean (SEM) tender-point (TP) counts during examination broken down by treatment period and study week. Chart shows mean count of facial tender points *(filled circles)* and SE *(vertical lines)* as a function of study week. Relative to baseline, these counts tended to decline during treatment and remain depressed during follow-up.

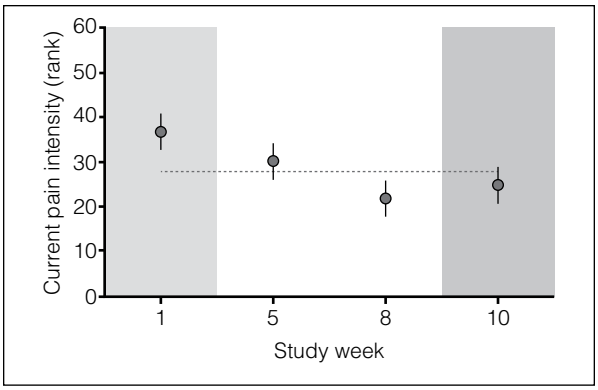

Fig 6 Mean (SEM) rank of spontaneous pain (current paint intensity) at time of examination broken down by treatment period and study week. Chart shows mean ratings of current pain intensity *(filled circles)* and SE *(vertical lines)* as a function of study week. Relative to baseline, pain ratings declined during treatment and remain depressed during follow-up.

function of the linear term (P = .002) and increased 3.8 (SE = 1.2) ranks per week as a function of the quadratic term (P = .001) over the 10 weeks. There was no evidence that participants differed in these slope parameters, suggesting a good fit for all participants. Viewed differently, there was a significant reduction (P < .05) in the mean RzE/m during weeks 5, 7, and 8, relative to the average of the two baseline periods. Thus, data indicate that EMG events were reduced in a statistically reliable fashion during contingent stimulation with the Grindcare device, but returned to baseline levels within the first week of follow-up.

Changes in Nightly Pain Reports

If treatment was an effective intervention for pain, one would expect to see high average report levels of nightly pain intensity before treatment, reduced levels during treatment, and, depending on the duration of effect, either reduced or reverting levels during follow-up. By contrast, Fig 4 shows a haphazard sequence of nightly pain reports over the study period, and statistical analysis failed to show any systematic changes during treatment (linear and quadratic trends over 10 weeks, P = .58 and P =.82, respectively). Thus, while the data demonstrated a

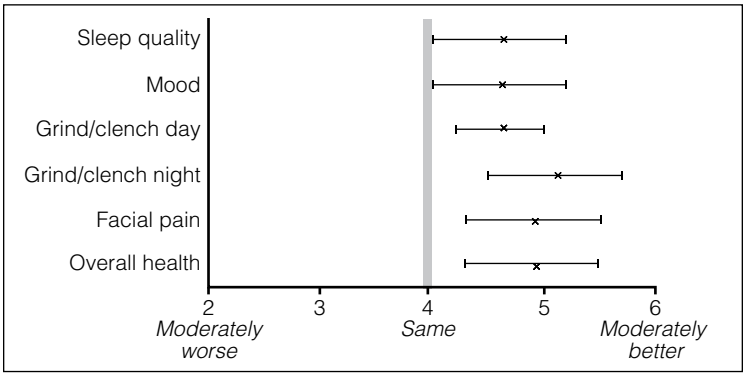

Fig 7 Mean and 95% confidence interval of perceived symptom change at end of follow-up (week 10). Chart shows mean ratings of change in other perceived symptoms *(filled circles)* and 95% confidence limits *(horizontal lines)* during follow-up. All showed improvement over the trial.

significant relationship between Grindcare treatment and reduced EMG events, there was no evidence to support any change in nightly pain reports.

Changes in Palpated (TP Count) Pain

Similarly, one would expect to see high average report levels of elicited pain before treatment, reduced levels during treatment, and, depending on the duration of effect, either reduced or reverting levels during follow-up. The data supported this expectation. Figure 5 shows mean (SE) TP counts at each of the four examinations. From a relatively high starting point, scores decreased during treatment and rose during follow-up. Mixed-model analyses confirmed that these changes were unlikely to have occurred by chance, producing a best-fitting model with significant linear and quadratic terms. This model suggested an (adjusted) initial score of 12.8 TPs (SE = 1.1) that decreased 1.3 (0.4) TPs per week as a function of the linear term (P = .01), and increased 0.1 (0.04) TPs per week as a function of the quadratic term (P = .001). There were substantial differences among subjects in level of TP intensity—almost 60% of what would otherwise have been relegated to residual variability was attributable to the random intercept (P = .06), and including this term in the model greatly increased precision. However, there were no statistical differences between participants in the slope parameters (P = .12), suggesting a similar trajectory over time among all participants. Compared to the baseline period, there was a reduction (P < .05) in the mean TP count during each subsequent evaluation. Thus, while EMG events were reduced only during the contingent stimulation period, TP counts were reduced in a statistically reliable fashion during contingent stimulation as well as during follow-up.

Changes in Spontaneous Pain (Current Pain Intensity)

The intensity of spontaneous pain at the time of the in-person examination would also be expected to vary with treatment as for the other two measures of pain. Figure 6 shows the mean rank (SE) of "current pain" reported at each in-person examination (preliminary analysis identified skew in these data, and they were transformed to ranks). At visit 1, subjects' pain was maximal—the mean (SD) rank of 36.8 (13.6) was well above the middle value shown in Fig 6. Scores then decreased during treatment and remained low during follow-up, suggesting a sustained reduction in spontaneous pain during participation. Analysis produced a best-fitting model, suggesting that these changes were not likely to have occurred by chance. The model showed an (adjusted) initial rank of 37.2 (SE = 3.5) that decreased 2.8 (1.4) ranks per week as a function of the linear term (P = .05), but there was not additional benefit of including a quadratic term (P = .32), unlike measures of grinding and intensity of palpated pain. Like palpated pain, there were substantial differences between subjects in level of pain report—about 50% of what would otherwise have been relegated to residual variability was attributable to modeling the random intercept (P = .10) and including this term in the model greatly increased precision. However, there were no statistical differences between participants in the slope parameters (P = .14). Compared to the baseline period, there was a significant reduction (P < .05) in the mean pain report at study weeks 8 and 10. Thus, current pain at the time of in-person examination was reduced in a statistically reliable fashion during the final week of contingent stimulation and during follow-up, similar to the trajectory shown for TP counts.

Table 2 Tender-Point Counts Predicted by the Linear and Quadratic Effects of Time (Model 1), Linear and Quadratic Effects of Electromyographic Events (Model 2), and Both (Model 3)

	Model 1		Model 2		Model 3	
Effect	Regression coefficient (SEM)	*P*	Regression coefficient (SEM)	*P*	Regression coefficient (SEM)	*P*
Intercept	12.77 (1.08)	< .001	12.54 (2.0)	< .001	13.59 (1.87)	< .001
Linear time	–1.32 (.37)	.001	–	–	–0.9 (.37)	.02
Quadratic time	0.10 (.04)	.01	–	–	0.07 (.04)	.07
Linear RzE/m	–	–	–0.01 (0.009)	.12	–0.01 (.01)	.24
Quadratic RzE/m	–	–	< .001 (< 0.001)	.14	< 0.001 (< 0.001)	.27

Table 3 Current Pain (at In-Person Examination) Predicted from Linear and Quadratic Effects of Time (Model 1), Linear and Quadratic Effects of Electromyographic Events (Model 2), and Both (Model 3)

	Model 1		Model 2		Model 3	
Effect	Regression coefficient (SEM)	*P*	Regression coefficient (SEM)	*P*	Regression coefficient (SEM)	*P*
Intercept	37.24 (3.53)	< .001	26.12 (6.79)	< .001	38.68 (5.54)	< .001
Linear time	–2.81 (1.35)	.05	–	–	–3.40 (1.42)	.02
Quadratic time	0.15 (0.14)	.315	–	–	.21 (.15)	.18
Linear RzE/m	–	–	0.01 (0.03)	.70	–.001 (.03)	.96
Quadratic RzE/m	–	–	< 0.001 (< 0.001)	.81	< 0.001 (< 0.001)	.90

Perceived Symptom Change

As was the case for the report of painful symptoms, more global indices of well-being would also be expected to improve with treatment. These were assessed as a retrospective report of well-being at week 10. Figure 7 shows that subjects reported about a 1-point improvement ("mild improvement") in global health, facial pain, and grinding and clenching during both the night and day (all $P < .05$). Mood and sleep quality failed to improve in a statistically reliable fashion, with 95% CI including the value of 4, equivalent to "no change." Thus, consistent with the report of palpated pain and current pain, global well-being improved over the course of the study.

Relationship Between Changes in EMG Events and Changes in Pain Over Time

Prior analyses revealed that both elicited pain and spontaneous pain at the time of examination decreased during treatment and remained depressed during follow-up. EMG events also decreased during treatment, although these changes returned toward baseline levels during follow-up. To evaluate the role of variation in EMG events to variation in examination session pain reports, covariates representing the linear and quadratic effects of EMG events were added to those representing the effect of time in the prediction of elicited and spontaneous pain. If pain and EMG events were changing in close association, the prediction of pain from these new covariates would be redundant with that attributable to treatment, and would appear as a reduction in the unique effect of treatment.

Elicited Pain. As shown in Table 2, the terms representing changes in EMG events over time were not significantly related to TP counts. Model 1 demonstrated the effects of treatment time on the number of painful TPs. Model 2 showed that the TP count was not related to EMG events. Model 3 showed that after adjusting for changes in EMG events, treatment-time effects were reduced but remained a significant predictor of TP count. Viewed differently, while the original model posited a linear reduction of 1.3 TP per week, the latter model posited a reduction of only a 1.0 TP; thus, taking EMG changes into account, the remaining (unique) effect attributable to time was reduced by about 25%. The quadratic term showed little change. This suggests that while some of the reduction in TP count could be attributed to a reduction in EMG events, there remained a significant change in the report of elicited pain that was independent of changes in EMG events.

Current Pain Intensity. As shown in Table 3, model 1 demonstrated the effect of time on the intensity of current pain. Model 2 showed that current pain was not related to EMG events, and model 3 showed

that adjusting for changes in EMG events over time did not reduce the effect of treatment time on change in current pain intensity. If anything, the fit was more precise after adjustment for EMG events. While the original model posited a linear reduction of 2.8 units per examination, the latter model posited a reduction of 3.4; thus, taking EMG changes into account, the effect of treatment time was increased. The quadratic terms were not important in either analysis. Thus, the report of current pain at examination could not be attributed to changes in EMG events.

Discussion

The current study determined that the Grindcare device, designed to reduce nightly oromotor events associated with bruxism, was associated with a reliable reduction of these EMG events during contingent stimulation treatment periods, but the frequency of EMG events quickly returned to baseline levels when contingent stimulation was withdrawn. These findings replicated the contingent stimulation treatment effect reported in prior research.[17] The current study's sample differs from that in the prior report, in that participants had clinically significant pain and met RDC criteria for M/TMD, in addition to having shown prior sleep oromotor activity consistent with sleep bruxism.

The present study's principal question was whether reduction of EMG activity, successfully achieved through contingent stimulation, could also reduce M/TMD pain, thus revealing a link between oromotor activity and pain for at least some TMD patients. Analyses based on the primary pain measure (ie, nightly pain reports, over ≤ 70 nights) revealed no consistent pattern over change during or following treatment. By themselves, these findings indicate an independence of nighttime oromotor activity and spontaneous pain severity, even in patients selected for relatively high levels of both characteristics. By rejecting a "vicious cycle" model of muscle hyperactivity and pain,[2] the findings are supportive of newer etiologic perspectives[13,14] that view M/TMD as a complex disease involving multiple mechanisms.

Results for elicited pain differed from those based on nightly pain: Compared to baseline, counts of painful masticatory muscle TPs were reduced during treatment and follow-up. This might have been due to the often-observed[22] independence of measures of spontaneous and elicited pain in M/TMD patients. It also might have been due to regression toward the mean. Subjects were selected to have moderate or greater levels of pain in order to enroll in this study; if these individuals were experiencing temporary exacerbations of their pain, one would have expected those levels to return to more usual levels over the course of the study. The pattern could be the result of the relatively increased demand characteristics of the in-person examination compared to at-home assessment. Finally, it could also have been due to the difference in the number and specific times of observation periods for nightly spontaneous pain (up to 70 nightly reports) versus elicited pain (four examinations) over the course of the study. When changes in spontaneous pain ("current pain") at the time of the four examinations were assessed, a pattern of reduction was revealed that mirrored the reduction for elicited pain. Thus, it seems likely that the divergent change patterns in nightly spontaneous and elicited pain could be attributed to nonspecific effects associated with either the different assessment periods for each measure or the demand characteristics of the examination setting.

While nightly pain was not consistently reduced over the course of the study, the study did test whether changes in pain TPs or current spontaneous pain at the time of examination could be explained by changes in nightly EMG events. Although a model including EMG events reduced the absolute magnitude of the linear effect of time on TP counts, the significance of the reduction of the elicited pain remained virtually unchanged. Thus, the data provide limited evidence that the reduction in TP count occurred in the same individuals who evidenced a reduction in EMG events. Most critically, there remained a significant change in the report of elicited pain that could not be attributed to changes in EMG events. Reductions in the intensity of spontaneous pain reported at the time of examination could also not be attributed to changes in EMG events over the course of the study. Thus, changes in pain and EMG events appear to have evolved independently of one another.

Although these data tend to argue against the role of sleep bruxism in explaining variability of pain in patients with both M/TMD and sleep bruxism, it should be noted that the measure of EMG activity that was used has not yet been validated as an accurate measure of sleep bruxism activity. The measure gathered by the Grindcare device has been shown to differentiate between awake clenching and awake grimacing or relaxation, but many other types of oromotor activity may occur during the night,[11,23–25] particularly among sleep bruxers and especially prior to sleep or during nighttime awake periods. Unlike PSG studies, recording or scoring of EMG events with the Grindcare unit will not differentiate awake from sleep periods. Despite the fact that the Grindcare unit–derived measure of EMG events is likely

to contain some error, the measure was sufficiently robust to detect reduction of EMG events during treatment. Moreover, supplementary analyses of PSG records from earlier research[11] indicated that M/TMD patients spend approximately 90% of post–sleep-onset time actually sleeping, so that waking periods after sleep onset are likely to represent a modest amount of potential Grindcare recording time.

As well, while there are inherent limitations in measures of EMG events as a proxy for sleep bruxism, these costs must be balanced against the unique ability of the Grindcare device to measure nightly covariation of EMG events. Due to both cost and participant burden, nightly PSG-based measurement of sleep bruxism would not have been practicable over a period of up to 70 nights.

The major limitation of this study was the absence of a control group. Without one, the specific meaning of pre/post changes in EMG events and pain are uncertain. As well, a regression to the mean hypothesis regarding reduction in pain could be tested by showing that a control group also reported less pain over time.

Although the sample size was quite modest, it was sufficient to detect changes in EMG events as well as changes in spontaneous and elicited pain at the time of the physical examinations. Given the haphazard pattern in nightly pain reports, it is unlikely that a larger sample would show significant results.

The sample of 14 subjects was not selected to be a representative sample of M/TMD patients, but one in which a reasonably high level of both pain and sleep bruxism were present, providing the foundation for potential covariation. Depending on the specific standard used to score bruxism, nearly all of the 14 subjects were eventually characterized as displaying moderate to severe sleep bruxism in earlier PSG studies[11]; only about one-fourth of a larger sample of M/TMD patients had scored similarly.

From a clinical perspective, this study does not indicate that Grindcare is likely to be an appropriate treatment for M/TMD patients who seek to reduce their masticatory muscle pain. However, it suggests that the intervention will likely succeed in reducing EMG events related to sleep bruxism and may have utility for reducing ongoing dentition wear. It may also eventually be shown to be a cost-effective method to screen patients for sleep bruxism.

In conclusion, these data do not support the hypothesis that there is a causal relationship between changes in EMG events and masticatory muscle pain, but confirm prior reports[17] that treatment with contingent electrical stimulation can significantly reduce EMG events. The study supports a growing body of literature showing independence of variation in sleep bruxism or other nighttime oromotor activity and variation in pain severity in M/TMD patients.

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References

1. Manfredini D, Guarda-Nardini L, Winocur E, Piccotti F, Ahlberg J, Lobbezoo F. Research diagnostic criteria for temporomandibular disorders: A systematic review of axis I epidemiologic findings. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011;112:453–462.
2. Laskin DM. Etiology of the pain-dysfunction syndrome. J Am Dent Assoc 1969;79:147–153.
3. Schwartz LL. A temporomandibular joint pain-dysfunction syndrome. J Chronic Dis 1956;3:284–293.
4. Manfredini D, Lobbezoo F. Relationship between bruxism and temporomandibular disorders: A systematic review of literature from 1998 to 2008. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2010;109:e26–e50.
5. Lobbezoo F, Lavigne GJ. Do bruxism and temporomandibular disorders have a cause-and-effect relationship? J Orofac Pain 1997;11:15–23.
6. Svensson P, Jadidi F, Arima T, Baad-Hansen L, Sessle BJ. Relationships between craniofacial pain and bruxism. J Oral Rehabil 2008;35:524–547.
7. Le Resche L, Truelove EL, Dworkin SF. Temporomandibular disorders: A survey of dentists' knowledge and beliefs. J Am Dent Assoc 1993;124:90–94,97–106.
8. Glaros AG, Glass EG, McLaughlin L. Knowledge and beliefs of dentists regarding temporomandibular disorders and chronic pain. J Orofac Pain 1994;8:216–222.
9. Tegelberg A, Wenneberg B, List T. General practice dentists' knowledge of temporomandibular disorders in children and adolescents. Eur J Dent Educ 2007;11:216–221.
10. van der Meulen MJ, Ohrbach R, Aartman IH, Naeije M, Lobbezoo F. Temporomandibular disorder patients' illness beliefs and self-efficacy related to bruxism. J Orofac Pain 2010;24:367–372.
11. Raphael KG, Sirois DS, Janal MN, et al. Sleep bruxism and myofascial temporomandibular disorders: A laboratory-based polysomnographic investigation. J Am Dent Assoc 2012;143:1223–1231.
12. Lavigne GJ, Rompre PH, Montplaisir JY. Sleep bruxism: Validity of clinical research diagnostic criteria in a controlled polysomnographic study. J Dent Res 1996;75:546–552.
13. Maixner W, Diatchenko L, Dubner R, et al. Orofacial pain prospective evaluation and risk assessment study—the OPPERA study. J Pain 2011;12(11 suppl):T4-11.e1–e2.
14. Benoliel R, Svensson P, Heir GM, et al. Persistent orofacial muscle pain. Oral Dis 2011;17(suppl 1):23–41.
15. Lobbezoo F, van der Zaag J, van Selms MK, Hamburger HL, Naeije M. Principles for the management of bruxism. J Oral Rehabil 2008;35:509–523.

16. Huynh NT, Rompre PH, Montplaisir JY, Manzini C, Okura K, Lavigne GJ. Comparison of various treatments for sleep bruxism using determinants of number needed to treat and effect size. Int J Prosthodont 2006;19:435–441.
17. Jadidi F, Castrillon E, Svensson P. Effect of conditioning electrical stimuli on temporalis electromyographic activity during sleep. J Oral Rehabil 2008;35:171–183.
18. Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: Review, criteria, examinations and specifications, critique. J Craniomandib Disord 1992;6:301–355.
19. Jadidi F, Norregaard O, Baad-Hansen L, Arendt-Nielsen L, Svensson P. Assessment of sleep parameters during contingent electrical stimulation in subjects with jaw muscle activity during sleep: A polysomnographic study. Eur J Oral Sci 2011;119:211–218.
20. Faul F, Erdefelder E, Buchner A, Lang A-G. Statistical power analyses using G*Power 3.1: Tests for correlation and regression analyses. Behav Res Method 2009;41:1149–1160.
21. Rompre PH, Daigle-Landry D, Guitard F, Montplaisir JY, Lavigne GJ. Identification of a sleep bruxism subgroup with a higher risk of pain. J Dent Res 2007;86:837–842.
22. Janal MN, Raphael KG, Klausner J, Teaford M. The role of tooth-grinding in the maintenance of myofascial face pain: A test of alternate models. Pain Medicine 2007;8:486–496.
23. Dutra KM, Pereira FJ Jr, Rompre PH, Huynh N, Fleming N, Lavigne GJ. Oro-facial activities in sleep bruxism patients and in normal subjects: A controlled polygraphic and audio-video study. J Oral Rehabil 2009;36:86–92.
24. Lavigne GJ, Khoury S, Abe S, Yamaguchi T, Raphael K. Bruxism physiology and pathology: An overview for clinicians. J Oral Rehabil 2008;35:476–494.
25. Velly-Miguel AM, Montplaisir J, Rompre PH, Lund JP, Lavigne GJ. Bruxism and other orofacial movements during sleep. J Craniomandib Disord 1992;6:71–81.