

Hemifacial spasm (HFS) is a progressive disorder of intermittent, irregular contractions of the muscles of one side of the face. It usually begins with unilateral fasciculations of the periocular orbicularis oculi and surrounding muscles and gradually spreads to involve the muscles of facial expression and the platysma [1].

HFS is typically unilateral, involves the whole side of the face, and may continue during sleep. Mild facial weakness is usually present. HFS is usually caused by irritation of the facial nerve near its origin in the brainstem—usually a kink in the anterior inferior or posterior inferior cerebellar artery, although other vessels may be involved. Magnetic resonance imaging (MRI) is usually recommended in the work-up of these patients to rule out posterior fossa lesions that should be treated surgically.

HFS must be differentiated from myokymia and facial tics. Myokymia is a continuous unilateral localized fasciculation within the orbicularis oculi of the eyelid that occurs in normal individuals under conditions of stress and fatigue. It is usually transient and nonprogressive. Facial tics are habitual and usually begin in childhood and can be suppressed voluntarily.

In cases in which vascular compression of the facial nerve is confirmed by MRI and magnetic resonance angiography (MRA), microvascular decompression of the facial nerve (Jannetta procedure) can be used with a success rate of greater than 80%. However, serious complications such as hearing loss, otitis media, permanent facial palsy, epilepsy, and even death may occur.

Botulinum toxin type-A has become the first line of treatment for HFS. It blocks the release of acetylcholine at the nerve terminal in the neuromuscular junction. Resprouting of new terminal axons to the motor endplate occurs in about

3 months. It is simple and effective and has a success rate of more than 90%. Currently, the product most commonly used to treat HFS is Botox® (Allergan, Inc., Irvine, California, USA). The toxin is supplied as a freeze-dried residue in 50-unit, 100-unit, and 200-unit vials. This is diluted with 1 cc to 4 cc of non-preserved saline to yield a concentration of 10.0 U/0.1 mL, 5.0 U/0.1 mL, or 2.5 U/0.1 mL, as needed. If Botox becomes ineffective, Dysport® (Medicis Pharmaceutical Corp, Scottsdale, Arizona, USA) may be helpful. The initial dosing is variable, but a Botox-to-Dysport ratio of 1 U:2.5–3 U is generally accepted. It is possible that differences in purification result in a greater spread effect with Dysport [2].

Injection Technique

Step 1

The patient is carefully examined, and the sites of spasms are carefully recorded.

Step 2

A 30-gauge needle is used to inject 2.5–5.0 U of Botox in the preselected sites. Injections are given first in the orbicularis oculi muscle, the procerus and corrugator muscles, and the frontalis muscle (Fig. 32.1). The injections within the orbicularis oculi muscle are quite superficial and can extend much farther laterally than previously thought. Injections into the corrugator superciliaris and procerus are deeper. Injections are avoided in the medial aspect of the lower lid to avoid tearing from lacrimal pump dysfunction. Similarly, postinjection ptosis can be reduced by avoiding injections in the central upper eyelid. Also, because the lateral orbicularis extends 2.5 cm outside the orbital rim, injections farther lateral may achieve greater paralysis while decreasing chances of ptosis and diplopia (Fig. 32.2). See Chap. 33 for anatomic discussion.

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Fig. 32.1 A 30-gauge needle is used to inject 2.5–5.0 U of Botox in the preselected sites. (a) Injections are given first in the orbicularis oculi muscle, the procerus and corrugator muscles, and the frontalis muscle. (b) The injections within the orbicularis oculi muscle can extend much farther laterally than previously thought. Printed with permission, Cleveland Clinic Center for Medical Art & Photography © 2017. All Rights Reserved

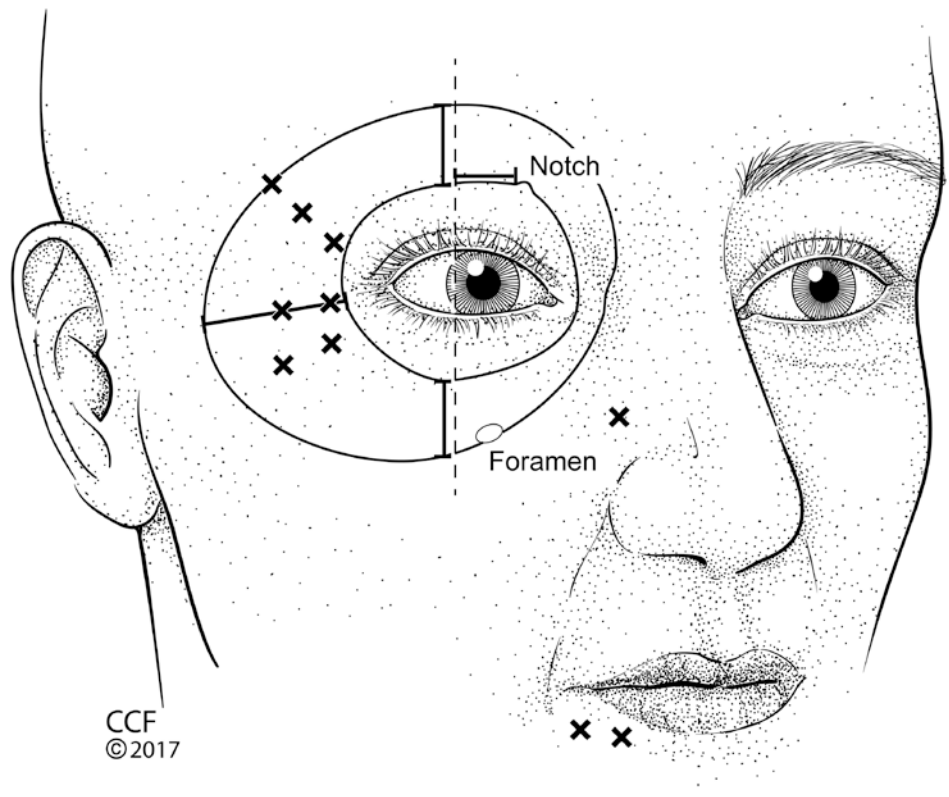
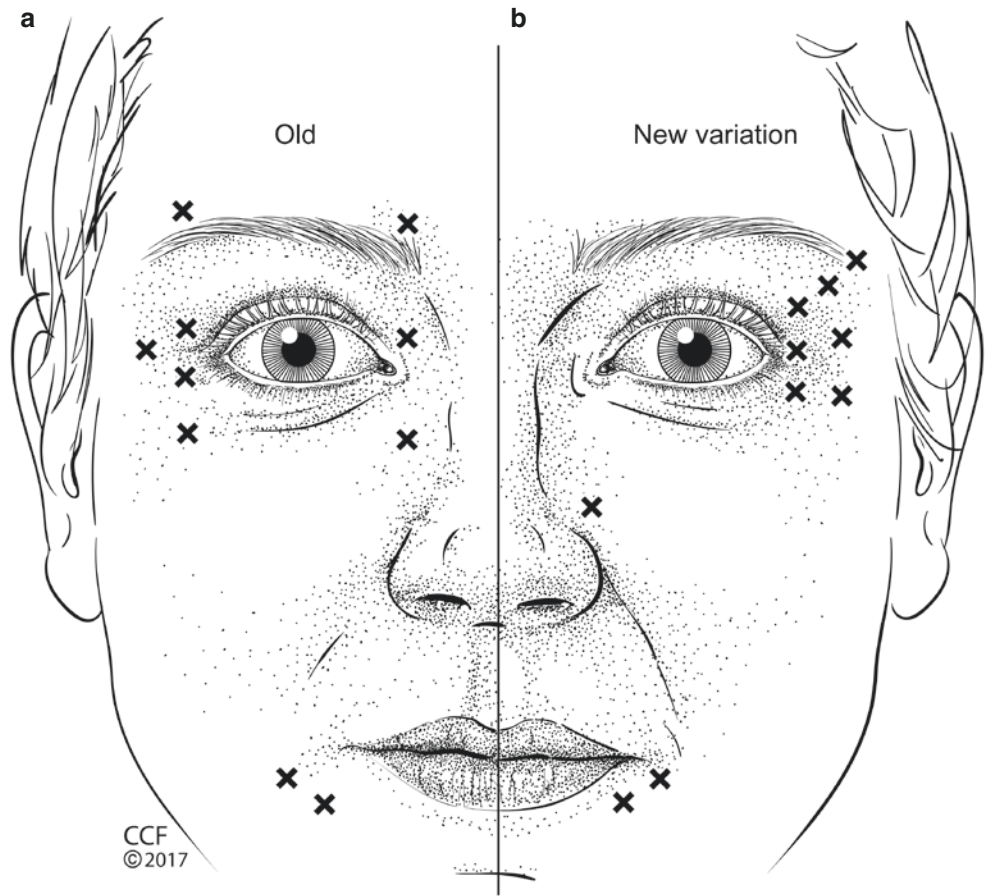


Fig. 32.2 Because the lateral orbicularis extends 2.5 cm outside the orbital rim, injections farther lateral may achieve greater paralysis while decreasing chances of ptosis and diplopia. Printed with permission, Cleveland Clinic Center for Medical Art & Photography © 2017. All Rights Reserved

Step 3

Injections are then given into the nasolabial fold, upper lid, chin, and other musculature of the face. However, the retractor muscles at the corner of the mouth should be avoided to prevent drooping of the mouth.

Step 4

Sensitivity to botulinum toxin varies among patients as does their underlying facial musculature. So, the sites and doses of injections can be modified according to the patient's response. Due to the associated facial palsy, a dose lower than that needed for blepharospasm is usually sufficient. Approximately 25–30 U of Botox are usually necessary to treat the side of the face affected by HFS.

Step 5

After injection, profound paralysis of injected muscles begins in approximately 24–48 h and lasts for approximately 2–3 months. Patients are given artificial tears to guard against dry eye syndrome due to a change in eyelid dynamics, which is a common side effect. Most patients report a satisfactory

improvement in their condition with a sustained benefit from repeated injections.

Complications

Complications are usually a result of paralysis of the facial musculature. Ptosis can be prevented by avoiding injections in the central part of the upper eyelid. Drooping of the angle of the mouth can be prevented by avoiding injections in this area. Lagophthalmos, mild ectropion, and tearing can occur with orbicularis paralysis. However, treating the orbicularis farther from the orbital rim likely decreases the chances of ptosis and diplopia while paralyzing the large portion of this muscle that exists well into this region. The use of artificial tears is a safeguard against exposure keratopathy. No major systemic side effects have been reported with the use of botulinum toxin.

References

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2. Walker TJ, Dayan SH. Comparison and overview of currently available neurotoxins. *J Clin Aesthet Dermatol*. 2014;7:31–9.