

¹Adductor Laryngeal Dystonia (Spastic Dysphonia): Treatment with Local Injections of Botulinum Toxin (Botox)

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Summary: Adductor spastic dysphonia (SD) is a laryngeal dystonia characterized by a choked, constrained voice pattern with breaks in vocal flow. Treatment with a variety of therapies including speech and pharmacotherapy have minimal benefit; only one-third of patients undergoing recurrent laryngeal nerve section have benefitted at 3 years. We have used local injections of botulinum toxin (Botox) bilaterally into vocalis muscles in 42 patients with SD. Injections were through a teflon-coated hollow electromyography (EMG) recording needle. Unilateral small doses (2.5–3.75 U) were of no clinical benefit. Bilateral small doses resulted in sustained improvement lasting 84.4 ± 9.3 days. The degree of improvement was $61.1 \pm 4.6\%$. Common side-effects included a brief period of breathy hypophonia (8.5 ± 2.5 days) and a mild sensation of choking/aspiration of fluids (1.7 ± 0.6 days); there were no serious adverse effects. Vocal cord paralysis was not necessary for benefit. Follow-up vocalis muscle EMGs revealed denervation. All patients responded to retreatment (longest follow-up 3.5 years). Patients with prior recurrent laryngeal nerve surgery and residual uncomplicated dysphonia had similar results. Our results indicate that local injection of low-dose Botox is the treatment of choice for SD. **Key Words:** Spastic dysphonia—Adductor—Abductor—Botox—Vocalis muscles—Breathy hypophonia—Botulinum toxin—Dystonia—Laryngeal dystonia.

Dystonia is a neurological condition characterized by abnormal involuntary movements causing sustained or intermittent inappropriate postures (1). Spastic dysphonia (SD) is dystonia involving the vocalis muscles resulting in either a

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strained and strangled, effortful speech pattern with breaks and pauses (adductor type) or breathy, whispering speech (whispering, abductor type) (2). The relationship of SD to other focal, segmental, and generalized dystonias is established, and has been reviewed (3,4). Similar to the other focal or cranial-cervical dystonias, onset is usually as an adult and progression beyond cranial structures is uncommon. Treatment (Table 1) with nonmedical and medical therapies is typically with minimal benefit. Laryngeal nerve section may provide initial relief, but relapses are common (5). The cause for relapse may be due to hyperadduction of the nondenervated cord with exaggeration of the dystonic spasms, or reinnervation on the surgical side (6).

We have used local injections of botulinum toxin (Botox) to relieve dystonic contractions in blepharospasm, torticollis, oromandibular, vocal cord, tongue and limb dystonia, and hemifacial spasm, and reported our initial experience with a small number of patients in our pilot studies begun in the summer of 1984 (7-9). Miller (10) and Ludlow (11) have employed similar approaches in the treatment of spastic dysphonia. We now present our experience using this modality for the treatment of adductor spastic dysphonia in 42 patients.

PATIENTS AND METHODS

The study was approved by the Institutional Review Board. Patients clinically diagnosed with adductor spastic dysphonia were invited to participate in the study. In order to confirm the diagnosis, patients underwent a detailed neurologic and otolaryngologic assessment. Videotapes and voice recordings were performed before treatment; fiberoptic laryngoscopy was performed to evaluate for any anatomic abnormalities and to confirm hyperadduction with talking. In questionable cases, the videotapes were reviewed. Initially, we invited only patients who had previously failed treatment with pharmacotherapeutic agents. Because of the remarkable success of this modality compared with other available treatments, this treatment was subsequently offered to patients as primary therapy. We invited both nonsurgical patients and those who had previously undergone laryngeal nerve section or crush procedures. Xylocaine injections into the recurrent laryngeal nerve were not performed before therapy with Botox; our experience had demonstrated that failure to respond to xylocaine injections performed elsewhere did not predict response to therapy at our Center. Injections of Botox were performed as an outpatient procedure except for the postsurgical patients.

TABLE 1.^a *Laryngeal dystonia: classical treatments*

Nonmedical: psychotherapy, acupuncture, speech therapy, hypnosis, biofeedback
Pharmaco-Rx: anticholinergics, benzodiazepines, baclofen, carbamazepine, etc.
Surgery: recurrent laryngeal nerve crush/section, arytenoid displacement, laser therapy

^a See (5,10,15-23).

Because of the theoretical risk of stridor in the latter group of patients, they were hospitalized for 3 days after injection for observation.

Botox was obtained from Dr. Alan Scott (San Francisco, CA). Frozen lyophilized toxin was reconstituted with normal saline (without preservative) to a final concentration of 25 U/ml. Toxin was drawn into a tuberculin syringe and injected through a monopolar, hollow teflon-coated injection electromyography (EMG) needle (Fig. 1A) connected to an EMG recorder via an alligator clip attached to the hub of the needle. The otolaryngologist with experience in performing laryngeal EMGs performed all injections. The needle was placed into the thyroarytenoid vocalis complex by impaling the muscle through the crico-thyroid cartilage (Fig. 1B), using a previously described technique (12). Once the muscle was identified, the toxin was slowly injected. The calculation of the dose to be injected evolved as we gained experience with injecting this small muscle, as described below.

Patients were scored in a nonblinded fashion using a subjective rating, estimating the percent of normal speech, where 0% = no speech, or full disability, and 100% = normal speech. Patients scored themselves before each injection and a videotape and voice recording was performed. In addition, the physicians made a similar rating and the most conservative rating was recorded and used as the percent improvement in all cases. Patients were instructed to telephone one of the senior authors (MFB or AB) within 3 days; follow-up was scheduled for 2 weeks after the injection. For patients living long distances and unable to return to the medical center, telephone interviews were conducted; some sent us an updated videotape or cassette recording. Patients kept diaries of vocal function, noting the time of onset of vocal weakness, peak weakness, duration of breathy hypophonia, duration of choking, or aspiration of liquids, and any other notable events. No correlation was made between the observed improvement and vocal fold paralysis. With our technique, using a very low dose, vocal fold paralysis was decidedly rare, and a correlation was not possible.

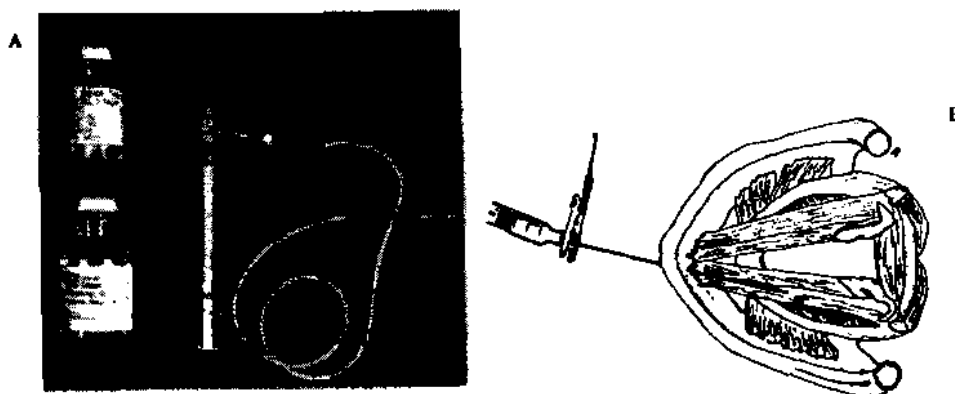


FIG. 1. A: A 27-gauge electromyographic-injection needle used for local injections of botulinum toxin when injected into the vocalis muscles. The wire attached to the alligator clip is used as input into the EMG machine. B: Needle in place in the vocal fold.

Postinjection EMG studies were performed on 2-week follow-up in a limited number of patients early in our study. The studies were not carried out systematically because they did not appear to have direct clinical relevance.

RESULTS

Forty-two patients participated; four had had previous surgery. Patient characteristics are summarized in Table 2. Eight patients had another relative with dystonia, including one with focal laryngeal dystonia, and another with segmental cranial dystonia affecting larynx and neck muscles.

Nonsurgical Cases

At the inception of this study, no patient ever had Botox injected into the vocalis muscles; therefore, our treatment program was initially empiric. Recognizing that the vocalis muscle is small, our initial strategy comprised three goals: (a) to minimize the total dose of toxin administered, (b) to minimize vocalis muscle trauma, and (c) to minimize adverse effects. Our first approach at finding the appropriate dose for an effective first-treatment program was to inject one vocalis muscle with 2.5–7.5 U Botox. The first patient was given 2.5 U Botox into one vocalis muscle; there was neither benefit nor adverse effects. The second patient was given 7.5 U into one vocalis muscle with 95% improvement lasting 118 days. The injection was initially followed by 25 days of breathy hypophonic speech. The third patient was given 7.5 U Botox into one vocalis muscle with no change in the clinical characteristics of her speech. The fourth patient was given 3.75 U Botox into one vocalis muscle with no effect. Following unilateral injection of Botox, all patients demonstrated a sluggish vocal cord, but not paralysis, on direct laryngoscopy. We could not explain why one patient responded and the other three did not have any clinical effect. Miller (10) and Ludlow (11) subsequently reported that the minimum dosage to achieve an effect with the unilateral injection technique is 15 U.

Given that only one patient had a response to unilateral injections at a maximum dose of 7.5 U, we had the option of either increasing the dose administered to one

TABLE 2. *Characteristics of patients with laryngeal dystonia: treatment with Botox*

42 patients (21 males, 21 females)
Mean age onset 38.8 ± 2.7
Mean duration of symptoms $10.0 \text{ yr} \pm 1.7$
Etiology
Idiopathic 39
Tardive 2
Birth injury 1
Distribution of dystonia when treated
Focal 26
Segmental cranial 7
Multifocal 6
Generalized 3

vocal cord, or injecting both cords with a low dose. In keeping with our stated goal of minimizing the total exposure to Botox, we decided to explore bilateral, low-dose injections. We therefore initiated a first-treatment program of injecting both vocalis muscles with 3.75 U Botox. Thirteen patients were treated in this manner; all experienced a beneficial effect (Table 3). Typically, the patient would observe an initial softening of the voice within the first day; maximum hypophonia, when present, occurred by the third day. Hypophonic, breathy speech typically lasted 4–14 days and was mild, but lasted 57 days in one patient and was severe and quite disabling. Some patients also experienced a sensation of choking when drinking fluids; there were no episodes of aspiration. They compensated by drinking slowly until this adverse effect resolved. Patients requested re-injection before returning to their pretreatment level of functioning. The mean pretreatment level of function was $26.1 \pm 3.3\%$, $n = 30$; the re-injection level of function was $41.7 \pm 8.1\%$, $n = 12$ ($p < 0.05$ for mean function injection #1 vs. #2). Toxin was readministered when clinically significant disabling symptoms returned.

When performed, 2-week follow-up vocalis muscle EMGs revealed evidence of denervation.

TABLE 3. *Laryngeal dystonia: treatment with Botox (Finding an effective first treatment program)*

Dose injected = 3.75 U/cord (N = 13)	Bilateral injections		
	Mean	SEM	Range
Onset of effect	1.1 days	0.1	0.2–2 days
Peak effect	2.8 days	0.7	0.9–8 days
Duration breathy hypophonia (N = 6)	9.1 days	5.1	0.0–57 days
Duration choking (N = 2)	2.0 days	1.1	0.0–10 days
Minimum duration benefit (N = 10) ^a	89.8 days	20.8	45–270 days
Prior function	28.6%	5.9	0–60%
Best postfunction	90.0%	3.9	60–100%
Improvement (best-prior)	61.4%	8.3	15–100%
Dose injected = 2.5 U/cord (N = 20)			
Onset of effect	1.0 days	0.2	0–2.0 days
Peak effect	2.0 days	0.3	0–4.0 days
Duration breathy hypophonia (N = 10)	8.1 days	2.5	0–25 days
Duration choking (N = 6)	1.5 days	0.6	0–6 days
Minimum duration benefit (N = 16) ^a	77.5 days	8.3	20–140 days
Prior function	23.3%	3.5	0–50%
Best postfunction	84.4%	4.4	50–100%
Improvement (best-prior)	61.1%	5.4	10–92%
Dose injected = combined series of 3.75 and 2.5 U/cord (N = 33)			
Onset of effect	1.0 days	0.1	0–2 days
Peak effect	2.3 days	0.3	0–8 days
Duration breathy hypophonia (N = 16)	8.5 days	2.5	0–57 days
Duration choking (N = 8)	1.7 days	0.6	0–10 days
Minimum duration benefit (N = 26) ^a	84.4 days	9.3	20–270 days
Prior function	26.1%	3.3	0–60%
Best postfunction	86.7%	3.1	50–100%
Improvement (best-prior)	61.1%	4.6	10–100%

Dose = 2.5 vs. 3.75 U/cord, NS $p < 0.05$, for all indices.

^a Number of patients available for analysis with >3 months follow-up.

Because of the prolonged period of breathy hypophonia in some patients (Table 3), we modified our first-treatment protocol by reducing the injected dose to 2.5 U/cord. The results are summarized in Table 3 and were not found to be significantly different from those seen when administering 3.75 U/cord. Although there was a trend for decreased duration of breathiness, choking, and duration of benefit at the lower dose, the difference was not statistically significant. The incidence of these adverse effects was not reduced in the group receiving the lower dose. Further analysis with a larger cohort will be required to determine whether there are fewer side-effects at the lower dose. However, two patients failed to respond clinically at the lower dose injected. Toxin had been successfully administered to other patients the same day and from the same lot of toxin, suggesting that the etiology of nonresponse was not due to inactivity of the toxin. These two patients subsequently returned and responded to repeat injections.

Because of the lack of significant difference in the duration or degree of benefit at the two doses, our current protocol is to initially inject 2.5 U/cord except when the patient finds it impossible to return for a return visit in 2 weeks should the injection have no effect. In such a situation, the patient receives an injection of 3.75 U/cord.

For repeat injections, we initially injected either 2.5 or 3.75 U/cord. However, we have begun to explore unilateral injections of 2.5 U Botox *on follow-up* in an effort to minimize adverse effects with retreatment. We are exploring this approach because patients return for follow-up injections before fully returning to baseline disability. This suggests that there may be some residual weakness in the vocalis muscles, and re-injecting one cord while the other cord is still slightly weak may be adequate to permit a clinical response. Once again, our approach is empiric; the goal is to minimize the total exposure to toxin.

There are too few follow-up injections utilizing this strategy for statistical analysis. However, some patients experience significant benefit. Those who have no effect return in 2 weeks for an injection of 2.5 U into the opposite cord.

Surgical Cases

Four patients presented for treatment after having undergone recurrent laryngeal nerve section, crush, or other procedures (Table 4). All had evidence of vocal cord paralysis with midline vocal cords on fiberoptic laryngoscopy. Because of our concern about the possibility of laryngeal stridor following injection of Botox in an individual with one cord paralyzed, all patients were hospitalized for the procedure and 3 nights of observation.

The clinical effect, summarized in Table 4, is similar to the nonsurgical group. However, Patient C had no clinical improvement; the voice was slightly more fluent but his voice became unacceptably breathy. On review of his videotape, he may have had a mixture of adductor and abductor dysphonia; weakening the remaining cord may have exacerbated the abductor component. The diagnosis of this patient was problematic as he had had multiple surgical procedures on the recurrent laryngeal nerve, and direct attack on the vocalis muscles. Because of his desperate situation, we elected to treat him with the understanding that the diag-

TABLE 4. *Laryngeal dystonia: treatment with Botox (postsurgical cases)*

Patient	A	B	C	D
Yr between surgery and Botox	9	5	5	4
Dose injected (U)	3.75	2.5	2.5	2.5
Onset of effect (days)	0.8	1.5	2.0	1.0
Peak effect (days)	2.0	2.0	3.0	3.0
Duration breathy hypophonia (days)	10	0	— ^a	0
Duration choking (days)	0	0	0	0
Minimum duration benefit (days)	70	60	0	>30 ^b
Prior function (%)	30	50	10	45
Best postfunction (%)	80	95	10	70
Improvement (best-prior, %)	50	45	0	25

Patient A,B: left recurrent laryngeal nerve (RLN) section; patient C: left RLN crush followed by RLN section and two left vocal cord thinning procedures.

^a See text; ^b recently injected.

nosis was not absolute. This was the only patient in this series in whom the diagnosis was problematic.

Two patients have received subsequent injections with similar good results.

Adverse Effects

Adverse effects are summarized in Table 5. As noted previously, breathy hypophonia ranged in severity from mild to severe, and when prolonged, was disabling. Choking on fluids was mild in all cases. Hyperventilation occurred during the period of breathiness when the patient attempted to speak loudly and the cords were too weak to adduct; no patient lost consciousness. All patients were instructed not to force their speech and were referred to a speech therapist following the return of phonation in order to maximize speech function without abusing the vocal cords. Because one patient developed a severe sore throat when injected during the resolution of a viral upper respiratory infection (URI), we have adopted a policy of injecting patients only when they have no residual evidence of a URI. On two occasions, a patient reported feeling a drop of fluid in the pharynx during the injection, and had no clinical effect from that injection. We suspect that the

TABLE 5. *Laryngeal dystonia: adverse effects associated with 95 treatments of local injections of Botox into the vocalis muscles*

N	%	Adverse effect
43	45	Breathy hypophonia ^a
21	22	Mild choking on fluids
2	2	Hyperventilation
1	1	Coughed slight blood tinged sputum
1	1	Viral sore throat; injected at tail end of a viral upper respiratory infection
1	1	Puritis without a rash

^a In our rating, we probably over-rate "breathiness" as any evidence of a hypophonic voice. This might explain why we have a high percentage of patients with breathiness. Breathless aphonia was rare.

EMG-injection needle tip had protruded through the vocal cord into the laryngeal airway and the toxin never entered the muscle.

Long-term follow-up was 0-6 months for 24 patients, 7-12 months for 9, 13-18 months for 6, and >33 months for 3. One patient has dropped out of the study because of the travel distance to New York. One patient was lost to follow-up. No patients have become refractory to therapy. We examined our database to determine if there was a progressively longer period of benefit between injections (Table 6). A trend was apparent, but there were insufficient data for formal analysis.

Patient acceptance was unusually high. Most patients were ecstatic with the results. Comments included: "I am having a great time with my new toy (voice)"; "I could read a book to my kid for the first time"; "My pupils clapped when I came in the room"; "I can't believe it . . . after all of these years"; "My efficiency in selling stocks has soared"; "I can work again after being out of work for 7 years"; "I am finally enrolled in courses . . . I couldn't return to college before you fixed my voice"; and from a mother: "Previously we could not understand her at all . . . now we can listen to her at the dinner table."

DISCUSSION

Laryngeal dystonia is a disabling condition for which treatments have previously had limited value. In our experience, local injection of Botox into the vocalis muscles is a safe, effective, and satisfactory mode of therapy. For most patients, we currently inject 2.5 U into each vocalis muscle for the initial treatment. However, for patients who find it a hardship to travel to New York, we inject 3.75 U/cord for the first visit. When possible on follow-up, we re-inject alternate vocal cords with 2.5 U/cord.

Ludlow (11) and Miller (10) have injected higher doses of toxin (up to 60 U over three visits) into one vocalis muscle in patients with spastic dysphonia. Because the goal of this treatment scheme is to cause paralysis of the injected cord, we see no advantage to their techniques over surgical therapy. We postulate that surgery may ultimately fail because the remaining innervated cord hyperadducts and subsequently augments the return of speech dysfunction. We have considered that their approach may permit the opposing cord to hyperfunction and hyperadduct, thereby aggravating the dystonic state, promoting additional dysfunction. However, their recent studies indicate that the intrinsic muscle abnormalities on the

TABLE 6. Average duration of benefit after sequential treatments

Injection no.	N	Mean benefit (days)	SEM
1	33*	86.3	8.3
2	14	85.4	11.8
3	7	101.9	7.5
4	6	108.0	15.6
5	4	110.0	25.6

* Patients referenced in Table 3.

noninjected side were normalized following unilateral injections (13). On theoretical grounds, a higher dose may cause excessive, and possibly unnecessary, traumatic damage to the injected cord (14). Although Ludlow (11) examined patients up to 4 months following injection and reported no evidence of trauma, and our experience with the lower dose injections concurs, the long-term effect on the muscle physiology is not known.

Botox injection therapy (both bilateral and unilateral) has advantages over surgical treatments, as noted in our previous publications (7,8,9). First, the injections are performed with the patient awake and there is no risk of anesthesia. Second, graded responses of muscle weakening can be obtained by using low dosages initially and then repeating the injections to achieve the desired result. Once patient sensitivity to the toxin is determined and the dose is titrated for each patient, an individualized treatment protocol can be used for future injections. Third, if breathy hypophonia occurs, the weakness wears off and phonation returns. Fourth, the procedure is acceptable to the patients and they are satisfied with the results. Finally, the procedure is less costly than surgery.

It is too soon to tell whether patients will become refractory to this form of therapy. Symptom relapse after recurrent laryngeal nerve surgery approaches 60% after 5 years of follow-up (5). Although we are greatly concerned about the potential long-term complications of injecting a potent toxin into the vocalis muscle, we have abandoned recommending surgery to our patients and currently recommend this successful form of therapy as primary therapy. Other less invasive therapies should also be explored.

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LEGENDS TO THE VIDEOTAPES

PATIENT 1. This 64-year-old woman went to a funeral in Europe at the age of 62 and subsequently developed difficulty speaking. Her speech became progressively coarser and interrupted as the day progressed; then the problem became persistent. After telling a joke or laughing, the voice would normalize for just a few words. Benzodiazepines and anticholinergics were of no benefit. This problem markedly interfered with her job as a teacher. The initial segment was performed 3 years after onset on June 2, 1987, just prior to injecting 2.5 U Botox into each vocalis muscle. The second segment was performed 3 weeks later on June 23.

PATIENT 2. This 37-year-old man developed a mild sore throat at the age of 34. The coarsening of his voice did not resolve and he developed progressive constriction of his voice pattern. The first video segment is prior to his first injection of 2.5 U Botox per vocal cord. He became fluent that evening; there was no aspiration or breathiness. The second segment is 2 weeks after the injection.

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