Botulinum toxin is a polypeptide protoxin synthesized by *Clostridium botulinum* that results in localized reduction of muscle activity by inhibiting acetylcholine release at the neuromuscular junction. In 2004, the US Food and Drug Administration approved its application in the treatment of various medical conditions, such as facial wrinkles, strabismus, cervical dystonia, blepharospasm, and hyperhidrosis. Later, its application extended to improving dental esthetics and gummy smile. It was found to be a safe and effective alternative to medical therapy to treat various head and neck disorders that have a neurologic component. In this review, we will highlight the mechanism of action and therapeutic benefits of botulinum toxin in the management of head and neck disorders.

Van Ermengem first discovered botulinum toxin (BTX) in 19th century. Scott et al. in 1973 performed animal experiments by injecting BTX into extraocular muscles and reported its ability to paralyze a given muscle. BTX is one of the most potent naturally occurring biological poisons. Before its discovery in medicine, it was responsible for many accidental deaths. Its first medical use was in 1980, to treat strabismus. Nine years later, the cosmetic effects of the toxin on wrinkles were noted, but it was only in 2002, after Food and Drug Administration (FDA) approval, that BTX gained widespread popularity as an alternative to cosmetic surgery.

Currently, the clinical indications for BTX are rapidly growing from treatment of overactive skeletal and smooth muscles to management of hypersecretory diseases (e.g., hyperhidrosis), blepharospasm, and painful disorders such as chronic migraine and cervicofacial dystonia. Studies have also shown its application in several dental conditions such as temporomandibular disorders, trigeminal neuralgia, muscular spasm, bruxism, oromandibular dystonia, gummy smile, masseteric hypertrophy, and sialorrhea. This review discusses the therapeutic use of BTX in head and neck disorders.

**BIOCHEMISTRY OF BTX**

Botulinum toxin is isolated from an anaerobic spore-forming bacterium, *Clostridium botulinum*. Chemically, it is a 2-chain metalloprotease composed of heavy and light chains with 8 immunologically distinct serotypes (A, B, C1, C2, D, F, G). All but one (C2) are neurotoxins. Serotype A (BTX type A or onabotulinum toxin A, Botox [Allergan, Parsippany, NJ]) has been the most widely used for a variety of movement and spasticity disorders as well as in cosmetic procedures.

**MECHANISM OF ACTION**

**Neuromuscular blockade**

BTX has a neuromuscular blocking effect that results from inhibiting the exocytosis of acetylcholine from presynaptic nerve terminals. BTX is internalized into the cytosol from the neuromuscular junction by binding to different gangliosides, namely synaptic vesicle –2, synaptotagmin I, or synaptotagmin II. Heavy chains of BTX facilitate uptake of the whole molecule into the cytosol, where light chains cleave soluble N-ethylmaleimide–sensitive factor attachment protein receptor (SNARE) complexes in the motor neuron. SNARE proteins play an important role in the fusion of synaptic vesicles with the presynaptic plasma membrane, resulting in release of the neurotransmitter acetylcholine (Ach). Light chains of BTX types A and E mainly cleave synaptosome-associated protein 25 kDa (SNAP 25), and BTX types B, D, F, and G cleave synaptobrevin, a vesicle-associated membrane protein. BTX type C serotype cleaves SNAP 25, syntoxin, and SNARE proteins. The cleavage effect of light chains of BTX prevents the release of Ach from motor neurons, leading to flaccid paralysis.

**Statement of Clinical Relevance**

Use of botulinum toxin results in localized reduction of muscle activity by inhibiting acetylcholine release at the neuromuscular junction. It was found to be a safe and effective alternative to medical therapy to treat various head and neck disorders that have a neurologic component.
paralysis of muscle fibers (Figure 1). The onset of paralysis occurs 6 hours after BTX injection, and clinical effects are noticed within 24-72 hours. Recovery usually occurs 90 days after BTX injection, but in certain cases the weakening effect continues from 6 weeks to 6 months (median 3-4 months). The degree and period of denervation are influenced by injection dosage.

Aside from blocking the release of the neurotransmitter Ach, botulinum toxin (particularly BTX type A) inhibits the release of local nociceptive neuropeptides such as substance P, calcitonin gene-related peptide, and glutamate, as well as the expression of transient receptor potential vanilloid 1. This process emphasizes the role of BTX-A in inhibiting peripheral sensitization of nociceptive fibers with reduced central sensitization.

PREPARATION
Botulinum toxin is prepared by laboratory fermentation of *C botulinum*, which lyses and liberates the toxin into the culture. The toxin is then harvested, purified, crystallized with ammonium sulfate, diluted with human serum albumin, lyophilized, bottled in vials, and sealed. Each vial of BTX contains 100 U of *C botulinum* type A neurotoxin complex. It retains its potency for 9 months at room temperature (25°C) and for 3 years at refrigerated temperatures (2-8°C).

THERAPEUTIC USES IN HEAD AND NECK DISORDERS
Botulinum neurotoxins have been used for years to treat a wide variety of medical and dental diseases. They were used initially in improvement of facial esthetics but have since gained popularity in pain management and treatment of disorders with accelerated muscle contraction. Table I summarizes the therapeutic uses of botulinum toxin.

Facial pain and neuralgias
The orofacial region is plagued by a number of acute, chronic, and recurrent painful maladies. Chronic pain presents a diagnostic challenge to clinicians. Therefore, diagnosis and management of orofacial pain can be frustrating for clinicians. Recently, BTX therapy has been used in the management of facial pain, mainly in trigeminal neuralgias, myofascial pain, and headache disorders.

Temporomandibular joint disorders
Temporomandibular disorders (TMDs) are a set of craniofacial changes with multifactorial etiology involving the temporomandibular joint (TMJ), masticatory muscles, or musculoskeletal structures in the head and neck region. TMDs can be associated with headache, preauricular pain, neck pain, decreased jaw excursion, jaw locking, noisy jaw movements, clinical findings of joint clicking, tenderness of joint on palpation, and...
malocclusion. Myofascial pain is the most prevalent TMD, characterized by myofascial trigger points that are identified on palpation as discrete foci of hypercontracted areas within the muscle. In myofascial pain, patients mainly report tenderness in the masseter, temporalis, and lateral pterygoid muscles.

Treatment options for TMDs include stabilization splints, pain management, behavioral medicine, physical therapy, and consuming a soft diet. When the symptoms and pain surpass the effectiveness of these techniques, other treatment modalities can be used to treat TMDs. Recently, botulinum toxin injections have been tried to relieve the tenderness associated with TMDs and to restore TMJ function. BTX type A prevents the release of Ach at the neuromuscular junction, effectively reducing transmission from nerve to muscle, thus reducing excessive muscle contractions and myofascial pain. Various clinical trials have evaluated the effectiveness of BTX toxin in reducing myofascial pain. Denglehem injected BTX type A in the masseter and temporalis muscles of 26 patients with chronic pain linked to TMJ disorders and found that patients had a significant decrease in pain and improved jaw movement. The effects were prolonged 3 months after the injection. Pihut et al. conducted a prospective outcome study in 42 patients diagnosed with masseter muscle pain related to TMJ dysfunction and tension-type headache. Patients were given an intramuscular injection of 21 U of BTX type A in the area of the greatest crosssection surface of both masseter bellies, and they had decreased pain in the temporal region bilaterally. Injection techniques for masticatory muscles and dosages of Botox therapy are summarized in Table II. Although BTX therapy is effective at reducing painful muscle contractions and has a longer duration of action than other conventional treatments, it is not preferred as the first line of treatment due to its cost. Other drugs, such as bupivacaine, were found to be equally efficacious and more cost-effective than BTX. Graboski et al. injected 25 U of BTX type A or 0.5 mL of 0.5% bupivacaine in 8 trigger points of 18 patients with myofascial pain syndrome. Both treatments were effective at reducing the duration or magnitude of pain, but due to the high cost of BTX-A, bupivacaine was more cost-effective for myofascial pain syndrome. Kim et al. evaluated the effect of BTX-A in TMD treatment. A total of 21 TMD patients were treated with BTX-A injections in the bilateral masseter and temporalis muscles. Most patients experienced a decrease in clinical manifestations of TMD, including pain relief and improved masticatory function, after the treatment. The authors concluded that BTX-A injections in the masticatory musculature of TMD patients could be considered as a useful option for controlling complex TMD and helping its associated symptoms.

Evidence from recent studies has shown that BTX-A injections cause mandibular bone loss and uncontrolled structural changes in affected and unaffected muscles. Therefore, it has been emphasized that BTX type A injections can be considered as a treatment of choice for complex TMDs but not as a primary option in masseter muscle pain management, and the dose should be kept as small as possible. Although BTX is a promising agent for TMDs, further investigation with large clinical trials is required to test its safety and effectiveness for FDA approval.

**Bruxism.** Bruxism is a movement disorder characterized by grinding and clenching of teeth without a functional purpose. It is characterized by clinical signs and symptoms of tooth grinding or tapping sounds by patients, the presence of tooth grinding at eccentric
### Table II. Masticatory muscles and dose ranges for botulinum toxin injections

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Origin</th>
<th>Insertion</th>
<th>Injection technique</th>
<th>Botox type A</th>
<th>Botox type B</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Temporalis</strong></td>
<td>Temporal fossa</td>
<td>Medial and anterior aspect of coronoid aspect of mandible</td>
<td>Two types of injection, superficial and deep, used to weaken muscle. Superficial performed in upper regions of muscle in fan shape; Deep requires anatomic consideration (more useful).</td>
<td>5-25 U</td>
<td>1000-3000 U</td>
</tr>
<tr>
<td><strong>Masseter</strong></td>
<td>Anterior two-thirds of zygomatic arch and zygomatic process of maxilla</td>
<td>Lateral surface of angle and lower ramus of mandible</td>
<td>Five diffuse injections into masseter muscle are recommended, preferably areas with highest activity on EMG, muscle bulk, and greatest discomfort. Creating a line from the corner of the ala of the nose to the tragus of the ear, all injections into the masseter should be below this line to avoid injection into the pterygoid fossa, which could result in unwanted effects.</td>
<td>20-50 U</td>
<td>1000-3000 U</td>
</tr>
</tbody>
</table>
| **Lateral pterygoid** | Upper head: infratemporal surface of sphenoid bone  
Lower head: lateral surface of lateral pterygoid plate | Pterygoid fovea below coronoid process of mandible and TMJ meniscus       | Extraoral approach: Establish the location of condylar head and insert needle through skin in coronoid notch and advance 45° posteriorly to engage condylar head. Withdraw needle slightly and advance anteriorly and deeper. Patient is asked to mobilize the mandible side to side, and injection is performed once proper position is ascertained. | 5-10 U       | 1000-3000 U  |

(continued on next page)
positions, masseter muscle hypertrophy on voluntary contraction, masticatory muscle stiffness in the morning, hypersensitivity of teeth to cold air, clicking of the TMJ, and tongue and cheek indentation. Common treatment options for bruxism are occlusal interventions, occlusal intraoral appliances, and pharmacologic management with antidepressants, muscle relaxants, and dopamine agonists. Recently, botulinum neurotoxin has proven to be promising in alleviating the symptoms of bruxism.

Chikhani et al. have suggested that intramuscular injections of BTX can reestablish the balance between closing and opening muscles, relieve pain, treat masseteric hypertrophy with improvement of the facial outline, and recover the normal kinetics of TMJs. Some patients experienced a “fixed” smile for 6 to 8 weeks as a side effect, which could have been due to slight diffusion of the BTX to superficial muscles of the face. More studies on larger populations are required to test the effectiveness of BTX on bruxism. Studies have also suggested that BTX injections cause mandibular bone loss and uncontrolled structural changes in the masseter and temporalis muscles. In daily clinical practice, injections of BTX in the masseter and temporalis muscles are an efficient treatment for bruxism, but they should be used with caution and the dose should be kept to a minimum to avoid structural changes in the muscles injected.

Zhang et al. evaluated the occlusal force and therapeutic efficacy of the masseter muscles after intramuscular injection of BTX-A for the treatment of TMD and bruxism. Thirty TMD patients associated with bruxism were randomized into 3 groups (n = 10 in each group) and treated either with bilateral intramuscular injection of BTX-A or placebo into the masseter, or not treated (control). An occlusal force analysis system was used to collect several measures: duration of biting and closing, maximum occlusal force, and distribution of occlusal force. Occlusal force in the intercuspid position was reduced in all 3 groups. There was a significant difference between the BTX-A and placebo groups (F(df = 1) = 8.08, P = .01) but not between the control group and the other 2 (F(df = 1) = 4.34, P = .047). The duration of occlusion was significantly increased in the BTX-A group after 3 months of treatment (t = 4.07, P = .003). The asymmetric distribution of occlusal force was reduced in all 3 groups, but not significantly (Levene’s test, F [df = 2] = 0.25, P = .78; analysis of variance, F [df = 2] = 0.50, P = .61). The authors proposed that treatment of TMD with BTX-A is effective at reducing occlusal force, but psychological intervention is an important part of the treatment.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Origin</th>
<th>Insertion</th>
<th>Injection technique</th>
<th>Dose</th>
<th>No. of injections</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medial pterygoid</td>
<td>Deep head; Medial side of pterygoid plate</td>
<td>Medial aspect of angle of mandible</td>
<td>Extraoral: Injection via submandibular route</td>
<td>5-25 U</td>
<td>2-3/side</td>
</tr>
<tr>
<td>Extraoral: Injection via submandibular route</td>
<td>(access to superior aspect of muscle) before injection. Can be the muscle as to stay superficial medial injection could approach infratemporal fossa and its contents.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dose type A</td>
<td>1000-3000 U</td>
<td>1/side</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dose type B</td>
<td>5-25 U</td>
<td>2-3/side</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

EMG, electromyography; TMJ, temporomandibular joint.
BTX is also a good and safe option for treating nocturnal bruxism: 25 to 100 U of BTX type A injected into the masseter muscle provides therapeutic relief in patients with severe bruxism refractory to medical therapy.\(^{27,28}\) One randomized controlled trial (n = 30) showed that Botox was efficacious in reducing myofascial pain symptoms in bruxers compared with control patients who received saline placebo injections.\(^{29}\) Tan et al.\(^{28}\) evaluated the effectiveness of BTX in treating severe disabling bruxism, in which patients manifested diurnal or nocturnal tooth grinding and the majority also had movement disorders. A total of 241 injections of 61.7 ± 11.1 MU of BTX type A per side of masseter muscle were given to 18 patients during 123 treatment visits. The mean maximum and total duration of response were 11.7 ± 4.1 weeks (range 2.5-18 weeks) and 19.1 ± 17 weeks (range 6-78 weeks), respectively. The mean peak effect of BTX was 3.4 ± 0.9, suggesting that there was moderate improvement in severity and function. BTX is a safe and effective treatment for patients with severe bruxism. As it is an expensive treatment modality, it should be considered as a therapeutic option for complicated or disabling bruxism, especially associated with movement disorders and refractory to other conventional therapy. Because TMDs are multifactorial, psychological counseling is also an integral part of treatment, especially in complex forms.

**Migraine.** Migraine is the most common neurologic disorder, consisting of the following clinical criteria: a lifetime history of at least 5 recurrent headaches, untreated or unsuccessfully treated headache with a duration of 4-72 h, and at least 2 of these pain characteristics: unilateral, pulsating, moderate to severe intensity, and aggravated by physical activity. In addition, it can be associated with one of the following: nausea/vomiting, photophobia, or phonophobia.\(^{30}\)

BTX type A was approved by the FDA in 2010 for the treatment of mainly chronic migraine, and it is the only prophylactic therapy specifically for chronic migraine. The International Headache Society defines chronic migraine as headache for more than 3 months in the absence of medication overuse. Respite from migraine provided by BTX is due to muscle relaxation and the resultant decrease of pressure on the trigeminal nerve, inhibition of hyperexcitable motor and sensory neurons, and suppression of central and peripheral sensitization of the trigeminal sensory nerve around muscle trigger points.\(^{31}\) For frontal migraines, injection of 35 to 40 U of BTX in the frontalis, corrugators, and procerus is the most common technique. For temporal migraines, 20-25 U per side on the anterior aspect is given. For occipitalis, 10 to 20 U BTX is given on both sides and just superior to the occipital protuberance. A dose of 20 to 30 U BTX is recommended for splenius capitus and semispinalis muscles, but identification of these muscles is difficult. Injections are repeated every 12 weeks.\(^{32-34}\)

Tension headache is another prevalent headache associated with increased pericranial muscle tone, with an unclear pathogenic mechanism. This headache is often bilateral, pressing, and not aggravated by physical exercise, with episodes ranging from 30 minutes to continuous; often there are no associated symptoms of nausea, photophobia, or phonophobia.\(^{35}\) BTX type A is injected into the temporalis, pericranial, and cervical muscles of the neck for 18 months, and patients usually show an improvement in headache severity and increased headache-free intervals.\(^{36,37}\)

**Trigeminal neuralgia.** Trigeminal neuralgia, also known as tic douloureux, is defined by the International Association for the Study of Pain as “sudden unilateral, severe, brief, stabbing recurrent episodes of pain in distribution of one of more branches of the trigeminal nerve.”\(^{38}\) It is sharp, shooting, electric shock—like, frightful pain lasting for seconds to minutes. Carbamazepine remains the drug of choice, but oxcarbazepine is equally effective and has fewer side effects. These treatment modalities have been found to improve the quality of life of neuralgia patients, but the antiepileptic drugs have central nervous system side effects and many patients become refractory to these drugs over time. Surgical treatment can relieve pain symptoms, but has been associated with sensory side effects.\(^{39,40}\) Recently, BTX type A injections have generated interest as a cure for this debilitating orofacial disease by paralyzing trigger zones.

It is hypothesized that botulinum toxin provides pain relief by inhibiting the release of glutamate, calcitonin gene-related peptide, and substance P, resulting in inhibition of peripheral sensitization of nociceptive fibers and reduced central sensitization.\(^{38}\) Piovesan et al.\(^{31}\) reported success in achieving nearly complete pain relief in 13 patients within 10 days after subdermal injections of BTX-A at a mean dose of 3.22 U/cm² directly into the affected facial region. Allam et al.\(^{42}\) reported the effectiveness of local injections of BTX-A for pain relief and long-term control in a patient with intractable trigeminal neuralgia. Two units of BTX type A were subcutaneously injected into 8 points distributed along the territory of the ophthalmic and maxillary branch. The authors were able to reduce trigeminal neuralgia pain with BTX type A injections in the V1, V2 territory during the period of the study, as well as to withdraw all the medication. There was also concomitant reduction of pain in the mandibular branch, which was not injected.

Ngeow et al.\(^{9}\) reported a case of persistent trigeminal neuralgia in a medically compromised 65-year-old
woman who did not respond to pharmacotherapy. She had undergone several peripheral neurectomies as well as a failed right posterior fossa exploration that resulted in a cerebrospinal fluid leak. A single dose of 100 U of BTX-A diluted in 2.5 mL saline was injected into the external nasal trigger zone (60 U) and the mental nerve region (40 U). She achieved complete pain relief in the external nasal region for 5 months. Pain recurred, and the site was again injected with 100 U of BTX-A. Pain relief at the mental region was partial. This was finally controlled with peripheral neurectomy.

Botulinum toxin has a working life of 3 months when injected. These studies demonstrated its effectiveness for short-term (60-to-90-day) control of trigeminal neuralgia. However, Ngeow et al.9 showed that repeated injections are useful in promoting a continuous pain-free state. Botox was found to be effective in combination with pharmacotherapy before considering more invasive therapies such as surgery or gamma knife radiosurgery. As such, Botox is a particularly valuable treatment for elderly patients and those with adverse anesthetic comorbidities.43

First bite syndrome. First bite syndrome, described by Haubrich in 1986, is the sudden onset of acute and severe pain in the parotid region at the initiation of mastication. It lasts less than a minute and leads to a fear of oral intake. It is typically seen after parotidectomy or deep parotid space surgery.44 It is caused by a loss of sympathetic innervation to the parotid gland with subsequent hypersensitivity of myoepithelial cells to parasympathetic neurotransmitters. This hypersensitivity leads to maximal contraction of myoepithelial cells during the first bite of a meal, and intraparotid injection of 40-60 U of BTX under ultrasound guidance paralyzes these myoepithelial filaments and relieves symptoms.35,46

Salivary gland secretory disorders
Salivary gland secretory disorders occur after major salivary gland surgery, head and neck cancer surgery, and posttraumatic sialoceles of the parotid duct. They pose a major diagnostic challenge for the clinician.

Sialorrhea. Sialorrhea, also known as drooling or ptyalism, is considered abnormal if it persists beyond 4 years of age. Pathologic sialorrhea occurs due to various neurologic disorders such as Parkinson disease, cerebral palsy, and amyotrophic lateral sclerosis or as a side effect of medications. In recent years, botulinum toxin has been found to be effective and safe in the treatment of sialorrhea and may significantly decrease saliva production when injected intraglandularly. Parasympathetic-dependent secretory function is depressed, whereas basal flow rate is maintained by the adrenergic pathway, thus avoiding the risk of xerostomia.47,48 Both 250 U abobotulinum toxin A and 2500 U rimabotulinum toxin B administered by ultrasound-guided intrasalivary gland injection are safe and effective in treating sialorrhea, even after long-term follow-up. BTX blocks the release of acetylcholine from motor and autonomic nerves by cleaving soluble N ethyl-maleimide sensitive factor attachment protein receptors; as a result, parasympathetic-dependent secretory function is depressed, whereas basal flow rate is maintained by the adrenergic pathway, thus avoiding the risk of xerostomia.47,48

For the treatment of sialorrhea, each parotid gland receives an injection of 25-60 U BTX per treatment, fractionated into 4 doses, as the gland is divided into anterior, posterior, superior, and lower compartments, each receiving 15 U. All injections should be intraglandular, under ultrasound guidance. The submandibular gland should receive 40 U BTX fractionated into 2 doses of 20 U each for the anterior and posterior portions.49

Frey syndrome. Frey syndrome, also described as gustatory sweating or auriculotemporal syndrome, presents clinically as unilateral facial sweating and flushing on salivary stimulation and mastication. It occurs most commonly after parotid surgery. Frey syndrome is caused by aberrant regeneration of postganglionic parasympathetic fibers feeding the parotid gland, which are severed during parotid surgery.50,51 The first symptomatic treatment of Frey’s syndrome currently available is BTX type A injected intracutaneously. Local injection of BTX in the area of gustatory sweating is an effective means of reducing the activity of sweat glands in the preauricular area. Most patients are symptom-free for 6 months, with effects lasting up to 15 months.52

For management of Frey syndrome, Capaccio et al.53 developed a protocol whereby each parotid gland receives 25-60 U of intraglandular BTX injections per treatment divided into 4 doses, as the parotid gland contains anterior, posterior, upper, and lower compartments and each should receive 15 U BTX injection.

Head and neck movement disorders
Head and neck movement disorders remain a diagnostic challenge for clinicians and encompass a wide variety of disorders with various clinical manifestations, such as dyskinesia, dystonia, and oromandibular dystonia, a focal dystonia that involves the mouth, jaw, and/or tongue. It can be classified as idiopathic or tardive dystonia or secondary to other neurologic disorders and subdivided into jaw opening, jaw closing, jaw deviation, and lip pursing. The muscles involved in jaw opening dystonia are usually the digastrics and lateral
Botulinum toxin injections in lower facial contouring

Masseter muscle hypertrophy. Masseter muscle hypertrophy (MMH) is a benign increase in the size of the masseter muscle that can affect one or both sides of the face. Pain can be a symptom, but most frequently the clinician is consulted for cosmetic reasons. In some cases, prominent exostoses at the angle of the mandible is noted. Treatment of MMH ranges from conservative to invasive therapies. Milder cases do not require therapy; however, in severe cases or for cosmetic reasons, BTX type A injections or surgery can be considered.

BTX therapy usually reduces muscle size and muscle hyperactivity in these patients. It causes disuse atrophy in one of the balanced muscles (masseter), but as the effect of the toxin wears off, there is balanced recovery leading to symmetric appearance of the face, and the effect lasts even until 24-month follow-up. Dosage and duration of effect of BTX type A injections in patients has not yet been specified. Kim et al. recommended 100 to 140 U of BTX A for 10 to 16 mm muscle thickness and Choe et al. recommended ≥20 U for any thickness >10 mm. Recently, Xie et al. tailored the dose of BTX A to define the number, dosage, effect, and complications of the drug. Overall, dosage ranges from 20 to 50 U/muscle of BTX type A to treat MMH, but the duration of effect varies individually.

Contraindications to use of botulinum toxin. Although botulinum toxin is considered a safe, noninvasive, nonsurgical treatment modality, it has many contraindications that can limit its usage. The following patients should avoid BTX injection: patients who are psychologically unstable; are dependent on intact facial expressions for their livelihood, such as singers, musicians, and actors; have neuromuscular disorders such as myasthenia gravis; are allergic to any component of BTX A or BTX B; have any medications that interfere with neuromuscular impulse transmission and can potentiate the effects of BTX, such as amino-glycosides, calcium channel blockers, and penicillamine; are pregnant or lactating (BTX is a category C drug); or have an infection at the injection site.

Side effects with botulinum therapy. With botulinum therapy, the injected muscles could be sore for few days. BTX can cause temporary partial weakening of the injected muscles. Long-term use could cause atrophy of injected muscle, which is reversible on discontinuation of therapy. There are also side effects such as flu-like symptoms, palpitations, tingling sensations, and nausea. These side effects remain for only 1 to 2 days.

CONCLUSION

Various conservative therapies, medicines, and minor and major surgical procedures have been used in the past to treat facial pain, secretory disorders, and head and neck movement disorders. Few patients failed to respond to these treatment modalities, with variable responses. BTX has progressed from being used in cosmetic procedures only to a spectrum of clinical applications, as discussed above. It is a superior treatment option over pharmacotherapy or surgery for head and neck disorders, with low mortality and morbidity. By nature it is a poison, but it has proven to be a successful curative agent. In the future, more clinical trials will incorporate this agent toward widespread usage.

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