Hemifacial Spasm: Evaluation and Management, with Emphasis on Botulinum Toxin Therapy

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INTRODUCTION

Hemifacial spasm is a disorder of the facial nerve characterized by involuntary synkinetic movement of facial muscles on the afflicted side associated with minimal facial weakness. Management of this disorder has transcended several medical and surgical specialties. The condition has implications for ophthalmological practice because of visual obstruction produced by involuntary eyelid closure, which is most troublesome to patients. Neuro-ophthalmic or neurological evaluation is necessary for accurate diagnosis and exclusion of vascular mass lesions in the posterior fossa, which are very uncommon. Neurosurgical experience has been useful in elucidating the pathogenesis and offering an alternative therapeutic modality. Additionally, since the condition adversely affects a patient's ability to communicate via facial expression and produces substantial disfigurement, a plastic surgeon may occasionally be consulted. This chapter surveys the clinical characteristics of hemifacial spasm and reviews the application of therapy with botulinum toxin type A therapy for this disorder.

CLINICAL CHARACTERISTICS

Hemifacial spasm usually appears between the fourth and fifth decades of life, most often as an involuntary twitch in the orbicularis oculi muscle. The movement begins in the lower eyelid and extends to the upper lid. The involuntary movement progresses to gain sufficient force to cause increased blink rates and forced closure of the eyelids on the involved side. Shortly thereafter, the involuntary muscle twitching becomes more generalized to involve not only the lid protractors but muscles over the upper lip on the affected side. As the condition progresses, the twitching becomes an obvious involuntary movement over all facial muscles on one side as well as the thin superficial muscle of the neck,
the platysma (Fig. 1). Involvement of the platysma causes asymmetric bands and twitching of the neck muscles in addition to muscles of facial expression. Occasionally, with involvement of the stapedius muscle of the middle ear, the patients may hear an auditory hallucination characterized as a “thumping” associated with the spasm. This auditory hallucination occurs only in a small minority of patients. Occasionally, the patient may complain of hearing loss.

In the most severe forms of involuntary movement, there is a more sustained distortion on muscles on one side of the face (Fig. 1). In more advanced cases, the distortion caused by a mass contraction of all muscles supplied by one facial nerve causes a forced closure of the eyelid, eyebrow elevation, elevation of the nasolabial fold, ectropion of the lower lip with chin deformity, deviation of the tip of the nose toward the left side, and tight band formation within superficial muscles of the neck. The spasms most notably involve the orbicularis oculi, frontalis, zygomaticus major and minor, orbicularis oris, and mentalis muscles on one side of the face. With mass contraction of these muscles, the eyelid is forced closed, the nasolabial fold is prominently pulled up, the vermilion border is displaced toward the involved side, and the lower lip turns outward. Occasionally the eyebrow become elevated during periods of mass contraction.

If the condition exists over a period of many years, the weakness and involuntary movement can cause fixed contractures in which the disfigurement remains constant rather than intermittent (Fig. 2).

Unlike some other movement disorders, hemifacial spasm is clearly an intermittent disease in most situations. This “on-off” characteristic can occasionally delay accurate

**Figure 1** Typical appearance of a patient with advanced hemifacial spasm. Note forced involuntary eyelid closure, elevation of brow, distortion of the lateral angle of the mouth, and ectropion of the lip on the involved side.
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Figure 2  After 35 years of involuntary facial spastic movements associated with facial weakness, this patient with hemifacial spasm developed fixed facial contractures.

diagnosis. When the patient presents to the clinician, often there is no involuntary movement, and the examining physician may be unable to make a clear diagnosis. As the disease progresses, the incidence and duration of the spasms tend to increase. In a 10-hour videotaping review of five patients, the involuntary spasms were present for 5–80% of the observation period.

The involuntary movements may be classified into several types. Increased blinking associated with synchronous lower face twitching is most characteristic. The average blink rate of a normal eyelid is 15–25/min with a normal distribution (Fig. 3). When blink rate is quantified over a substantial period for the lid involved with hemifacial spasm, the blink rate can be seen to be distributed over two superimposed normal statistical curves (Fig. 3). One curve represents the blink rate when the spasms are "off," and the other curve represents the blink rate when the spasms are "on." When pathological blinking is seen, there is almost always synchronous twitch of varying degree in muscles in the lower face, and this provides strong evidence of the diagnosis. In certain situations, another characteristic of the movement disorder is continuous stimulation and tone increase of all the facial muscles that is not sufficient to cause the upper eyelid to close. In this situation, the disfigurement is one of asymmetry of the palpebral fissures and alteration of the skin creases on one side of the face. In this case, the increased blink rate may not be the obvious component of the syndrome. When sudden high-intensity bursts of ectopic impulses stimulate the muscles on one side of the face, the eyelid is forced to close, the brow becomes elevated, and there is distortion of the lower face characterized by an updrawn mouth and turned-out lower lip (Fig. 4). This phenomenon is termed a paroxysm and is always associated with forced lid closure.
Figure 3  The blink rate is normally distributed, with an average rate of 15–25/min, in patients without facial dyskinesia. Because of the "on-off" phenomenon observed with hemifacial spasm, the blink rate is distributed over two superimposed normal curves, one curve representing the "on" periods and the other curve the "off" periods.

Figure 4  Sudden bursts of ectopic impulses force the lid closed, with maximal facial contraction for periods of 5–60 sec.
Morbidity caused by hemifacial spasm results from visual loss on the affected side due to increased blink rate and involuntary eyelid closure. Impaired facial communication and psychological alterations resulting from the disfigurement inherent in the disease are additional problems for patients. When hemifacial spasm occurs on the side of a patient’s only seeing eye, it can have serious visual consequences. More commonly, patients will relate difficulty with interpersonal expression. When casually conversing with such a patient, the average person will not understand the reason for facial movements and will incorrectly interpret the condition as excessive winking due to nervousness or psychological impairment. This disease of facial expression is found most bothersome by patients with occupations requiring frequent interpersonal communication or public speaking.

**PATHOPHYSIOLOGY**

The exact cause of hemifacial spasm remains uncertain. The best understanding of the disease comes from neurosurgical observation and experience (1) and electromyographic data (2). The working theory is that an aberrantly tortuous blood vessel at the base of the brain results in a displacement of the intracranial portion of the seventh cranial nerve and a pressure phenomenon, with deterioration of the myelin sheath. A focal area of demyelination results in ectopic impulse generation in the facial nerve, with propagation of the impulse through ephaptic transmission, causing the involuntary synchronous motion of half the face. A small degree of facial muscle weakness often accompanies the involuntary movements. Nelson, using electromyographic methods, has described a phenomenon of ectopic impulse generation as well as ephaptic transmission of the impulse down the nerve (2). Ephaptic generation is defined as side-by-side axonal impulse propagation that occurs as a result of damage to the facial nerve. Ephaptic transmission explains the synchronous movements of the face observed in physical examination. Although this explanation may account for a number of cases, there are patients with hemifacial spasm who present in their early 20s with no apparent neuroradiological evidence of tortuous blood vessels. Alternative explanations may involve aberrant stimulation of the facial nerve nucleus or fasciculus. In a small percentage of patients (< 1%) (3–5), there may be mass lesions in the posterior fossa. In the Boston experience, the lesions most commonly found were arteriovenous malformations in the region of the cerebral pontine angle (Fig. 5).

It is clear that a pathophysiological explanation of the phenomena of hemifacial spasm must account for a high incidence of negative neuroradiological findings, a small degree of facial weakness consistently seen in patients, and synchronous contractions on the afflicted side.

**DIFFERENTIAL DIAGNOSIS**

Hemifacial spasm must be differentiated from many other conditions involving involuntary movement of one side of the face. The most common involuntary movement seen in ophthalmological practice is myokymia of the orbicularis oculi muscle. This movement is characterized by a small twitch in the lower lid interpreted as a nuisance by the person affected. Myokymia, however, never causes contraction within the orbicularis muscle forceful enough to cause complete eyelid closure. Hence, this condition does not really cause involuntary blinking. Orbicularis oculi myokymia has no pathological significance and usually resolves spontaneously. Rarely, localized myokymia may be the first symptom of hemifacial spasm.
Hemifacial spasm must be differentiated from aberrant regeneration of the facial nerve, as both conditions can produce synchronous contractions on one side of the face. Aberrant regeneration of the facial nerve is distinguished from hemifacial spasm by a preceding bout of facial paralysis, usually from Bell's palsy but occasionally as result of herpes zoster oticus or skull base fracture or following acoustic neuroma resection. Aberrant regeneration of the facial nerve is associated with facial weakness, some degree of hemifacial contracture with constant distortion of facial landmarks, and synkinetic lid closure on the affected side. Synkinetic lid closure is defined as involuntary eyelid closure associated with lower face movements during speech, chewing, or eating (Fig. 6). The
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Figure 6  Synkinetic eyelid closure connected with lower facial movement is diagnostic of aberrant regeneration of the facial nerve.

syndrome of aberrant regeneration of the facial nerve also occasionally includes excessive tearing (epiphora) linked with gustatory sensation (crocodile tears). The involuntary movement associated with aberrant regeneration of the facial nerve is distinctly different from hemifacial spasm in that (1) the synkinetic lid closure is constantly present, (2) residual facial weakness usually is more apparent than the mild degree of weakness seen in hemifacial spasm, and (3) epiphora is not seen with hemifacial spasm.

Hemifacial spasm needs to be distinguished from hemifacial myokymia, a distinctly rare condition in which low-amplitude twitches and fibrillations occur in all muscles on one side of the face. Usually, in this condition, not enough force is generated to cause the eyelid to close completely. Hemifacial myokymia is often associated with demonstrable pathology in the posterior fossa (6).

Hemifacial spasm must be distinguished from benign essential blepharospasm and blepharospasm associated with Meige’s disease and generalized movement disorders (7–10). The distinction can easily be made, since hemifacial spasm affects muscles only on one side of the face. Essential blepharospasm and blepharospasm associated with Meige’s disease and other generalized dystonias are most commonly bilateral, although the presentation rarely may be more unilateral. In patients with unilateral Meige disease, there are asynchronous movements on the involved side.

**THERAPY**

There are two forms of therapy effective for the treatment of hemifacial spasm, (1) microvascular decompression of the intracranial portion of the seventh cranial nerve via posterior craniotomy and (2) intramuscular injection of botulinum toxin (BTX).
Neurosurgical Decompression of the Intracranial Facial Nerve

Microvascular decompression of the facial nerve was first described by Jannetta et al. (1). In this procedure, a posterior craniotomy is needed to expose the facial nerve in the vicinity of arteries of the ventral brain stem, with use of a neurosurgical operating microscope. Insulating material is placed between the facial nerve and blood vessels of the region. The insulating effect of gelfoam or other neurosurgical material essentially relieves the pressure of the tortuous vessel on the seventh cranial nerve, which is thought to provoke the movement disorder.

There are obvious disadvantages to this approach. The first and most obvious is that patient acceptance of neurosurgical procedures for non-life-threatening conditions is generally low. The efficacy reported for this procedure is approximately 70–89% (1) in the long term. In short-term follow-up, the success rate has been described as approximately 60%. It is clear that the neurosurgeon performing the procedure should be skilled in the technique for the best possible results. Potential complications of this procedure are the second important drawback and have been the major reason for reluctance within the medical community. Complications, although rare, can include quadriplegia from hemorrhage or vasospasm during surgery or in the postoperative period. Hearing loss on the operative side has also been reported. Although complications clearly occur in a very small fraction of cases (fewer than 1%), patients often reject the procedure when advised of the potential risks. Long-term evaluations of this procedure have not been published. It is the opinion of this author that extended videophotographic studies with well-defined efficacy criteria need to be reported for this surgical procedure to fully assess the rates of successful outcomes. Nevertheless, the concept of decompression and its demonstrated efficacy have made a substantial contribution to medical understanding of the cause of hemifacial spasm.

Botulinum Toxin Type A

Botulinum toxin type A (BTX-A) is clearly the first choice for treatment of hemifacial spasm. It is relatively easy to administer and provides relief in the vast majority of patients (over 95%) (11–17). The major disadvantages of the application include (1) the need for repetitive injections indefinitely, (2) potential for lagophthalmos and ophthalmic complications, and (3) facial asymmetry.

Botulinum toxin is quantified with a biological assay assessing the lethality of an injection of a given quantity into a standard white mouse. The international unit is the lethal dose of 50% of a cohort of white mice injected. The total dose used in the treatment of hemifacial spasm should start between 10 and 15 IU. This dose is substantially below the quantity needed to treat other forms of blepharospasm. For instance, Meige’s disease and essential blepharospasm should generally be treated with higher doses, 30–40 IU. Table 1 compares dose-response relations for Meige’s disease, essential blepharospasm, and hemifacial spasm. The reasons for the differences involve the pathophysiology of hemifacial spasm as compared with Meige’s disease and essential blepharospasm. Meige’s disease and essential blepharospasm are basically dystonias, that is, movement disorders arising from derangement in information processing within the central nervous system. Central nervous system regulation of facial movement and blink is altered in such a fashion that involuntary impulses arise, with the impulses propagated over normal peripheral nervous structures. Hemifacial spasm is distinctly different as it is associated with facial neuropathy and facial muscle weakness. Partially denervated muscle, such as
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Table I Dose Response Relations, Comparing the Various Forms of Blepharospasm

<table>
<thead>
<tr>
<th></th>
<th>Blepharospasm</th>
<th>Hemifacial spasm</th>
<th>Aberrant regeneration</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients/no. of injections</td>
<td>112/697</td>
<td>71/269</td>
<td>12/40</td>
</tr>
<tr>
<td>Mean dose (IU)</td>
<td>35.4790a</td>
<td>20.1877b</td>
<td>22.4426b</td>
</tr>
<tr>
<td>Dose: standard deviation</td>
<td>8.4529</td>
<td>1.3919</td>
<td>1.8705</td>
</tr>
<tr>
<td>Average no. of injections</td>
<td>6.22</td>
<td>3.79</td>
<td>3.33</td>
</tr>
<tr>
<td>Average duration (months)</td>
<td>3.1</td>
<td>5.6</td>
<td>6.28</td>
</tr>
</tbody>
</table>

aOne-half of the total dose given for blepharospasm is shown, because this condition is bilateral whereas aberrant regeneration is unilateral.
bDose requirements for aberrant regeneration of the facial nerve and hemifacial spasm are significantly lower than requirement for Meige syndrome (blepharospasm), p < .001.

occurs with hemifacial spasm, appears to be more sensitive to the weakening effect of BTX (Table 1).

With repeated injections of the toxin, there is some degree of resistance. As the number of BTX treatment sessions received by a patient increases, the dosage needed in subsequent injections generally becomes higher. This is particularly true for Meige’s disease and essential blepharospasm, and less so for hemifacial spasm. Comparative dose requirements after multiple injections for various syndromes causing blepharospasm are outlined in Fig. 7.

Figure 7 Dose requirement after multiple injection periods over an average of 3–4 years for various syndromes causing blepharospasm (Meige syndrome, aberrant regeneration of the facial nerve, and hemifacial spasm).
Generally, the dose requirements for hemifacial spasm level off between 20 and 25 IU. Injection of higher doses of BTX can result in excessive weakness of the orbicularis oculi muscle, paralytic lagophthalmos, and exposure keratopathy, resulting in pain and blurred vision. Patients with hemifacial spasm are more susceptible to this complication than patients with essential blepharospasm, because of the preexisting weakness associated with the former disease. Occasionally, dose levels of 20–25 IU may be excessive and can cause symptomatic exposure keratitis.

Patients with known dry-eye syndromes should receive especially low starting doses to avoid postinjection keratitis.

As the doses needed to treat facial dyskinesias are very low, there has never been a case report of BTX intoxication in the application of this treatment modality. In contrast, in the treatment of spasmodic torticollis, a form of dystonia, the dose requirements range between 200 and 300 IU. Since the therapeutic index (therapeutic dose/LD₅₀) of BTX-A is low, systemic complications from disseminated weakness are expected to be very rare. The minimal estimated lethal dose of BTX for a human is approximately 2500 IU (18).

The injection sites are important in the treatment of hemifacial spasm as well as other forms of blepharospasm. When BTX is injected for the treatment of hemifacial spasm with involuntary eyelid closure, it is important to target the orbicularis oculi muscle for weakening, as this muscle promotes involuntary eyelid closure and increased blinking. The injection sites for the treatment of hemifacial spasm are shown in Fig. 8A. Although these injection points may be appropriate for most patients, modification of the injection points occasionally is necessary if most of the involuntary movement is in the lower face.

The efficacy of treatment can be measured by assessing blink rate (Fig. 8B) and quantitating palpebral fissure asymmetry (Fig. 8C) during an extended period using video recordings.

**Figure 8**  (A) Injection points used to treat blepharospasm associated with hemifacial spasm. Note that the lateral injections affect the ipsilateral zygomaticus major and minor muscles, which are retractors to the nasolabial fold and the lateral angle of the mouth. Injections points occasionally need to be customized to individual patients, such as those who demonstrate the involuntary movement primarily in the lower face. (B) Efficacy of botulinum toxin injections using blink rate as criterion, before and 2 weeks after injection. Note that there is substantial reduction in the blink rate after treatment. (C) Efficacy of botulinum toxin injections using periods of palpebral fissure asymmetry as criterion, measured over 30–45 min before and after injection. Palpebral fissure closure from orbicularis muscle spasm causes both loss of visual field and disfigurement from hemifacial spasm.
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Average Blinks per minute

Before Injection 2 wks After Inject

Pt 1 Pt 2 Pt 3 Pt 4

Asymmetry over 5 min in sec

Before injection 2 wks After Inject

Pt 1 Pt 2 Pt 3 Pt 4

Note: The graphs show the changes in blink rate and asymmetry over 5 minutes before and after botulinum toxin injection for four patients.
Over the past 8 years, it has been recognized that the effectiveness and rate of complications of BTX administration can be technique-dependent (19,20). The site of toxin injection is anatomically important and consequential to the effectiveness of the therapy. Botulinum toxin is injected at four points into the orbicularis oculi (Fig. 8A). Two injection points are used in the upper lid, one to the medial extreme of the upper lid and the other on the lateral extreme of the upper lid. A lateral injection point and a lateral inferior lid injection point complete the multiple-point injection strategy. The location of injections over multiple points is important in achieving a diffuse biological effect of the toxin throughout the orbicularis muscle. The lateral orbicularis injections also cover the upper portions of the zygomaticus major and minor muscle, which are responsible for the position of the nasolabial fold and the lip. Another implication of the injection points concerns toxin diffusion to contiguous muscles, which causes complications. For instance, injection in the superior portion of the upper lid into the anatomical lid fold can have deleterious effects caused by diffusion into the levator palpebrae superioris muscle, which keeps the upper lid in the appropriate position (Fig. 9A,B). Toxin diffusion into the levator palpebrae superioris muscle will result in decreased muscle tone, which results in ptosis (drooping of the upper eyelid) (Fig. 10). Ptosis can further obstruct a patient’s vision and create further disfigurement in patients with hemifacial spasm. This complication is usually transient, lasting 2–3 weeks. Placing the upper-lid injections close to the lash line and away from the midline of the lid maximizes the distance between the upper-lid points of injection into the orbicularis muscle and the orbicularis muscle’s antagonist, the levator palpebrae superioris muscle. In fact, the anatomical remoteness of the pretarsal orbicularis muscle from its antagonist (the levator) is an important reason for the success achieved by regional BTX injections for blepharospasm. Because of this distance, containment of the denervative effect to the orbicularis oculi without weakening the eyelid retractors is possible.

In the lower lid, the injection points should be lateral and, particularly, away from the inner aspect of the lid. Nelson and co-workers (19) have identified another complication, diplopia (double vision), occurring as a result of diffusion of BTX into the inferior oblique muscle from medial lower lid injections. As the inferior oblique muscle is the most anteriorly located extraocular muscle in the orbit, arising posterior to the posterior lacrimal crest along the medial orbit, it is easy to understand that medial lower lid injections can diffuse into this region, causing another complication of therapy. The inferior oblique muscle actually lies very close to medial lower lid skin as it passes through the capsulopalpebral fascia.

Multiple injection points around the orbicularis and other targeted muscles are important for a directed homogeneous effect. In another chapter, it was demonstrated that the diffusion potential of point injection of BTX is clearly dose-dependent. At higher doses, the toxin not only produces a greater degree of weakness of the injected muscle but has the potential to diffuse away from the point of injection and cause complications by affecting nontargeted muscles. Additionally, the multiple-point injection not only allows lower doses in given areas within the muscle, but also, if the muscle’s innervation zone is diffuse, it probably saturates the innervation zone more completely. For instance, the innervation zone of the orbicularis oculi muscle is probably very diffuse (21). The facial nerve projects to this muscle from a number of different ramifications from the temporal, zygomatic, and buccal branches. This complex motor nerve network to the orbicularis oculi from multiple branches of the facial nerve affects this muscle’s responses to resections and reconstructive surgery of the eyelids. The orbicularis muscle is not easily
Figure 9 Injection of botulinum toxin into the lid fold may cause ptosis from diffusion of the biological activity of the toxin into the levator muscle of the eyelid. (A) The thin arrows demonstrate the position of the lid fold. Lid fold injections bring the injection point into close proximity to the muscular portion of the levator palpebrae superioris muscle. Weakening of the levator muscle results in ptosis (see Fig. 10). (B) Part of the surgical field of a ptosis operation (levator advancement), demonstrating the proximity of the muscular portion of the levator to the lid fold.
muscle are probably more effective in producing the most beneficial results. A similar phenomenon may explain why BTX is more effective when used in multiple-point injections in the treatment of adult-onset spasmodic torticollis (23).

Facial dynamic asymmetry is a problem in using BTX for hemifacial spasm. Botulinum toxin weakens injected muscles. Alterations in the injected muscle's resting tone can cause a small degree of resting asymmetry. However, because of depressed contractility of injected facial muscles, there can occasionally be more substantial facial asymmetry during periods of active facial expression, when facial muscle contractility is greatest as compared with periods of neutral facial expression (Fig. 11). It is rewarding to be able to eliminate the amplitude of spasmodic contractions by using BTX injection. However, an unfortunate complication of the injection is that the amplitude of volitional contractions on the side of the face injected is also occasionally noticeably impaired. It is possible to titrate the dose in such a fashion that volitional contractions are much less impaired than the high-velocity contractions associated with the spasm.

The term “facial dynamic asymmetry” is used to advance the concept that facial symmetry needs to be evaluated during periods of active expression, such as smiling and laughing, as well as with facial muscles at rest. Although BTX therapy can rarely cause asymmetry with facial muscles at rest (static asymmetry), the much more common asymmetry occurs during periods of active expression. The static asymmetry is noted by a droop on the lateral angle of the mouth and depression of the nasolabial fold (Fig. 12A,B). This complication would indicate that an excessive dose of BTX was administered. The dynamic asymmetry accentuated during active facial expressions is, again, more common and is demonstrated by symmetry in upper lip excursions and dentition show during smiling, lack of crease accentuation and elevation along the nasolabial fold and other dynamic creases of the lower face, and drooping of the side of the lower lip (Fig. 13A). The major reason for the asymmetry appears to be excessive effect on the zygomaticus major and minor muscles resulting from excessive biological effect diffusing from the more inferolateral injections to the orbicularis oculi muscle. Occasionally, this asymmetry results from injection of excessive amounts of toxin directly into the zygomatic muscles. As BTX tends to reach its peak effect of muscle weakening within 14 to 17 days after the
Figure 11  Example of facial dynamic asymmetry. Note asymmetry of upper lip and nasolabial fold excursion and asymmetry of exposure of the teeth.

Injection, patients will generally call within 2 weeks with this complaint. The clinician who receives such a complaint has several options. The first is to reassure the patient that the complaint will be temporary and that as the effect of the BTX recedes, the degree of asymmetry will clearly diminish. The patient should be reassured that this complication is always reversible. Another more immediate approach would be to use an injection of BTX contralateral to the side affected (24) (Fig. 13B). This contralateral injection should be placed over the intersection between the zygomaticus major and minor muscles lying over the zygomatic bone. The action of these muscles is primarily to raise the lips, the lateral angle of the mouth, and the nasolabial fold, and injection of BTX to the contralateral side diminishes the contraction amplitude of these facial muscles and yields a more symmetrical dynamic facial appearance with more even dentition show during smiling (Fig. 14). The author has found this approach to be effective in a number of patients (24,25). A dose of 5–15 IU given at three points just over the zygomatic arch on the contralateral side usually suffices to produce this effect (Fig. 13B).

This approach in creating facial symmetry derives from the observation that it is difficult to diagnose patients with simultaneous bilateral facial nerve disease. Because there is symmetry in the paralytic facial movements, the clinician has difficulty perceiving facial weakness. If a patient expresses displeasure with asymmetry of dynamic expression, the asymmetry is reduced by altering contractility of the normal side of her face. A similar approach has been suggested in managing forehead symmetry after frontalis branch damage of the face in patients undergoing cosmetic face-lifts (26). The major side effect encountered with contralateral zygomaticus injections is impaired excursions of the upper lip, which can be noticeable to the patient during speech. Smile characteristics will also change.
Another problem frequently encountered in patients with hemifacial spasm is a concomitant dry eye syndrome. Dry eye is generally assessed using Schirmer paper wetting over a 5-minute period after topical anesthetic is placed in the conjunctival fornix. Patients with dry eye syndrome may experience more severe exposure faster application of BTX. Decreased blink response seen after administration of the toxin results in excessive evaporation away from the precorneal tear film. Nevertheless, patients with hemifacial spasm and dry eye syndrome can be effectively treated with BTX. The author suggests the use of silicone punctal plugs to halt the egress of tears from the conjunctival fornix into the nasolacrimal sac (Fig. 15). The increased wetting produced by nasolacrimal obstruction allows BTX to be used more effectively in patients with dry eyes. Patients with dry eye syndrome should also be given lower doses (5–10 IU). If this dose is not enough to
In summary, the complications of therapy for hemifacial spasm include:

1. Ptosis from toxin diffusion into the muscular portion of the levator palpebrae superioris muscle
2. Double vision, from diffusion of the toxin into the orbit and involvement of the extraocular muscles, particularly the inferior oblique
3. Epiphora from weakening of Horner’s muscle, which functions as the lacrimal pump muscle, taking tears from the conjunctiva and delivering into the nose

mitigate symptoms, it can be increased in 2 weeks. It is wise to titrate the dose up in these patients, as has been previously mentioned.

Figure 13  Diagram of facial asymmetry with dynamic facial movement. Contralateral injections (asterisks) of smaller doses of botulinum toxin (5–10 IU) help maintain symmetry of important facial landmarks during dynamic expression.
Figure 14  Before and after contralateral injection of botulinum toxin to increase facial symmetry during active expressions. (A) Asymmetric exposure of teeth and depressed excursions of the nasolabial fold and lateral angle of the mouth during smiling. (B) This asymmetry is corrected by contralateral injections.
Figure 15  Punctal plugs can be helpful in the treatment of dry eye syndrome, which can be aggravated by the use of botulinum toxin injections to the eyelids. Note the silicone plug placed in the inferior punctum. This plug can be left in place indefinitely.

4. Facial asymmetry from excessive weakening of facial muscles contiguous to the orbicularis oculi, that is, the zygomaticus major and minor muscles
5. Exposure keratopathy from excessive weakening of the orbicularis oculi muscle, with lagophthalmos and corneal drying

To date, no long-term changes in muscle fiber size or permanent muscle fiber atrophy has been demonstrated in biopsied human muscle after multiple toxin injections over several years (22).

**Duration of Action of Repetitive Injections**

The duration of action of BTX for the treatment of hemifacial spasm is distinctly longer than in therapy for other forms of blepharospasm (Fig. 16). The duration of action is very similar to that seen in patients with aberrant regeneration of the facial nerve who are treated with BTX (25). The average duration of effect is 5.5 months (Table I). The duration of effect can vary among individuals, and patients are often asked to come back when the symptoms are returning. It is often helpful to let the patients make their own judgments in this respect.

Sensitization, with decreasing effectiveness, after repeated injections is theoretically possible for patients with hemifacial spasm. Antibody formation has been demonstrated with large-dose applications, as in therapy for torticollis, and with intermediate doses such as those used to treat Meige syndrome. Because the dose requirement is lower for the treatment of hemifacial spasm, the risk of sensitization is probably considerably less. The incidence of sensitization and the long-term impact of this phenomenon still are subjects of active investigation. Sensitization has occurred in one patient treated for blepharospasm.

**CONCLUSION**

In summary, hemifacial spasm is a disorder of the facial nerve characterized by intermittent synchronous involuntary contractions of facial muscles. The condition not only impairs vision but is also cosmetically disfiguring and impairs interpersonal communica-
tion. Although microvascular decompression of the facial nerve can be effective, most patients will prefer therapy with BTX injections. The injections need to be given periodically and indefinitely, usually at 4- to 6-month intervals. There have been no long-term adverse effects of repeated BTX injections for this condition over 7 to 8 years of clinical studies.

REFERENCES

Hemifacial Spasm and Botulinum Toxin Therapy