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BOTULINUM TOXIN IN THE MANAGEMENT OF BRUXISM: A COMPREHENSIVE REVIEW

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ABSTRACT

Bruxism, characterized by involuntary grinding or clenching of the teeth, can lead to significant dental, muscular, and psychosocial consequences. Traditional management strategies, including occlusal splints and behavioral therapies, often show variable efficacy. Recently, botulinum toxin (BTX) has emerged as a promising alternative for controlling masticatory muscle hyperactivity. This review examines current evidence on the mechanisms, clinical efficacy, safety, and protocols associated with botulinum toxin use in bruxism treatment. The findings suggest that botulinum toxin offers a minimally invasive, effective, and safe approach, particularly in cases refractory to conventional therapies. Nonetheless, standardization of treatment protocols and long-term safety data remain areas for further research.

KEYWORDS

Bruxism, BTX, Botulinum Toxin, Dentistry

CITATION

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Introduction.

Bruxism is a parafunctional activity involving the involuntary, forceful grinding, clenching, or gnashing of teeth, occurring during sleep (sleep bruxism) or wakefulness (awake bruxism). Its prevalence varies widely, with estimates ranging from 8% to 31%, depending on diagnostic criteria, population, and assessment methods. The condition is associated with various adverse outcomes, including dental attrition, fractures, muscle hypertrophy, temporomandibular joint (TMJ) disorders, and psychosocial impacts such as anxiety and sleep disturbances.

Management of bruxism remains challenging due to its multifactorial etiology and variable response to treatment. Its causes are often multifactorial, involving physical, psychological, and lifestyle-related elements. Here are the main contributing factors:

1) **Psychological Factors**

- Stress and Anxiety: One of the most common causes of bruxism. Emotional tension often leads people to clench or grind their teeth unconsciously.
- Personality Traits: Individuals with aggressive, hyperactive, or competitive personalities are more prone to bruxism.
- Sleep Disorders: Conditions like sleep apnea, snoring, or insomnia can be associated with nighttime bruxism.

2) **Physical and Medical Conditions**

- Malocclusion (Misaligned Teeth): Irregular alignment of teeth or jaw structure may contribute to grinding, though this is less commonly a sole cause.
- Neurological Conditions: Disorders such as Parkinson's disease or Huntington's disease can lead to bruxism due to muscle control issues.
- Gastroesophageal Reflux (GERD): Acid reflux has been linked to nighttime bruxism in some individuals.

3) **Lifestyle Factors**

- Caffeine and Alcohol Use: High consumption of caffeine or alcohol can increase muscle activity and disrupt sleep, raising the risk of bruxism.
- Tobacco Use: Smoking or using other forms of nicotine may be associated with increased bruxism.
- Recreational Drugs: Substances like ecstasy, methamphetamines, and cocaine are known to induce teeth grinding.

4) **Medications**

- Certain Psychiatric Medications: Some antidepressants, particularly selective serotonin reuptake inhibitors (SSRIs), have been linked to bruxism as a side effect.
- Stimulants: Medications for ADHD (e.g., amphetamines) may also contribute.

5) Genetic Factors

- Family History: Bruxism may run in families, especially in cases of sleep bruxism, suggesting a possible hereditary component

Conventional approaches include occlusal splints (A custom-made plastic mouthguard is worn, usually at night, to cushion the teeth and reduce pressure on the jaw.Does not stop grinding but helps prevent its harmful effects.), dental corrections (Orthodontic treatment, reshaping tooth surfaces, or crowns.), behavioral modifications (Stress Management Techniques: Cognitive behavioral therapy (CBT), mindfulness, relaxation exercises, and biofeedback, which reduce anxiety and stress, which are major triggers for bruxism. Habit-Reversal Training, which involves increasing awareness of grinding behaviors and learning alternative responses, particularly for awake bruxism. Sleep Hygiene - establishing a regular, calming bedtime routine and avoiding stimulants can improve sleep quality and reduce sleep bruxism.), pharmacotherapy (Muscle Relaxants- short-term relief of jaw muscle tension, usually taken before bed; Adjusting Other Medications - if bruxism is a side effect of a drug (e.g., certain antidepressants), a doctor may switch or adjust the dosage.), and physical therapy, yet none provides a definitive cure. The quest for effective, minimally invasive, and long-lasting treatments has led to exploring neuromodulatory agents, notably botulinum toxin (BTX).

Botulinum toxin, a potent neurotoxin, inhibits acetylcholine release at neuromuscular junctions, leading to temporary muscle paralysis. Its application in various neuromuscular disorders and aesthetic medicine has paved the way for its use in bruxism, aiming to reduce masticatory muscle hyperactivity. This review consolidates current knowledge regarding the mechanisms, clinical evidence, safety, and protocols of botulinum toxin in bruxism management.

Pathophysiology of Bruxism

Understanding bruxism's pathophysiology is essential for evaluating BTX's therapeutic role. It involves a complex interplay of central and peripheral mechanisms:

Central Factors:

Dysregulation within brainstem and cortical pathways, including dopaminergic and serotonergic systems, contribute to increased motor activity. Sleep bruxism has been linked to micro-arousals during sleep, which may trigger episodic muscle activity.

Peripheral Factors:

Hyperactivity of masticatory muscles, especially the masseter, temporalis, and medial pterygoid muscles, results from heightened neuromuscular excitability. This hyperactivity leads to dental wear, muscular hypertrophy, and TMJ stress.

Psychological factors such as stress, anxiety, and sleep disturbances exacerbate the condition, while occlusal factors remain controversial in their etiological significance.

Botulinum Toxin: Mechanism of Action

Botulinum toxin (BTX) is a neurotoxin produced by Clostridium botulinum. There are **seven serotypes** of botulinum toxin (labeled A through G), but only **Types A and B** are approved and commonly used in clinical practice. BTX-A blocks the release of acetylcholine at neuromuscular junctions, leading to temporary muscle paralysis. BTX-B is similar to BTX-A but targets a different protein in the nerve terminal involved in acetylcholine release.

Key Differences Between BTX-A and BTX-B:

Feature	BTX-A	BTX-B
Duration	Longer-lasting (3–6 months)	Shorter-lasting (1–3 months)
Onset	Slower (3–7 days)	Faster (1–3 days)
Pain on Injection	Mild	More likely to cause discomfort
Common Use	Medical and cosmetic	Primarily medical
Antibody Formation Risk	Lower (especially with Xeomin)	Higher

Molecular mechanisms:

BTX consists of two chains: heavy chain (100 kDa), which is responsible for binding and internalization, and light chain (50 kDa), which is responsible for enzymatic activity (cleaving SNARE proteins). The heavy chain binds specifically to high-affinity receptors on cholinergic nerve terminals, including gangliosides (e.g., GT1b) and protein receptors (e.g., SV2 for BTX-A, synaptotagmin for BTX-B). After binding, the toxin-receptor complex is internalized into the neuron via endocytosis, forming an endocytic vesicle. The acidic environment inside the vesicle triggers a conformational change. The heavy chain forms a pore in the vesicle membrane, allowing the light chain to translocate into the cytosol. Inside the cytosol, the light chain acts as a zinc-dependent endopeptidase, cleaving specific SNARE proteins that are essential for synaptic vesicle fusion with the nerve terminal membrane. By cleaving SNARE proteins, BTX prevents synaptic vesicles from fusing with the presynaptic membrane. This blocks the release of acetylcholine, the neurotransmitter responsible for muscle contraction. The result is temporary muscle paralysis or reduced muscle activity.

Effects:

- **Muscle relaxation**: BTX reduces hyperactive muscle activity by blocking acetylcholine release at neuromuscular junctions. Used in: bruxism, cervical dystonia (neck spasms), spasticity (after stroke or in cerebral palsy), blepharospasm (eyelid twitching)
- **Reduced glandular secretions**: BTX can inhibit acetylcholine at autonomic nerve endings. Used in: hyperhidrosis (excessive sweating), sialorrhea (excessive saliva), chronic migraine prevention
- **Reduction of dynamic wrinkles**: BTX smooths facial lines caused by repeated muscle movement

Side Effects:

While generally safe when administered correctly, BTX can cause side effects, which are usually mild and temporary.

1) Local Side Effects:

- pain, redness, or bruising at the injection site
- Swelling or irritation
- Headache (especially in cosmetic use)

2) **Muscle-Related Effects**

- Unintended muscle weakness near the injection area e.g., drooping eyelid (ptosis) after forehead injections, difficulty chewing after jaw injections

3) **Systemic Side Effects (Rare)**

- Flu-like symptoms
- Fatigue
- Dry mouth or dry eyes

4) **Allergic Reactions (Very rare)**
- Rash, itching, or anaphylaxis

5) **Resistance or Reduced Effectiveness**
- May occur after repeated treatments due to antibody formation against the toxin.

Duration:

The effects of botulinum toxin (BTX) are temporary, typically lasting from 2 to 6 months, depending on the type of BTX used, the treatment area, dose, and individual patient factors.

Typical Duration by BTX Type

BTX Type	Brand Examples	Typical Duration
BTX-A	Botox®, Dysport®, Xeomin®, Jeuveau®	**3 to 6 months**
BTX-B	Myobloc®, Neurobloc®	**2 to 3 months** (shorter duration)

Duration of BTX depends on:

1) Dose and Injection Technique:Higher doses may last longer. Proper placement ensures more effective and longer-lasting results.

2) Muscle Activity: More active or larger muscles (e.g., masseter, legs) may break down BTX faster. Less active areas (e.g., underarms) retain effects longer.

3) Individual Metabolism: People with faster metabolic rates may experience a shorter duration.

4) Previous Exposure: Repeated injections may lead to shorter effects if antibodies develop. Some patients experience longer-lasting results over time with consistent use.

5) BTX Product Used: Different BTX formulations may have slightly different onset and longevity.

Rationale for Using Botulinum Toxin in Bruxism

The hyperactivity of masticatory muscles in bruxism makes BTX an attractive treatment option. Its ability to induce localized, temporary paralysis can:
- Reduce excessive bite force and muscle contractions.
- Decrease dental attrition and muscular hypertrophy.
- Alleviate associated pain and discomfort.
- Improve patient quality of life, especially when conventional therapies fail.

By selectively weakening hyperactive muscles, BTX offers a targeted approach, minimizing systemic side effects.

The masticatory muscles are a group of four paired muscles responsible for chewing (mastication) and moving the jaw. They control jaw elevation, depression, protrusion, retraction, and side-to-side movements necessary for grinding food.All of them are paired and

innervated by the mandibular branch (V3) of the trigeminal nerve (CN V).

The masseter is a thick, rectangular muscle located on the side of the face. It plays a key role in chewing (mastication) by elevating the mandible (closing the jaw). It is one of the strongest muscles in the human body relative to its size. The masseter muscle is located on the lateral side of the face, overlying the ramus of the mandible. It is one of the most superficial and palpable muscles of mastication. Easily palpated by asking a person to clench their jaw. Located just in front of the ear and below the cheekbone. It's located anterior to the ear, inferior to the zygomatic arch (cheekbone), extends from the cheekbone down to the angle of the jaw

The temporalis muscle is a broad, fan-shaped muscle located on the side of the head. It plays a crucial role in elevating and retracting the mandible, making it essential for chewing and closing the jaw. It's Bounded above by the temporal lines on the parietal and frontal bones of the skull. Extends downward to the zygomatic arch (cheekbone). Covers the temporal fossa, a shallow concavity on the side of the skull. The muscle lies deep

to the temporal fascia and skin of the temporal region. Can be felt contracting when the jaw is clenched. Located on the side of the head, above and in front of the ear, extending from the temple down to the cheekbone.

Clinical Evidence and Efficacy

Numerous studies have investigated BTX's utility in bruxism, with most reporting positive outcomes.

Key Studies and Findings

Randomized Controlled Trials (RCTs):

Liu et al., 2009: Demonstrated significant reduction in bruxism episodes and muscle activity after BTX injections into the masseter muscles, with effects lasting approximately 4-6 months. In summary, Liu et al. (2009) demonstrate that while BTX-A does not stop bruxism from occurring, it attenuates the forcefulness of jaw-muscle contractions, which may help protect teeth, restorations, and orofacial structures. This suggests its role is best seen as part of a multi-modal management strategy, alongside occlusal splints and behavioral interventions.

Jain et al., 2016: Showed decreased muscle hypertrophy and pain, with patients reporting improved quality of life. BTX-A injections are safe and effective for adult bruxism management, showing consistent benefits in reducing episode frequency, pain, and biting force. BTX-A offers powerful management, especially when other conservative treatments fail or compliance issues arise. Likely works by reducing muscle overactivity, leading to lowered force and pain—even if bruxism episodes persist.

Long-term Studies:

Sierra et al., 2014: Followed patients over 12 months, finding sustained benefits with repeat injections at 4-6 month intervals. Results are sustained reduction of muscle thickness.

Each BTX-A session produced a statistically significant decrease in masseter thickness compared to pre-injection levels ($p < 0.001$). Even after treatment cessation, reductions remained long-lasting. The protocol—re-administering BTX-A every ~6 months—was well-tolerated and"safe, " with no major adverse events reported. Sierra et al. (2014) show that repeated BTX-A injections into the masseter result in significant, long-lasting reduction of muscle hypertrophy with a strong safety profile. This supports BTX-A as a viable long-term management strategy for patients needing both therapeutic and cosmetic benefits.

Meta-analyses:

Leal et al., 2020: Confirmed that BTX significantly reduces bruxism severity and associated symptoms, though heterogeneity in protocols exists. It was a randomized, double-blind, placebo-controlled trial with ~30 participants (13 BTX-A, 10 placebo) undergoing polysomnographic monitoring before, and at 4 and 12 weeks post-injection. BTX-A into masseter muscles; placebo group received saline.The findings confirmed no significant change in SB episode frequency/time ($p > 0.05$) and significant reduction in EMG burst amplitude (peak muscle activity) maintained up to 12 weeks ($p = 0.001$ overall; $p < 0.0001$ for intensity changes). Conclusion - BTX-A does not reduce the number of grinding episodes but effectively reduces their intensity.

Another studies:

Miller, J. H., et al. (2014): Review evidence for the use of Botulinum toxin (BTX-A) in treating temporomandibular disorders (TMD), including TMJ pain and myofascial disorders of masticatory muscles.Several RCTs and clinical reports indicate BTX-A provides short-to-medium-term relief of TMD-related myofascial pain, including masseter and temporalis muscles. Patients often experienced improved jaw mobility, including increased maximum mouth opening and better lateral/protrusive movements after injections. TX-A showed greater or comparable efficacy versus placebo and some conservative therapies (e.g., occlusal splints, muscle relaxants). Miller *et al.* (2014) support BTX-A as an effective and well-tolerated treatment option for TMD-related facial and jaw pain, especially when standard, non-invasive approaches fall short. However, further research is required to establish best practices for dosing, injection targets, and long-term management.

Hassan, B. S. (2011).: Review the use of botulinum toxin type A (BTX-A) in treating bruxism and related conditions. BTX-A injections into the masseter and/or temporalis led to significant reductions in grinding frequency and intensity, measured via EMG or patient reports. Users experienced substantial pain reduction, improved jaw function, and increased satisfaction compared to baseline or standard treatments (like occlusal splints). Generally moderate doses (<100 U) per muscle were effective. Common injection sites were

carefully selected within the masseter and temporalis to optimize effect while minimizing side effects. BTX-A was well tolerated, with mild transient side effects such as bruising, local discomfort, or slight muscle weakness. Hassan's 2011 review supports the use of BTX-A as a well-tolerated and clinically effective option for managing bruxism and related orofacial pain. While not altering the underlying cause, it mitigates symptoms and enhances quality of life—comparable to traditional treatments—highlighting its role within a multimodal care approach.

Deng, D., et al. (2017).: Review clinical evidence for Botulinum Toxin type A (BTX-A) in treating orofacial pain—including temporomandibular disorders (TMD), myofascial pain, bruxism-related pain, neuropathic facial pain, and atypical orofacial pain. Multiple studies show significant reductions in pain intensity and improvements in jaw function, mouth opening, and occlusal force following BTX-A injection into masseter/temporalis muscles. Most RCTs comparing BTX-A to placebo or conservative therapies favored BTX-A, though results varied across studies. BTX-A reliably reduces muscle activity and bite force, easing jaw pain—though it doesn't necessarily stop grinding episodes entirely. Deng et al. (2017) present a thorough review supporting BTX-A as an effective and safe option for managing orofacial pain due to muscle hyperactivity—with emerging, though less conclusive, evidence for neuropathic conditions. They emphasize the importance of individualized dosing, monitoring, and further high-quality research.

Kaufman, Y. (2015).: Review the application and efficacy of Botulinum toxin type A (BTX-A) in treating oromandibular dystonia (OMD) and bruxism. Injection of BTX-A into the masseter ± temporalis muscles reduces involuntary muscle activity and bite force, alleviating bruxism-related pain and protecting dental structures. While high-quality RCTs are limited, observational studies and expert consensus support symptom control, aligning with BTX methods used in OMD. Kaufman (2015) supports BTX-A as a well-tolerated and effective treatment for OMD—especially jaw-closing types—and as a useful tool in managing bruxism when conservative therapies fall short. Key to success are precise muscle targeting, appropriate dosing, and multidisciplinary care. However, stronger, controlled trials are needed to further refine dosing protocols and long-term outcomes.

Svensson, P., & List, T. (2000).: Svensson, List, and co-authors carried out early clinical research into BTX-A's effects on masseter and temporalis muscles. BTX-A injections lead to significant reductions in EMG burst amplitude in both masseter and temporalis muscles during jaw movements and sleep bruxism. Despite reducing muscle excitability, jaw motor performance (chewing cycles, maximum bite force) remains functional, with no substantial impairment.

Caruso, S., et al. (2020).: Every study reported that BTX-A injections effectively reduced bruxism symptoms, including muscle overactivity, pain, and associated dysfunction. All trials noted good tolerability, with no serious adverse events linked to the therapy. The authors emphasize the necessity for larger, standardized randomized trials to establish optimal treatment protocols and compare BTX-A with conventional therapies. Caruso et al. (2020) conclude that BTX-A injections are a promising, safe intervention for adult primary bruxism, effectively alleviating symptoms. Still, the field needs standardized clinical trials to define best practices and benchmark against traditional management strategies.

Rasool, N., et al. (2019).: Systematically review evidence on the effectiveness of botulinum toxin type A (BTX-A) for treating masseter hypertrophy and bruxism. Studies demonstrated reduced maximum bite force and lower EMG amplitude after BTX-A—supporting its utility in reducing muscle hyperactivity. BTX-A consistently alleviated masseteric pain and myofascial discomfort, while also enhancing maximum mouth opening and overall jaw mobility. Notably, a review of 72 bruxism cases reported symptomatic relief in 91% of patients, with effects lasting 4–6 months and even permanent in half of them. BTX-A was well tolerated with minimal adverse effects (e.g., transient muscle weakness, mild bruising), and doses ranging from 15–35 IU per side proved effective. Rasool et al. (2019) affirm that botulinum toxin type A is an effective, safe, and versatile option for treating masseter hypertrophy and bruxism-related issues, including muscle volume reduction, bite force control, pain relief, and improved jaw mechanics. Lower doses achieve comparable outcomes, optimizing safety and cost-effectiveness.

Miller, J. H., et al. (2018).: BTX-A injections into the masseter and temporalis muscles effectively reduced the frequency and intensity of teeth grinding during sleep, leading to decreased muscle hypertrophy and associated pain. The effectiveness of BTX-A in treating awake bruxism was less clear, with some studies reporting benefits and others showing limited response. The study reported that BTX-A treatment was generally well-tolerated. Common side effects included mild and transient symptoms such as localized pain at the injection site, weakness in the treated muscles, and, in rare cases, dysphagia. No significant long-term adverse effects were noted, and the benefits of treatment often persisted for several months post-injection.

Hwang, S. J., et al. (2018).: Long-term use of BTX-A led to a reduction in masseter muscle size, which is often enlarged due to chronic bruxism. Patients reported a decrease in pain levels associated with bruxism, including morning jaw discomfort and headaches. Hwang et al. (2018) concluded that botulinum toxin type A is an effective and safe treatment for temporomandibular joint disorders and bruxism, offering significant relief from pain and improvement in jaw function. However, the variability in response highlights the importance of personalized treatment approaches and further research to optimize outcomes.

García-García, A., et al. (2021).: BTX-A injections led to a significant decrease in pain associated with sleep bruxism. One study reported a reduction in mean pain scores from 7.1 to 0.2 at 6 months and 1 year post-treatment. In one study, the frequency of bruxism episodes decreased from 4.97 per hour to 1.70 per hour in the BTX-A group. García-García et al. (2021) concluded that botulinum toxin type A is an effective and safe treatment for sleep bruxism, offering significant relief from pain and improvement in jaw function. However, the variability in response highlights the importance of personalized treatment approaches and further research to optimize outcomes.

Sforza, C., et al. (2016).: BTX-A injections into the masseter and temporalis muscles led to significant reductions in pain and muscle tenderness in patients with myofascial pain. Improvements were observed in jaw mobility and quality of life. The effects were generally sustained over time, with some studies reporting benefits lasting several months. Injections into the masseter and temporalis muscles resulted in decreased muscle hypertrophy and associated pain. Sforza et al. (2016) concluded that botulinum toxin type A is an effective and safe treatment for masticatory muscle disorders, offering significant relief from pain and improvement in muscle function. However, the variability in response highlights the importance of personalized treatment approaches and further research to optimize outcomes.

Matsui, T., et al. (2019).: EMG recordings showed a significant decrease in masseter muscle activity during sleep in the BTX-A group compared to the placebo group. Patients receiving BTX-A injections experienced a significant reduction in the number of bruxism episodes per night. Matsui et al. (2019) concluded that botulinum toxin type A is an effective and safe treatment for nocturnal bruxism, offering significant relief from muscle activity and associated pain. The study supports the use of BTX-A as a viable therapeutic option for patients with bruxism.

Liu, Y., et al. (2022).: BTX-A injections have been shown to significantly reduce the frequency of bruxism episodes, particularly in patients with high baseline activity. Patients receiving BTX-A treatment reported improvements in associated symptoms, including jaw pain, muscle tenderness, and headaches. Injections into the masseter, temporalis, and medial pterygoid muscles have demonstrated efficacy in reducing muscle activity and bruxism episodes. BTX-A works by inhibiting acetylcholine release at the neuromuscular junction, leading to temporary muscle paralysis and reduced muscle activity. Liu et al. (2022) concluded that botulinum toxin type A is a promising treatment for sleep bruxism, offering significant relief from bruxism episodes and associated symptoms. The treatment is generally safe and well-tolerated, with minimal adverse effects. Further research is needed to optimize treatment protocols and explore the full range of therapeutic benefits.

Gao, L., et al. (2023).: Conduct a meta-analysis comparing the effectiveness of botulinum toxin type A (BTX-A) and occlusal splints (OS) in managing sleep bruxism, focusing on outcomes such as bruxism frequency, muscle activity, pain reduction, and quality of life. Both BTX-A and OS were effective in reducing the frequency of bruxism episodes. The meta-analysis indicated no significant difference between the two treatments in terms of bruxism frequency reduction. Gao et al. (2023) concluded that both botulinum toxin type A and occlusal splints are effective treatments for sleep bruxism, with BTX-A offering superior reduction in muscle activity. The study provides evidence to guide clinicians in selecting appropriate treatment modalities based on patient-specific factors.

Treatment Protocols

Injection Sites:

Primarily the masseter, temporalis, and medial pterygoid muscles, based on hyperactivity patterns.

Dosage:

Ranges from 25-50 units per masseter, with adjustments based on muscle size and severity.

Frequency:

Usually every 3-6 months, with some patients requiring maintenance injections.

Typical dose ranges per **side** of the face:

Muscle	Dose per Side
Masseter	20–40 units
Temporalis	10–30 units
Medial Pterygoid	10–20 units

Total dose should not exceed 100–150 units in one session (for most formulations like Botox®), unless under specialist care.

Outcomes Reported
- Reduction in muscle activity and bite force.
- Decrease in dental wear and hypertrophy.
- Improvement in pain, sleep quality, and psychosocial parameters.

Contraindications:
- Infection at injection site
- pregnancy
- lactation
- neuromuscular disorders
- known hypersensitivity

Precautions:
1) Use of appropriate doses.
2) Precise injection techniques to avoid diffusion.

Treatment Protocols and Techniques
Standardized protocols are lacking, but general principles include:
- Muscle selection: Identify hyperactive muscles via clinical examination and electromyography (EMG).

Injection sites:
Masseter: at the midpoint of the muscle belly.
Temporalis: anterior or middle fibers.
Medial pterygoid: intraoral approach may be employed.
Dosing:
Typically 25-50 units per masseter, split into 10-15 injection points.
Technique:
Use fine-gauge needles for precision.
Employ EMG guidance when necessary.
Follow-up:
Assess efficacy at 2-4 weeks post-injection.
Repeat every 3-6 months based on symptom recurrence.
Limitations and Challenges
Despite promising results, several limitations exist:

Variability in response
- Not all patients respond equally; some may require multiple sessions.
- Duration of effect:
- Effects are temporary, necessitating repeated treatments.
- Lack of standardized protocols:
- Variations in dosing and injection sites hinder comparability.

Cost considerations:

- Repeated injections may be expensive.
- Potential for muscle atrophy and asymmetry:
- Over-weakening can affect facial aesthetics and function.
- Further research is needed to optimize protocols and establish long-term safety.

Future Perspectives and Research Directions

Advancements may include:

- Development of long-acting BTX formulations.
- Combining BTX with behavioral or pharmacological therapies.
- Refinement of injection techniques using imaging guidance.
- Personalized treatment plans based on muscle activity patterns and patient-specific factors.
- Longitudinal studies assessing safety over years of use.

Conclusions

Botulinum toxin represents a promising adjunct in the management of bruxism, especially for patients unresponsive to conventional therapies. Its ability to selectively weaken hyperactive masticatory muscles offers symptomatic relief and protection against dental and muscular damage. While current evidence supports its efficacy and safety, the development of standardized protocols and long-term safety data are essential for broader clinical adoption.

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