Effect of Botulinum Toxin Injection on Nocturnal Bruxism
A Randomized Controlled Trial

ABSTRACT

Objective: To evaluate the effect of botulinum toxin type A on nocturnal bruxism.

Design: Twelve subjects reporting nocturnal bruxism were recruited for a double-blind, randomized clinical trial. Six bruxers were injected with botulinum toxin in both masseters, and six with saline. Nocturnal electromyographic activity was recorded in the subject’s natural sleeping environment from masseter and temporalis muscles before injection, and 4, 8, and 12 wks after injection and then used to calculate bruxism events. Bruxism symptoms were investigated using questionnaires.

Results: Bruxism events in the masseter muscle decreased significantly in the botulinum toxin injection group ($P=0.027$). In the temporalis muscle, bruxism events did not differ between groups or among times. Subjective bruxism symptoms decreased in both groups after injection ($P<0.001$).

Conclusions: Our results suggest that botulinum toxin injection reduced the number of bruxism events, most likely mediated its effect through a decrease in muscle activity rather than the central nervous system. We controlled for placebo effects by randomizing the interventions between groups, obtaining subjective and objective outcome measures, using the temporalis muscle as a control, and collecting data at three postinjection times. Our controlled study supports the use of botulinum toxin injection as an effective treatment for nocturnal bruxism.

Key Words: Electromyography, Masseter Muscle, Temporalis Muscle, Randomized Controlled Trial
Bruxism has been defined as an oral habit consisting of involuntary rhythmic or spasmodic nonfunctional gnashing, grinding, or clenching of teeth. Bruxism is a very common condition in the general population. About 85%–90% of the general population reports bruxism to some degree during a lifetime. The prevalence of chronic nocturnal bruxism ranges from 20% to 25% in children, 5%–8% in the adult population, and 3% in the elderly.

Although factors such as emotional stress, neurologic disorders, medications, and occlusal interferences have been proposed, both the etiology and pathophysiology of bruxism are still unclear. Bruxism seems to have a multifactorial etiology and to be centrally mediated.

According to the American Sleep Disorders Association, the diagnosis of nocturnal bruxism is based on the reports of tooth grinding or clenching and one of the following signs: abnormal tooth wear, sounds associated with bruxism, and jaw muscle discomfort. Research measurement of bruxism has used electromyographic (EMG) activity of masticatory muscles by either portable electromyography or polysomnography. Fully instrumented laboratory-based nocturnal polysomnographic study allows multidimensional analyses of sleep-related physiologic behaviors but requires the subjects to sleep in an unfamiliar sleep laboratory environment. Alternatively, portable EMG recording devices enable recordings in the subject’s home environment with minimum expense. We elected to use the portable EMG device described in the Methods section.

Previous bruxism studies using EMG recording devices have used various criteria to define bruxism. Recently, Haketa et al. reported a semiautomated computer method that reduced error, decreased analysis time, and had high interexaminer reliability of the EMG-based analyses. Based on these considerations, their method was selected. The fraction of maximum voluntary contraction (MVC) used as the threshold for an EMG bruxism event (defined in Methods) will affect the number of EMG bruxism events. Hence, 20% MVC was selected to underestimate rather than overestimate the number of EMG events.

Various treatment modalities such as occlusal splints, pharmacologic agents such as benzodiazepine or L-dopa, and psychobehavioral therapy have been investigated for the management of bruxism, but none is reported to be fully effective. Recently, locally injected botulinum toxin has been used in various movement disorders, but its usefulness and objective effects on nocturnal bruxism have not been evaluated using objective measures such as EMG activity. Moreover, these studies often lacked one or more controls such as a placebo injection or an uninjected muscle (Table 1).

The aims of this study were to evaluate the effect of injection of botulinum toxin A into the masseter muscle on nocturnal bruxism using a portable EMG device, on the differences between masseter and temporalis muscle in the EMG bruxism activity, and on the changes in the subjective bruxism symptoms. We hypothesized that in the botulinum toxin-injected group the number of EMG bruxism events would decrease in the masseter muscle compared with the temporalis muscle and that the subjective bruxism changes would decrease.

**METHODS**

**Subjects**

An advertisement was distributed to the faculty, staff, and students of Seoul National University Dental Hospital requesting volunteers who reported that they were tooth grinders, otherwise healthy, and aged 20–30 yrs. The subjects were recruited from those who responded. Exclusion criteria were temporomandibular disorders, pain in the orofacial region, insomnia, known botulinum toxin allergy, pregnancy, neuromuscular disease, bleeding disorders, antibiotic therapy use, pulmonary disease that produced coughing during sleep, or infectious skin lesion at the site of the injection.

Seven men and five women with nocturnal bruxism participated in this study. The mean ages were 25.0 ± 2.35 yrs for men and 24.8 ± 0.83 yrs for women. The study was approved by an institutional review board (an ethical committee implementing the Korean equivalent of the Declaration of Helsinki), and informed consent was obtained from each subject. The subjects were randomly assigned to control (24.8 ± 1.47 yrs, 4 men and 2 women) and botulinum toxin injection groups (25.0 ± 2.28 yrs, 3 men and 3 women).

**Equipment and Instruments**

A bruxism symptom questionnaire to evaluate the subjective bruxism symptoms consisted of three items: (1) How often do you think that you had bruxed at night during the past 1 mo? (2) How often have you heard from your sleeping partner that you bruxed during the past 1 mo? (3) How often during the past month have you felt jaw stiffness on waking? The responses to each item were based on a 0–5 scale where 0 = none, 1 = very seldom, 2 = seldom, 3 = often (half the mornings), 4 = very often, 5 = every day. The rationale was that previous reports have used sub-
<table>
<thead>
<tr>
<th>Citation</th>
<th>Muscle Injected</th>
<th>Bruxism Origin</th>
<th>Sample Size</th>
<th>Control Group</th>
<th>Duration of Effect, wks</th>
<th>Toxin Type</th>
<th>Dose/Muscle (MU)*</th>
<th>Outcome Measure</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Van Zandijcke et al. 14</td>
<td>Masseter and temporalis</td>
<td>Brain injury</td>
<td>1</td>
<td>No</td>
<td>12</td>
<td>BTX-A (brand name not given)</td>
<td>1st: 25; 2nd: 25 (at 2 wks)</td>
<td>Clinical observation</td>
<td>Marked reduction</td>
</tr>
<tr>
<td>Ivanhoe et al. 15</td>
<td>Masseter and temporalis</td>
<td>Brain injury</td>
<td>1</td>
<td>No</td>
<td>12</td>
<td>BTX-A (brand name not given)</td>
<td>50</td>
<td>Clinical observation</td>
<td>Remained free of bruxism</td>
</tr>
<tr>
<td>Watts et al. 16</td>
<td>Masseter</td>
<td>Cranial-cervical dystonia</td>
<td>12</td>
<td>No</td>
<td>13</td>
<td>BTX-A (Brand name not given)</td>
<td>50</td>
<td>Subjective symptom</td>
<td>67% subjects reported improvement</td>
</tr>
<tr>
<td>Tan and Jankovic 17</td>
<td>Masseter</td>
<td>Not stated</td>
<td>18</td>
<td>No</td>
<td>19.1 ± 17.0</td>
<td>Botox</td>
<td>61.7 ± 11.1</td>
<td>Subjective symptom</td>
<td>Significant improvement</td>
</tr>
<tr>
<td>Pidcock et al. 18</td>
<td>Masseter</td>
<td>Brain injury</td>
<td>1</td>
<td>No</td>
<td>8</td>
<td>Botox</td>
<td>1st: 15; 2nd: 15 (at 2 mos)</td>
<td>Clinical observation</td>
<td>Persistent suppression</td>
</tr>
<tr>
<td>See and Tan 19</td>
<td>Masseter</td>
<td>Amphetamine induced</td>
<td>1</td>
<td>No</td>
<td>12–16</td>
<td>Dysport</td>
<td>50</td>
<td>Subjective symptom</td>
<td>Significant improvement</td>
</tr>
<tr>
<td>Nash et al. 20</td>
<td>Masseter and temporalis</td>
<td>Huntington’s disease</td>
<td>1</td>
<td>No</td>
<td>12–24</td>
<td>BTX-A (brand name not given)</td>
<td>25–50</td>
<td>Subjective symptom</td>
<td>Improvement</td>
</tr>
<tr>
<td>Monroy and da Fonseca 21</td>
<td>Masseter</td>
<td>Autism</td>
<td>1</td>
<td>No</td>
<td>8</td>
<td>Botox</td>
<td>15</td>
<td>Clinical observation</td>
<td>Improvement</td>
</tr>
<tr>
<td>Guarda-Nardini et al. 22</td>
<td>Masseter and temporalis</td>
<td>Not stated</td>
<td>10 + 10</td>
<td>Yes</td>
<td>24</td>
<td>Botox</td>
<td>20 (temporalis) 30 (masseter)</td>
<td>Subjective efficacy</td>
<td>Significant difference</td>
</tr>
</tbody>
</table>

*One mouse unit (MU) of Botox is clinically equivalent to ~3 MU of Dysport. Thus, when the brand name is not given, one is unsure of the dose.
jective measures, and we wished to compare our results with the previous data.

The test-retest reliability of the questionnaire with a 2-wk time interval was assessed on eight subjects before this study. The $\kappa$ index for each of the three questions ranged from 0.52 to 0.74. Hence, the agreement was moderate to substantial.

EMG signals were recorded with a Myomonitor EMG system (Delsys Inc, Boston, MA) with a four-channel, HP Jornada 720 Handheld Computer, and Myomonitor WinCE software. The recorded signals were amplified then sampled at 1024 Hz. The acquired data were analyzed with Myomonitor software.

**Procedure**

Several procedures were performed before the injection. Each subject completed the bruxism questionnaire. The subjects were asked to clench as hard as they could, and the EMG activity was recorded for 3 secs. This clench procedure was repeated three times. Off-line each 3-sec recording was converted to a series of root mean square values described later. The maximal value within each 3-sec clench was obtained, and the three values were averaged.

The subjects were instructed and trained on the use of the portable EMG machine, so that they could record data with it at home. The EMG data of both masseter and temporalis muscles were collected for three consecutive nights at home for an average of 6 hrs per night for each subject. If the electrodes became detached during the recording, EMG data were recorded for an additional night. The processing of these data is explained below.

Subjects were randomly assigned to either the botulinum toxin injection group or the saline injection group. For the experimental group, 80 mouse units of botulinum toxin A (Dysport, Ipsen, Wrexham, United Kingdom) were diluted in 0.8 ml of saline. For the control group, 0.8 ml of saline was used. Botulinum toxin or saline was injected into each subject’s masseter muscles at three sites. The first site was the inferior, prominent part of the masseter muscle observed when the subject was asked to clench, and the other two sites were 5 mm from the first point anteriorly and posteriorly. Subjects and operators were blind to the material injected, and the persons collecting the data were blind to the group membership of the subjects.

At 4, 8, and 12 wks after the injection, each subject completed the bruxism symptom questionnaire, and EMG data were collected for two consecutive nights as described earlier.

**Data Processing and Statistics**

The EMG data from each recordings of night were converted to root mean square values. The segment of time over which the root mean square value was computed was 0.125 sec, and the overlap of time segments was 0.0625 sec. From these data, the number of bruxism events per hour was calculated using the criteria of Haketa et al.:

1. A root mean square EMG amplitude above the 20% MVC level.
2. Events with duration longer than 2 secs, and
3. Intervals between each separate event were longer than 2 secs. These criteria have been shown to avoid signals from talking, blowing air, or other nonclenching low-level activities. Data reported are the average of the EMG activity from all of the nights available. As mentioned in the Introduction, various fractions of MVC have been used as the threshold to define an EMG bruxism event. To see how important this threshold might be, the data were also analyzed using 10% MVC as the threshold for a bruxism event.

The design of this research has three factors: muscle (masseter and temporalis), time (preinjection: 4, 8, and 12 wks), and group (botulinum toxin injection and saline injection). The multivariate repeated-measures statistical tests give results for main effects (muscle, time, and group), interactions of pairs of the three main effects, and an interaction of all three effects (muscle by time by group). If the interactions are significant, one proceeds to post hoc contrasts of the cells.

The within-subjects nature of our research design led to some not-so-simple statistical hypotheses. First, the main effect for group is not expected to be significant because this test includes both groups before injection where no group difference is expected, and it also includes the temporalis muscle in both groups where no difference is expected. Second, the main effect for time is not predicted to be significant because it includes both the masseter (expected to change) and the temporalis (not expected to change) muscles. Thus, we expect the interaction tests, which isolate the masseter from the temporalis muscles and isolate the preinjection from the postinjection times, to reveal the effect of the botulinum toxin.

Post hoc testing with Helmert contrasts was performed to compare preinjection with postinjection times. The Helmert contrasts compare the current cell with the average of the subsequent cells. Hence, the first Helmert contrast would compare the preinjection mean with the average of the 4-, 8-, and 12-wk means. (We predict this one to be significant.) The second Helmert contrast would compare the 4-wk mean with the average of the 8- and 12-wk means, and the third Helmert contrast would compare the 8- and 12-wk means. (We would not expect these latter two to be significant.)

To investigate bruxism scores from the symptom questionnaire, an analysis of variance involving time and group was used. Time was treated as...
a repeated measure and group as a between-subjects measure.

RESULTS

No subjects withdrew from our study, and no adverse events were reported. No subjects reported any dry mouth symptoms, which suggests that the botulinum toxin was not injected into the parotid gland.

EMG Bruxism Events

The number of EMG bruxism events was compared before injection between the first night and the average of the subsequent two nights, and no statistical differences were found (paired t test, \( P > 0.2 \)). Hence, data from all three nights were pooled for the preinjection data and for both nights for the postinjection data.

The number of EMG bruxism events (Table 2) decreased after the botulinum toxin injection in the masseter muscle but not in the temporalis muscle and did not seem to differ in the three postinjection times. Figure 1 suggests that the number of EMG events recorded from the masseter muscle: (1) was roughly the same in both the saline and botulinum toxin groups preinjection, (2) was roughly the same before and after injection in the control group (open squares), (3) was markedly lower postinjection in the botulinum toxin group (filled circles), and (4) did not change much in either group among the three postinjection times.

A three-way analysis of variance (Table 2) using muscle and time as within-subject factors and group (injection material) as a between-subjects factor gave a significant three-way interaction (\( P = 0.027 \)). Post hoc testing showed that (1) the postinjection mean EMG events at 4, 8, and 12 wks did not differ within any of the four muscle-and-injection groups (all \( P > 0.20 \)) and (2) averaged over the three postinjection times, the masseter-botulinum mean (0.23 ± 0.29) was lower (\( P < 0.001 \)) than

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Group</th>
<th>Before</th>
<th>4 wks</th>
<th>8 wks</th>
<th>12 wks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Masseter</td>
<td>Botulinum toxin</td>
<td>2.77±1.86</td>
<td>0.15±0.29</td>
<td>0.26±0.35</td>
<td>0.26±0.24</td>
</tr>
<tr>
<td></td>
<td>Saline</td>
<td>2.48±1.26</td>
<td>2.24±1.06</td>
<td>2.50±1.37</td>
<td>2.66±1.44</td>
</tr>
<tr>
<td>Temporalis</td>
<td>Botulinum toxin</td>
<td>2.28±1.21</td>
<td>2.51±2.49</td>
<td>2.25±0.77</td>
<td>1.89±1.03</td>
</tr>
<tr>
<td></td>
<td>Saline</td>
<td>1.81±1.52</td>
<td>1.85±1.64</td>
<td>2.01±1.64</td>
<td>1.70±1.52</td>
</tr>
</tbody>
</table>

Muscle × time × group: \( F = 3.523, \, df = 2.325, \, 23.252; \, P = 0.027 \). Muscle × time: \( F = 4.507, \, df = 2.325, \, 23.252; \, P = 0.010 \). Muscle × group: \( F = 4.384, \, df = 1, \, 10; \, P = 0.063 \). Time × group: \( F = 4.118, \, df = 2.052, \, 20.523; \, P = 0.015 \). Muscle: \( F = 0.692, \, df = 1, \, 10; \, P = 0.456 \). Time: \( F = 3.763, \, df = 2.052, \, 20.523; \, P = 0.021 \). Group: \( F = 1.925, \, df = 1, \, 10; \, P = 0.231 \). Post hoc Helmert contrast for time, 4 wks vs. average of 8 and 12 wks: \( F = 0, \, df = 1, \, 10; \, P = 0.515 \). Post hoc Helmert contrast for time, 8 wks vs. 12 wks: \( F = 0.455, \, df = 1, \, 10; \, P = 0.612 \).

FIGURE 1 EMG events recorded from the masseter muscle. Preinjection (horizontal axis) and postinjection (vertical axis) in the control group (open squares) and in the botulinum toxin group (filled circles). (a) Four weeks. The number of bruxism events was roughly the same pre- and postinjection in the control group and was lower postinjection in the botulinum toxin group. (b) Eight weeks. (c) Twelve weeks.
significantly. However, the main effect for time was each averaged over the four times, did not differ group and the mean for the saline-injected group, 0.269), i.e., the mean for the botulinum-injected 0.362) nor was the main effect for group (P = 0.047). Muscle: F = 1.445, df = 1, 10; F = 0.257. Time: F = 6.837, df = 2.342, 23.418; P = 0.003. Group: F = 0.489, df = 1, 10; P = 0.500. Post hoc Helmert contrast for time, preinjection vs. postinjection: F = 13.828, df = 1, 10; P = 0.004. Post hoc Helmert contrast for time, 4 wks vs. average of 8 and 12 wks: F = 2.284, df = 1, 10; P = 0.162. Post hoc Helmert contrast for time, 8 wks vs. 12 wks: F = 2.331, df = 1, 10; P = 0.158.

masseoter-saline mean (2.47 ± 1.23) whereas the temporalis-botulinum mean (2.22 ± 1.54) did not differ (P = 0.483) from the temporalis-saline mean (1.85 ± 1.51). In other words, bruxism events in the masseter muscle decreased significantly in the botulinum toxin injection group after the injection. In the temporalis muscle, bruxism events did not differ between the groups or among the recording times.

When the data were analyzed using 10% MVC as the threshold for an EMG bruxism event, the number of events using the lower threshold increased compared with the 20% MVC threshold, but the statistical conclusions remained the same (Table 3).

**Table 3** Number of EMG bruxism events per hour during sleep using the 10% MVC criterion

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Group</th>
<th>Before</th>
<th>4 wks</th>
<th>8 wks</th>
<th>12 wks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Masseter</td>
<td>Botulinum</td>
<td>4.97 ± 2.33</td>
<td>0.59 ± 0.57</td>
<td>1.33 ± 1.10</td>
<td>1.70 ± 0.91</td>
</tr>
<tr>
<td></td>
<td>Saline</td>
<td>4.70 ± 1.17</td>
<td>4.13 ± 1.31</td>
<td>4.12 ± 0.89</td>
<td>4.40 ± 1.52</td>
</tr>
<tr>
<td>Temporalis</td>
<td>Botulinum</td>
<td>4.24 ± 2.25</td>
<td>4.40 ± 3.15</td>
<td>4.26 ± 1.67</td>
<td>4.83 ± 2.62</td>
</tr>
<tr>
<td></td>
<td>Saline</td>
<td>3.26 ± 1.77</td>
<td>2.96 ± 1.27</td>
<td>3.31 ± 1.84</td>
<td>3.59 ± 2.09</td>
</tr>
</tbody>
</table>

Muscle × time × group: F = 5.186, df = 1.783, 17.834; P = 0.019, Greenhouse-Geisser. Muscle × time: F = 7.956, df = 1.783, 17.834; P = 0.004. Muscle × group: F = 10.628, df = 1, 10; P = 0.009. Time × group: F = 5.332, df = 2.342, 23.418; P = 0.003. Group: F = 0.489, df = 1, 10; P = 0.500. Post hoc Helmert contrast for time, preinjection vs. postinjection: F = 13.828, df = 1, 10; P = 0.004. Post hoc Helmert contrast for time, 4 wks vs. average of 8 and 12 wks: F = 2.284, df = 1, 10; P = 0.162. Post hoc Helmert contrast for time, 8 wks vs. 12 wks: F = 2.331, df = 1, 10; P = 0.158.

**Table 4** Subjective symptoms of bruxism

<table>
<thead>
<tr>
<th>Group</th>
<th>Before</th>
<th>4 wks</th>
<th>8 wks</th>
<th>12 wks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Botulinum</td>
<td>1.75 ± 0.91</td>
<td>0.75 ± 0.70</td>
<td>0.81 ± 1.08</td>
<td>0.61 ± 0.64</td>
</tr>
<tr>
<td>Saline</td>
<td>1.89 ± 0.71</td>
<td>1.33 ± 1.08</td>
<td>1.47 ± 0.93</td>
<td>1.39 ± 1.00</td>
</tr>
</tbody>
</table>

Time × group: F = 1.105, df = 3, 30; P = 0.362. Group: F = 1.368, df = 1, 10; P = 0.269. Time: F = 8.315, df = 3, 30; P < 0.001. Post hoc Helmert contrast for time, preinjection vs. postinjection: F = 11.844, df = 1, 10; P = 0.006. Post hoc Helmert contrast for time, 4 wks vs. average of 8 and 12 wks: F = 0.065, df = 1, 10; P = 0.804. Post hoc Helmert contrast for time, 8 wks vs. 12 wks: F = 1.092, df = 1, 10; P = 0.321.

**DISCUSSION**

The first main finding was that the number of suprathreshold EMG bruxism events during sleep significantly decreased in the masseter muscle in the botulinum toxin injection group compared with the saline injection group. The number of bruxism events was markedly reduced at 4 wks after botulinum toxin injection and maintained for the 12-wk duration of our study.

The second main finding was that, in contrast to the decrease in suprathreshold EMG bruxism events in the masseter muscle injected with botulinum toxin, the suprathreshold EMG bruxism events in the temporalis muscle were remarkably constant. We interpret these findings as consistent with bruxism being centrally mediated. Although we believe that bruxism is centrally mediated, its effects are manifest in the peripheral muscle activity, and this study suggests that such peripheral activity can be effectively reduced by botulinum toxin.

We interpret the results of the three parts of Figure 1a–c as suggesting that minimal, if any,
contractions of facial muscles or other muscles were detected after the botulinum toxin injection and that in one subject some small part of the masseter may not have received sufficient botulinum toxin.

Our third main finding was that the subjective scores from both the botulinum toxin and the saline injection groups decreased, hence both groups believed that their bruxism had been reduced. This finding illustrates the need for a control group, the need for an objective measurement of bruxism, and suggests that the blinding of the subjects to the injection material may have been successful.

Botulinum toxin injection for patients with bruxism, first described by Van Zandijcke and Marchau, has been reported to be an effective treatment. However, all of the previous studies used subjects’ subjective symptoms as the only indicator to assess the therapeutic effect of botulinum toxin on bruxism and often failed to include a control group (Table 1). We used EMG analysis as an objective measure for evaluating the nocturnal bruxism, measured the temporalis muscle as an objective indicator to assess the therapeutic effect of botulinum toxin on bruxism, and often failed to include a control group (Table 1). We used EMG analysis as an objective measure for evaluating the nocturnal bruxism, measured the temporalis muscle as a within-subject control, and included a group where the injection was saline. Thus, we believe that we have improved on the controls used previously.

Some studies injected botulinum toxin in both the masseter and temporalis muscles for severe bruxism patients whereas other studies reported that masseter muscle injection alone could reduce nocturnal bruxism effectively. Our study showed that the bruxism activity was significantly reduced after botulinum toxin injection in the masseter muscle, but the activity still persisted in the temporalis muscle. It is expected that injection into the two masticatory muscles would be more effective than into one muscle. However, the unfavorable result of an “hourglass deformity” of the facial region resulting from depression of the temporal area when an equivalent dose of botulinum toxin was injected in the temporalis was reported in 28% of the subjects in a previous study. Further study about the effective dose for the temporalis muscle to decrease bruxism with minimal side effects should be considered.

The injected botulinum toxin is bound to the cholinergic motor nerve terminal, absorbed into the cytoplasm of the nerve terminal, and blocks acetylcholine release. Thus, injection of botulinum toxin into a muscle causes muscle paralysis. We speculate that decreased action potentials directly, not muscle atrophy, reduced bruxing events in the masseter muscle.

The previous studies reporting the effect of botulinum toxin on bruxism used the equivalent dose of Dysport units ranging from ~45 to 150 units. Our dose was in the middle of this range. All doses were apparently effective (Table 1).

Four limitations should be made clear. First, several complications after botulinum toxin injection into the masticatory muscles have been reported, including mastication difficulties, muscle pain, speech disturbance, and unnatural facial appearance. But these complications are reported to be transient, usually lasting from 1 to 4 wks after injection. Immunologic responses such as allergic skin reactions or formation of antibodies can occur in a small percentage of subjects. However, we did not observe any of these problems in our sample.

Second, the duration of the effects was narrowly focused because the study ended after 12 wks. Because our main focus was on a well controlled study of the physiologic effects of botulinum toxin on the nocturnal bruxing events, the design did not include the short-term (<4 wks) and very long-term (>12 wks) effects. The data in Figure 1 suggest that the effect of the botulinum toxin on the EMG activity in the injected masseter was robust in the time frame we studied. Thus, it did not reveal the longer term effects as the muscles regain their innervation. Again, our purpose was to investigate, in a way we believe was carefully controlled, the effects on bruxism when the botulinum toxin had paralyzed the muscle. When botulinum toxin is injected to a striate muscle, paresis occurs after 2–5 days and lasts 2–3 mos before it gradually decreases. Complete functional recovery is observed 6 mos after the initial injection. In a study that evaluated the effect of botulinum toxin A on severe bruxism patients, the mean total duration of response was 19.1 ± 17.0 wks (range, 6–78 wks). If the inability to activate the masseter muscle for several months might be able to modify the bruxing habit, reduced bruxism would be maintained after the effective period of botulinum toxin. Further study will be needed to evaluate the long-term effect of botulinum toxin on nocturnal bruxism.

Third, the questionnaire for subjective bruxism is not validated. Although the bruxism questionnaire is reliable, and the questions seem to have face validity in that they directly ask about bruxism, there is no validated scale for measuring bruxism and its severity and no gold standard for diagnosing bruxism outside the sleep laboratory. A recent review failed to find any validated questionnaires. Moreover, the absence of an interaction between time and group for our questionnaire data, which suggests that the two groups did not respond differently, suggests that developing validated index might be difficult.

Fourth, both the number of subjects, six per group, and the severity of bruxism were modest. Our sample size, however, was sufficient to reveal a statistically significant postinjection decrease of bruxism events in the muscles that were injected...
with botulinum toxin, the main aim of the study. The subjects were recruited from those complaining of bruxism, not from patients seeking treatment in clinic; moreover, the numbers of EMG bruxism events per hour (1–3 for the 20% MVC criterion and 2–8 for the 10% MVC criterion) were modest. Thus, our subjects were not severe bruxers, yet a significant effect was demonstrated.

CONCLUSIONS

Our results showed that the injection of botulinum toxin in the masseter muscle reduced the number of bruxism events during sleep, most likely mediated through its effect on muscle tone rather than central nervous system. Botulinum toxin injection can be used as an effective treatment for nocturnal bruxism.

REFERENCES


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