Epidemiologic Overview of Synkinesis in 353 Patients with Longstanding Facial Paralysis under Treatment with Botulinum Toxin for 11 Years

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Background: Patients with longstanding facial paralysis often exhibit synkinesis. Few reports describe the prevalence and factors related to the development of synkinesis after facial paralysis. Botulinum toxin type A injection is an important adjunct treatment for facial paralysis–induced asymmetry and synkinesis. The authors assessed the clinical and epidemiologic characteristics of patients with sequelae of facial paralysis treated with botulinum toxin type A injections to evaluate the prevalence of synkinesis and related factors.
Methods: A total of 353 patients (age, 4 to 84 years; 245 female patients) with longstanding facial paralysis underwent 2312 botulinum toxin type A injections during an 11-year follow-up. Doses used over the years, previous treatments (electrical stimulation, operations), and how they correlated to postparalysis and postreanimation synkinesis were analyzed.
Results: There was a significant association between cause and surgery. Most patients with facial paralysis caused by a congenital defect, trauma, or a tumor underwent reanimation. There were no sex- or synkinesis-related differences in the doses used, but the doses were higher in the reanimation group than in the no-surgery group. Synkinesis was found in 196 patients; 148 (41.9 percent) presented with postparalysis synkinesis (oro-ocular, oculo-oral) and 58 (16.4 percent) presented with postreanimation synkinesis. Ten patients presented with both types.
Conclusions: This study determined the high prevalence (55.5 percent) of synkinesis in patients with longstanding facial paralysis. Postparalysis synkinesis was positively associated with infectious and idiopathic causes, electrical stimulation, facial nerve decompression, and no requirement for surgery. Postreanimation synkinesis was present in 28.2 percent of reanimated patients and was significantly associated with microsurgical flaps, transfacial nerve grafting, masseteric-facial anastomosis, and temporalis muscle transfers. (*Plast. Reconstr. Surg.* 136: 1289, 2015.)

Facial paralysis is a disturbing condition that affects the aesthetic, functional, and psychological aspects of the affected individual. Patients with longstanding facial paralysis often exhibit asymmetry and synkinesis, even after successful reanimation surgery.[1–9] The paralyzed side may display signs such as shallow nasolabial folds, depression of the angle of the mouth, a lower eyebrow, flaccidity, lagophthalmos, and ectropion. The nonparalyzed side often exhibits wrinkles, furrows, and deviation of the nose and mouth toward the nonparalyzed side at rest and during facial mimics because the normal muscles

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act chronically against the weak antagonism of the contralateral muscles.[2] The overall facial asymmetry is exacerbated by aging and synkinesis. Synkinesis is defined as involuntary muscle contraction triggered by another voluntary movement and results from aberrant regeneration of facial nerves. Synkinesis is a common and unpleasant sequela of facial paralysis. All of the above-mentioned factors contribute to psychological disorders, low self-esteem, and poor quality of life.[2,3,10–12]

Since 1987, botulinum toxin type A injection into the nonparalyzed side has been used for the treatment of asymmetries caused by facial paralysis.[1–3,6,10,13,14] Botulinum toxin type A injection has shown promising results and improvement of quality of life when used as an adjuvant treatment of surgical reanimation.[2,4,5] In a series of 25 patients with facial paralysis, the asymmetry coefficient was significantly reduced by 48.4 percent after 1 month secondary to inhibition of exaggerated movements on the nonparalyzed side and improvement in the rates of movement on the paralyzed side. Although the basal rates of movement on the nonparalyzed side normalized after 6 months, a permanent 16.8 percent reduction in the asymmetry coefficient was accomplished because of this rehabilitation effect.[2,15] A subsequent series of 55 patients with facial paralysis who underwent treatment with onabotulinum toxin A or abobotulinum toxin A confirmed these data: the authors observed a permanent 9 percent reduction in the coefficient of asymmetry after 6 months.[16]

This effect on the paralyzed side after botulinum toxin type A injection in the nonparalyzed side could be attributable to facilitation of muscle activity and/or nerve regeneration, brain plasticity, or both. This observation was demonstrated in an experimental study. After nerve injury followed by nerve-graft reanimation surgery, the authors found a significant increase in functional recovery in rats that received botulinum toxin type A injections in contralateral muscles.[17]

More recently, small doses of botulinum toxin type A have also been injected on the paralyzed side to correct synkinesis.[10–12,18] Synkinesis seems to be related mainly to decompression of the facial nerve, electrical stimulation, and idiopathic and infectious causes.[15] However, few reports have described the factors related to the development of facial synkinesis. Although the true prevalence of synkinesis after facial paralysis is not clear, the reported prevalence varies from 8.9 percent to more than 51.0 percent.[3,12,15,16,19–21]

The objective of this study was to analyze the epidemiologic characteristics of patients with facial paralysis who underwent botulinum toxin type A injection to correct asymmetry. The follow-up period was 11 years. The prevalence of and factors related to the development of synkinesis were determined in patients with facial paralysis sequelae.

PATIENTS AND METHODS

The study protocol was approved by the Ethics Committee for the Analysis of Research Projects of the Hospital das Clínicas of the Medical School of the University of São Paulo (São Paulo, Brazil). A retrospective study was carried out from 2003 to 2014 involving 353 patients (age, 4 to 84 years; median, 40.0 years; mean, 39.8 ± 16.5 years) presenting with long-term facial paralysis asymmetry who attended plastic surgery outpatient clinics and were administered a botulinum toxin type A injection. We included patients who had experienced symptoms for more than 1 year. Patients with spastic paralysis and asymmetry attributable to craniofacial deformities were excluded from the analyses.

In total, 69.4 percent of patients were female patients. The right side was affected in 186 patients (52.7 percent), the left side in 162 patients (45.9 percent), and both sides in five patients (1.4 percent). The time from onset of facial paralysis symptoms varied up to 65 years (mean, 11.15 ± 11.6 years; median, 7.0 years). The cause of facial paralysis is shown in Figure 1. Infectious cause included patients with facial paralysis sequelae secondary to different infectious agents: seven patients with herpes zoster, five patients with chronic otitis media, two patients with toxoplasmic encephalitis, two patients with facial cellulitis, one patient with staphylococcal infection, one patient with cytomegalovirus infection, and one patient with Hansen disease secondary infection. The vascular cause included four patients who presented with facial paralysis as a sequela of an ischemic stroke and two hemangiomas. Patients in whom the cause could not be determined or with Bell palsy were classified as idiopathic cause.

Synkinesis was classified into two types according to the primary cause. Postparalysis synkinesis referred to the development of sequelae caused by facial paralysis itself, and postreanimation synkinesis referred to the development of sequelae after the performance of reanimation operations.

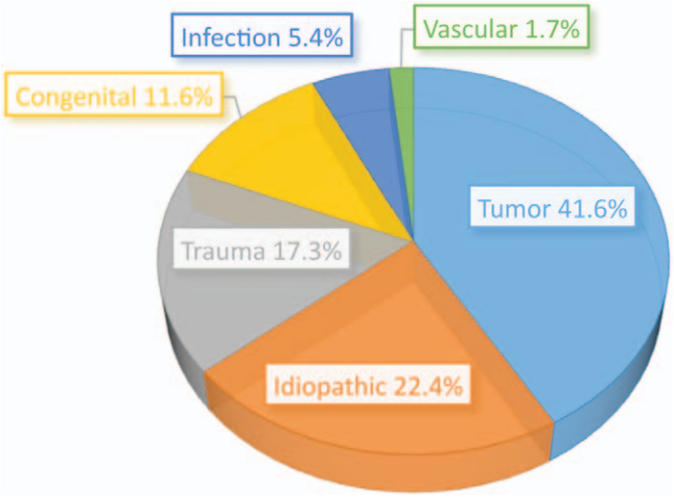

Fig. 1. Distribution of the causes of facial paralysis in the sample.

Previous Treatments

Operations were performed according to the specific need of each patient by another research team in our department of the Hospital das Clínicas of the Medical School of the University of São Paulo. A total of 761 operations had been performed previously. The number of operations per patient ranged from zero to 10 (median, 2.0; mean, 2.1 ± 2.0).

Table 1. Classification of Surgery Groups According to the Operations Carried Out, and Number of Procedures in the Total Sample*

	No.
Reanimation	
No. of operations	314
Transfacial sural nerve grafts	97
Microsurgical muscle transfers	83
Temporalis muscle transfers	75
Masseteric-facial anastomosis	18
Hypoglossofacial anastomosis†	12
Nerve grafts	11
Nerve sutures	10
Facial nerve decompression‡	8
Symmetrization	
No. of operations	212
Static suspension of the mouth§	81
Myectomies	34
Fat grafts	34
Brow lift	30
Rhytidoplasty	27
Neurectomies	6
Eye	
No. of operations	235
Gold weights	95
Canthopexy	91
Blepharoplasty	18
Cartilage grafts	10
Cartilage weights	10

*The symmetrization operations were distributed among the reanimation and symmetrization groups, and the eye operations were distributed among the reanimation, symmetrization, and eye surgery groups.
†Performed by neurosurgeons.
‡Performed by ear, nose, and throat surgeons.
§Performed with or without grafting of the palmaris longus tendon.

The patients were divided into four groups according to their surgical treatments: reanimation, symmetrization, eye operations, and no surgery. Table 1 presents the classification according to operations performed, with the number of operations of each type in the present sample. A patient assigned to the reanimation group could also have undergone symmetrization and eye operations, and a patient assigned to the symmetrization group could have undergone eye operations but no reanimation operations. Patients who did not undergo any of the above-mentioned operations were assigned to the no-surgery group. The demographics of each group are presented in Table 2. Previous treatment with electrical stimulation was found in 143 patients (40.5 percent).

Botulinum Toxin Type A Treatment

According to the brand of drug available on the day of the procedure, patients were treated with either Botox (Allergan, Inc., Irvine, Calif.) or Dysport (Ipsen Biopharm, Ltd., Slough, United Kingdom). The Botox 100-U vial was diluted in 2 ml of 0.9% (physiologic) saline solution. The Dysport 500-U vial was diluted in 4 ml of 0.9% saline, thereby resulting in a 1:2.5-U equivalence ratio when similar volumes were injected. We considered 1 U to be equal to 0.02 ml of the solution.

The dose applied to each muscle group on the nonparalyzed side varied according to the specific need of each patient, as outlined in the previously established Hospital das Clínicas of the Medical School of the University of São Paulo protocol.[2]

Table 2. Demographic Profile of Each Surgery Group

Surgery Group	No. (%)	Mean Age ± SD (yr)	Median Age (yr)	Female Sex (%)
Reanimation	206 (58.3)	40.0 ± 16.0	40.0	69.9
Symmetrization	21 (6.0)	50.6 ± 18.4	58.0	85.7
Eye surgery	24 (6.8)	38.2 ± 17.5	36.0	58.3
No surgery	102 (28.9)	37.2 ± 16.5	40.0	67.6

Two representative patients and the dose used in each injection point are shown in Figures 2 and 3. Strong or disfiguring synkinesis was also treated with low-dose injections on the paralyzed side.[12] The total dose per patient was that needed for satisfactory symmetrization as evaluated by the first author (A.G.S.) and the patient. If symmetrization was not satisfactory after 2 weeks, additional points were used and added to the total dose for that session. Side effects were assessed after 1 month. The number of treatments each patient received and the interval between the sessions were assessed.

Statistical Analyses

Statistical analyses were carried out using IBM SPSS Version 20 (IBM Corp., Armonk, N.Y.). A value of $p < 0.05$ was considered statistically significant. According to the Shapiro-Wilk test, the data were not distributed normally; therefore, nonparametric tests were used. The Mann-Kendall test was used to assess upward or downward trends of variables over time (dose in each session and interval between sessions).

The mean dose used for each patient was calculated. The Mann-Whitney *U* test was used to assess differences between the categorical variables sex and synkinesis related to the dose. The Kruskal-Wallis (analysis of variance) test was used to determine differences between surgery group related to age and the dose used, followed by the Mann-Whitney *U* test with Bonferroni correction for paired comparisons between groups. The likelihood ratio test (a type of chi-square test) was used to determine the degree of association between categorical variables (surgery group versus cause, surgery group versus gender, synkinesis versus cause, synkinesis versus electrical stimulation, synkinesis versus surgery group, and synkinesis versus operations).

RESULTS

Demographic Data

There was no difference regarding sex between the surgery groups ($p = 0.848$), but age was higher in the symmetrization group ($p = 0.008$). There

Fig. 2. A 29-year-old patient with facial paralysis of the left side after resection of a parotid tumor with immediate nerve suture. She was smiling before her first treatment (42 U of botulinum toxin type A) (*left*), after 1 month (*center*), and before her tenth treatment (*right*). Some improvement of movement on the paralyzed side can be seen after treatment.

Fig. 3. A 19-year-old patient with left-sided facial paralysis after resection of a vascular tumor (hemangioma). She was smiling before (*left*) and after (*right*) her first and sixth injections (25 U and 31 U of botulinum toxin type A, respectively). Previous operations included a transfacial nerve graft, microsurgical gracilis muscle flap, right-zygomatic myectomy, suspension with the palmaris longus tendon, and canthoplasty. Botulinum toxin treatment refined reanimation results.

was a significant association between cause of facial paralysis and the performance of surgery ($p < 0.001$). Most patients with facial paralysis of tumor-related, trauma-related, and congenital causes underwent reanimation (74.8, 57.4, and 48.8 percent, respectively). Among patients with idiopathic and infectious causes, a very similar prevalence was observed between the reanimation and no-surgery groups (Table 3).

Botulinum Toxin Type A Treatment

In total, 2312 botulinum toxin type A injections were administered. The number of treatments per patient varied from one to 22

Table 3. Distribution of the Surgery Groups According to the Cause of Facial Paralysis*

Cause of Paralysis	N	Reanimation		Symmetrization		Eye Surgery		No Surgery	
		n	%	*n*	%	*n*	%	*n*	%
Tumor	147	110	74.8	6	4.1	9	6.1	22	15.0
Idiopathic	79	32	40.5	6	7.6	5	6.3	36	45.6
Trauma	61	35	57.4	1	1.6	4	6.6	21	34.4
Congenital	41	20	48.8	4	9.8	3	7.3	14	34.1
Infection	19	6	31.6	4	21.1	2	10.5	7	36.8
Vascular	6	3	50	0	0	1	16.7	2	33.3
Total	353	206	58.2	21	5.9	24	6.8	102	29.1

*There was a significant association between cause of facial paralysis and performance of surgery ($p < 0.001$).

(median, 5.0; mean, 6.5 ± 5.4). Patients were told to return for a new session after 6 months. The median interval between sessions was 196 days, and this interval was consistent over time (Fig. 4).

The total dose used per patient in each session ranged from 2 to 106 U (median, 36.3 U; mean, 38.5 ± 17.3 U). An increasing trend in the total dose was observed from sessions 1 to 21. We also monitored the dose over time after excluding all sessions involving a dose of less than 15 U, and an upward trend was still observed (Fig. 5).

The statistical analysis regarding doses used according to each variable is shown in Table 4. There were no significant sex- or synkinesis-related differences in the dose used. The Mann-Whitney *U* test with Bonferroni correction was used for paired comparisons between the surgery groups and showed that the total dose was higher in the reanimation group than in the no-surgery group ($p < 0.001$). No other differences were detected between the surgery groups.

Synkinesis

General facial synkinesis was documented in 196 patients (55.5 percent). Of those, 148 (41.9 percent of the 353 patients) presented with postparalysis synkinesis and 58 (16.4 percent) presented with postreanimation synkinesis. Ten patients presented with both types simultaneously.

Table 5 presents the analysis of the presence of postparalysis synkinesis according to each variable. There was a significant association between cause and postparalysis synkinesis ($p < 0.001$). Causes more closely associated with postparalysis synkinesis were infectious (68.4 percent of 19 patients) and idiopathic (58.2 percent of 79 patients). Congenital cause exhibited the weakest association with synkinesis (19.5 percent of 41 patients).

There was a significant association between electrical stimulation and postparalysis synkinesis ($p = 0.002$); it was present in 51.7 percent of patients treated with electrical stimulation versus 35.2 percent of patients who did not receive this treatment. There was also an association of the presence of postparalysis synkinesis with the surgery group ($p < 0.001$). The no-surgery group had the highest prevalence of postparalysis synkinesis (65.7 percent) and the reanimation group had the weakest association (28.6 percent with postparalysis synkinesis). Postparalysis synkinesis was present in seven of eight

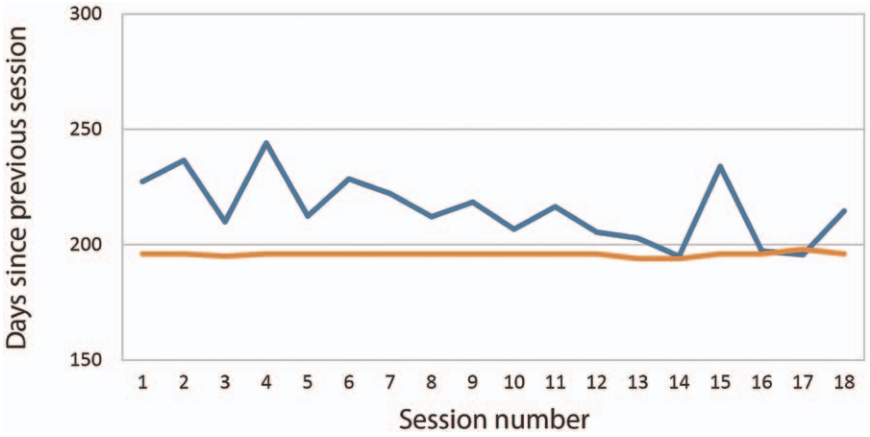

Fig. 4. Mean (*blue line*) and median (*red line*) interval between treatment sessions, showing no upward or downward trend over time.

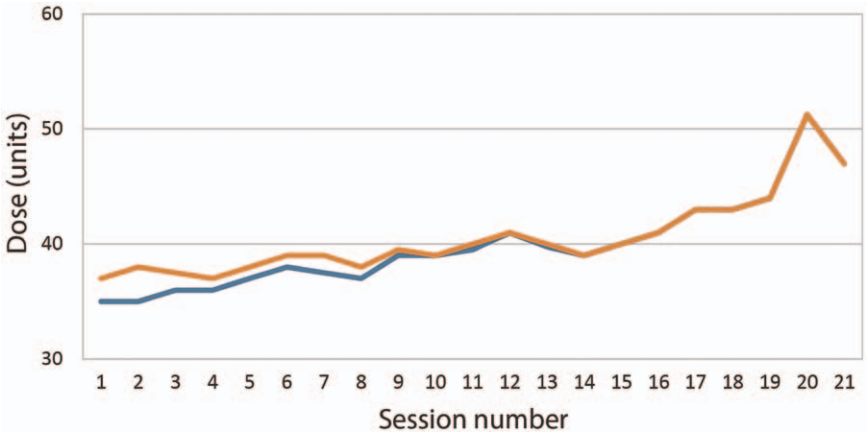

Fig. 5. Upward trend in the total dose used in each session (*red*) and upward trend in the total dose after excluding sessions involving less than 15 U (*blue*).

patients who had previously undergone facial nerve decompression; this association was significant ($p = 0.007$).

Table 6 presents analysis regarding the presence of postreanimation synkinesis according to the different variables. It was not associated with cause of paralysis ($p = 0.076$) or with electrical stimulation ($p = 0.466$). Postreanimation synkinesis could be found only in the reanimation group ($p < 0.001$). Of the 206 patients in the reanimation group, 58 (28.2 percent) developed postreanimation synkinesis. The association between postreanimation synkinesis and microsurgical flaps, transfacial nerve grafts (cross-face procedures), masseteric-facial anastomosis, and temporalis muscle transfers was statistically significant. Factors significantly associated with the presence of postparalysis and postreanimation synkinesis are summarized in Table 7.

DISCUSSION

In our country, patients with all possible causes and presentations of facial paralysis are referred to the Hospital das Clínicas of the Medical School of the University of São Paulo, thereby making the patient cohort described in this article very representative for epidemiologic analyses. These patients underwent reanimation (as needed) by another research team in the plastic surgery department. They also underwent myofunctional rehabilitation with speech therapists.

Table 4. Comparison of the Dose of Botulinum Toxin Type A According to Different Variables

Variable	Mean Dose ± SD (U)	Median (U)	*p*
Sex			0.252
Female	34.8 ± 15.0	35.0	
Male	37.2 ± 17.43	37.6	
Synkinesis			0.894
With synkinesis	35.7 ± 16.0	34.2	
No synkinesis	35.2 ± 15.45	37.0	
Surgery group			<0.001
Reanimation	39.43 ± 13.5	39.2	
Symmetrization	32.2 ± 17.0	32.0	
Eye surgery	34.0 ± 16.1	36.2	
No surgery	28.7 ± 17.2	29.5	

Most studies of botulinum toxin type A injection in patients with facial paralysis have been small series (≤69 patients), and few have described the protocols for injection points and doses.[2,16,20,22–26] The present study is the largest series of treatments performed by the same surgeon over 11 years (2312 applications in 353 patients) and involving active screening for synkinesis and other disorders. A high level of patient satisfaction with the outcomes of this procedure has been documented in terms of quality-of-life evaluations.[16,20] We used a published protocol regarding the criteria for the indications and doses in each muscle group.[2] We used onabotulinum toxin A or abobotulinum toxin A using a unit-conversion ratio of 1.0:2.5. This ratio was found to be the most appropriate after first testing a protocol with an equivalence ratio of 1:3.[16]

Side effects were self-limited and related to facial muscle weakness, including mild difficulty in speaking (especially phonemes /p/ and /b/), drinking, or eating and, in some cases, difficulty in adapting to the smaller smile in the first month after injection. There were no serious complications such as ptosis, diplopia, or resistance. Patients who also received low doses of botulinum toxin type A on the paralyzed side for management of synkinesis did not have an increased prevalence of side effects.

Table 5. Distribution of the Patients with Postparalysis Synkineses According to the Different Variables Analyzed

Variable	No.	Postparalysis Synkineses ($n = 148$) (%)	p
Cause			<0.001
Tumor	147	49 (33.3)	
Idiopathic	79	46 (58.2)	
Trauma	61	29 (47.5)	
Congenital	41	8 (19.5)	
Infectious	19	13 (68.4)	
Vascular	6	3 (50.0)	
Electrical stimulation			0.002
Yes	143	74 (51.7)	
No	210	74 (35.2)	
Surgery group			<0.001
Reanimation	206	59 (28.6)	
Symmetrization	21	8 (38.1)	
Eye surgery	24	14 (58.3)	
No surgery	102	67 (65.0)	
Early operation			0.007
Facial nerve decompression	8	7 (87.5)	

Table 6. Distribution of the Patients with Postreanimation Synkinesis According to the Different Variables Analyzed*

Variables	No.	Postreanimation Synkinesis ($n = 58$) (%)	p
Cause			0.076
Tumor	147	31 (21.1)	
Idiopathic	79	10 (12.7)	
Trauma	61	5 (8.2)	
Congenital	41	7 (17.1)	
Infectious	19	5 (26.3)	
Vascular	6	0 (0.0)	
Electrical stimulation			0.466
Yes	143	26 (18.2)	
No	210	32 (15.2)	
Surgery group			<0.001
Reanimation	206	58 (28.2)	
Symmetrization	21	0 (0.0)	
Eye surgery	24	0 (0.0)	
No surgery	102	0 (0.0)	
Operation (reanimation group)			
Facial nerve decompression	8	1 (12.5)	0.753
Microsurgical flaps	83	34 (41.0)	<0.001
Transfacial nerve grafts	97	28 (28.9)	<0.001
Temporalis muscle transfer	75	20 (26.7)	0.010
Masseteric-facial anastomosis	18	12 (66.7)	<0.001
Nerve sutures	10	1 (10)	0.554
Nerve grafts	11	4 (36.4)	0.104
Hypoglossofacial anastomosis	12	1 (8.3)	0.404

*The operations analyzed refer to patients normally included in the reanimation group. These patients could have been submitted to more than one operation; therefore, each operation was analyzed independently.

During subsequent sessions, the patients became better acquainted with the paralyzing effect and were more receptive to the treatment of additional muscles that had not been addressed initially (e.g., platysma, frontalis, nasal, and muscles of the paralyzed side with synkinesis). This patient acceptance was indicated by a progressive increase in the median dose along with an increase in the number of sessions. We did not observe clinical resistance over time. We will address the behavior of each muscle after subsequent sessions in a separate study.

The wide range in patient age (4 to 84 years) demonstrates that all patients can benefit from botulinum toxin type A treatment. Even the 31 patients who began treatment before the age of 16 years were treated satisfactorily using this protocol, with no need for anesthetic cream (which was not used because it is not available at Hospital das Clínicas of the Medical School of the University of São Paulo). As in other studies using botulinum toxin type A in patients with facial paralysis, our study cohort had a higher prevalence of female patients (69.4 percent), who tend to be more concerned than male patients about facial aesthetics.[13,15] Despite anecdotal data suggesting that men need higher doses, the dose required to achieve a satisfactory effect was not significantly different between male and female patients.

Patients with longstanding facial paralysis benefit considerably from two treatments of botulinum toxin type A per year. Long-term repeated injections have not shown evidence of loss of effectiveness or cumulative adverse effects.[27,28] We follow conventional recommendations for avoiding resistance, specifically, using a minimum effective dose that elicits a good effect, ensuring that there is a greater than or equal to 3-month interval between sessions, and avoiding booster injections.[29] The experience of the physician who administers the injection is important; however, careful follow-up of individual patients after each session will show better results and allow for appropriate adjustment of doses and injection points.

There was a significant association between cause and surgery. Most patients with facial paralysis caused by congenital disorders, trauma, or tumors had previously undergone reanimation surgery. In contrast, the numbers of patients who needed reanimation and those who did not undergo any surgery were similar in idiopathic and infectious causes. Simultaneously, the latter two causes showed a higher association with synkinesis than did other causes. These findings indicate that the absence of direct anatomical or

Table 7. Factors Associated with a Higher Prevalence of Each Type of Synkinesis

Postparalysis synkinesis
Infectious and idiopathic causes
Electrical stimulation
Partial paralysis with no need for reanimation surgery
Facial nerve decompression procedure
Postreanimation synkinesis
Microsurgical flaps
Transfacial nerve grafts (cross-face)
Masseteric-facial anastomosis
Temporalis muscle transfers

mechanical injury to nerves or muscles results in a tendency toward less severe presentations ("partial facial paralysis"); simultaneously, however, the number of patients with synkinesis increases because of aberrant growth of nerves. This observation is consistent with the finding that the no-surgery group, which included patients with less severe presentations of facial paralysis, had the highest prevalence of patients with postparalysis synkinesis (65.7 percent). In addition, the total dose of botulinum toxin type A needed in the reanimation group was higher than that in the no-surgery group because the reanimation group contained patients with more severe facial paralysis.

We also found a strong association between postparalysis synkinesis and facial nerve decompression. This finding can be explained by the fact that nerve decompression is carried out by ear, nose, and throat surgeons in patients with a worse prognosis after idiopathic facial paralysis. Because this operation is performed in the acute phase of the paralysis, it was the only reanimation operation analyzed for association with postparalysis synkinesis. These patients' synkinesis could not be characterized as postreanimation because their pattern was very diffuse; different, for example, from that of a patient submitted to a masseteric-facial anastomosis that presents a smile when he or she is eating. In the case of facial nerve decompression, we believe that the association with synkinesis was attributable not to the operation itself but to the severity of the presentation of these patients.

Facial synkinesis was found in 196 of 353 patients (55.5 percent). Of those, 148 (41.9 percent) presented with postparalysis synkinesis (oro-ocular, oculo-oral) and 58 (16.4 percent) had postreanimation synkinesis. Ten patients presented with both types simultaneously.

The term "postreanimation synkinesis" was introduced in the present study. This phenomenon has rarely been addressed when discussing the surgical treatment of facial paralysis. In one recent study,[21] synkinesis was seen in only 8.9 percent of patients with facial paralysis caused by iatrogenic trauma to the facial nerve induced during surgical intervention, whereas synkinesis was seen in 38.5 percent of patients with an idiopathic cause, similar to our findings. Those authors also found that 51 percent of patients developed synkinesis after reanimation. The mean time to onset of postoperative synkinesis was 12.7 ± 7.4 months and was significantly associated with transplantation of the gracilis muscle and transposition of the temporalis muscle to the eye.[21] In the present study, 58 of the 206 patients (28.2 percent) in the reanimation group developed postreanimation synkinesis. This prevalence was significantly associated with microsurgical flaps, transpositions of the temporalis muscle, cross-face procedures, and masseteric-facial anastomosis. The most likely reason for the lower prevalence of postreanimation synkinesis in our study cohort is the longer follow-up duration after the reanimation procedures, which provided rehabilitation time for some movements to become automatic.

There was a significant association between electrical stimulation and postparalysis synkinesis, which was found in 51.7 percent of patients who underwent previous treatment with electrical stimulation compared with only 35.2 percent of patients who did not. Like other authors, our research team does not recommend electrical stimulation; however, the high number of patients who underwent this treatment in the acute phase highlights the importance of disseminating the results of the present study.[30,31]

CONCLUSIONS

This study allowed for determination of the actual prevalence of synkinesis in patients with longstanding facial paralysis (55.5 percent) and differentiation between postparalysis and postreanimation synkinesis. Postparalysis synkinesis (41.9 percent) was positively associated with infectious and idiopathic causes, electrical stimulation, facial nerve decompression, and no requirement for surgery. Postreanimation synkinesis was present in 28.2 percent of reanimated patients and was significantly associated with microsurgical flaps, transfacial nerve grafting, masseteric-facial anastomosis, and temporalis muscle transfers.

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PATIENT CONSENT

Patients provided written consent for the use of their images.

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