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Short report

Botulinum toxin treatment of hemifacial spasm

JS ELSTON

From the Institute of Ophthalmology and Moorfields Eye Hospital, London, UK

SUMMARY Six patients with hemifacial spasm were treated with injections of botulinum toxin A into the orbicularis oculi; the abnormal movements around the eye were relieved for an average of 15 weeks. There were no systemic or significant local side effects, and in view of the risks involved in neurosurgical treatment, a trial of botulinum toxin injections is recommended in the first instance in this condition.

Idiopathic hemifacial spasm consists of irregular, unilateral clonic twitching or more sustained contractions of the facial muscles. Of insidious onset in the 5th or 6th decades, affecting females more than males, it often starts in the orbicularis oculi and progresses in a stereotyped way, to involve all the facial muscles, including the platysma. A mild facial weakness may develop, but facial sensation is normal, and there are no other neurological signs.[2] The spasms are a social and psychological problem, and may also be uncomfortable, but are only of functional significance if vision is poor in the contralateral eye.

Hemifacial spasm persists during sleep, is unaffected by a stroke and recurs following distal seventh nerve section when the nerve regenerates[3]: all these features suggest that a disturbance of either the facial nucleus or the facial nerve proximal to the stylomastoid foramen is responsible. Electrophysiological studies show a synkinesis between the contractions around the eye and other muscle groups,[4–6] consistent with either a peripheral nerve disturbance, and ephaptic transmission at the site of damage,[7] or central synaptic re-organisation.[8] Peripheral nerve damage causing deafferentation of the facial nucleus with subsequent axonal sprouting and functional reconnection could explain the consistency of the physical signs.[1]

Compression of the facial nerve by a posterior fossa tumour or angioma[10] or other distorting influence such as basal meningitis[11] may cause the condition. It has been suggested that idiopathic cases are due to vascular compression of the facial nerve at its root exit zone, either by the basilar artery,[12] a named branch such as the posterior inferior cerebellar artery, or small unnamed vessels.[2 4 8 13] Even a minute venule has been held responsible.[14] Vertebral angiography is normal in these cases, and the evidence is based on the findings at retromastoid craniectomy, and the relief of symptoms when a sponge is interposed between the abnormal vessel and the nerve. The electrophysiological findings revert to normal after this procedure.[8 15] It is a safe operation, but although up to 87·5% of patients have an excellent or good result,[16] the recurrence rate may be up to 25% over the next two years[16] and up to 25%[17] of patients suffer permanent unilateral deafness or facial palsy.

The neurovascular relations at the facial nerve root exit zone are complex, and anomalies are commonly found at necropsy.[18] Only four out of 16 patients in one series were thought to have a vascular abnormality at operation, but 14 were relieved of spasm by wrapping a sponge around the nerve.[19] In all operative series, the spasms often take several months to resolve, and it may be that either the operative manipulation of the nerve, or subsequent fibrosis in relation to the sponge is responsible.[15] Alternative forms of treatment, however, are not often helpful. Drugs usually have no influence, although carbamazepine may be useful.[20] Partial peripheral nerve section distal to the stylomastoid foramen,[3] selective neurectomy[21] or thermocoagulation[22] are complicated by facial weakness and recurrence of the spasm. Transtympanic facial nerve needling has the same problems. Following experience with injections of botulinum toxin A in the

Address for reprint requests: Dr JS Elston, Institute of Ophthalmology, Judd St, London WC1H 9QS, UK.

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Table *Clinical features and results of treatment of six cases of hemifacial spasm*

Case No	*Sex*	*Age at presentation (yr)*	*Side*	*Duration of symptoms (yr)*	*Abnormal signs*	*Previous treatment*
1	M	58	L	2	Nil	Nil
2	F	62	L	8	Mild facial weakness	Peripheral facial nerve section
3	M	65	R	3	Mild left paresis	Nil
4	F	48	L	5	Nil	Drugs, Psychotherapy
5	F	68	R	7	Mild facial weakness	Drugs
6	M	54	R	2	Nil	Nil

management of blepharospasm,[23] I have used this drug in six cases of hemifacial spasm.

Patients, Materials and Methods

Hemifacial spasm was diagnosed in six patients (table). The history, similar in all cases, was of spasms of closure of one eye presenting in the 5th to 7th decade, and progressing to involve all the facial muscles of the same side, with increasing frequency. There were no other neurological signs apart from mild facial weakness in cases 2 and 5, and a residual mild left hemiparesis following a stroke in case 3. Plain skull radiographs, in all cases, and CT head scan in cases 1, 4, and 6 were normal. There had been no response to any drugs or other treatments such as acupuncture and the condition had recurred after peripheral nerve section in case 2.

Botulinum toxin A (1·5 μg) was injected in 1 ml saline in four divided doses into the orbicularis oculi as previously described.[22] Two weeks later, in cases 2 and 4, a further 0·4 ng toxin was injected into the buccolabial musculature on the same side. Follow up thereafter was at monthly intervals, or at any stage if the spasms around the eye recurred.

Results

The abnormal movements around the eyes were relieved in all patients within 3 or 4 days after the injection. As the spasms resolved, tissue fluid tended to accumulate in the eyelids, but a true ptosis, caused by spread of the toxin to the levator palpebrae superioris only occurred in cases 1 and 3 after the first injection. It was mild and resolved over two weeks, and there were no systemic side effects. Injection of the buccolabial musculature in cases 2 and 4 relieved the lower facial spasms, but caused a mild temporary facial weakness, which resolved over 10 to 12 weeks.

The spasms around the eye began to return gradually after between 12 and 20 weeks (average 15·3 weeks, see table). The longest period of relief occurred in case 2, who had previously had a peripheral nerve section. All patients have responded well to a second, and in case 1 a third injection around the eye.

Discussion

Idiopathic hemifacial spasm is an uncomfortable, unremitting social handicap, but it is not life threatening and any treatment must have a very low morbidity to be acceptable. Although a neurosurgical procedure on the facial nerve relieves the condition in a high proportion of cases, there is a significant risk of side effects, and of recurrence.[17] Elderly patients may not be suitable for such surgery, others may not accept the risks, and many neurologists are reluctant to advise it. Peripheral facial nerve surgery carries a recurrence risk of up to 60%, or may produce a lasting facial palsy.[3]

Paralysing the orbicularis oculi with botulinum toxin A is a simple, cheap and effective outpatient treatment that has no systemic side effects, and local side effects resolve rapidly. Although it is possible to paralyse selectively any of the muscles involved in the spasms, extending the injections away from the eye produced a mild facial weakness. The major symptoms in hemifacial spasm, however, are due to spasmodic eye closure, and abolishing this alone relieved discomfort and provided cosmetic improvement in all patients. With an average of 15·3 weeks of relief of symptoms, four injections would be needed per year, and would be practicable since there is no evidence of an immune response to the toxin at the doses used.[24] Botulinum toxin A treatment is thus a simple means of controlling the major symptomatic manifestation of hemifacial spasm.

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of treatments	*Other sites of injection*	*Side effects*	*Period of relief of symptoms (Weeks)*
	Nil	Mild ptosis	12
	Buccolabial muscles	Nil	20
	Nil	Mild ptosis	14
	Buccolabial muscles	Nil	12
	Nil	Nil	17
	Nil	Nil	16

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