Facial palsy remains a challenging clinical entity, the underlying cause of which ranges from life-threatening conditions to self-resolving idiopathic disease. Although unilateral facial palsy is relatively common, having a reported incidence of 20 to 25 per 100,000 population, bilateral facial palsy is a rare clinical entity with an incidence of approximately one per 5 million and an occurrence of 0.3 to 2 percent in facial palsy cases. Although Bell palsy is the most common cause of unilateral facial palsy, the underlying etiologic factor in bilateral facial palsy is often an underlying medical condition such as Lyme disease, Guillain-Barré syndrome, leukemia, infectious mononucleosis, or trauma, and may require hospital admission for prompt diagnosis and management. This article describes a management approach to the patient with bilateral facial palsy and summarizes presenting features, diagnosis, and interventions performed on all patients who presented with or developed bilateral facial palsy while under our care at a tertiary care facial nerve center over the past 13 years.

**Background:** Bilateral facial palsy is a rare clinical entity caused by myriad disparate conditions requiring different treatment paradigms. Lyme disease, Guillain-Barré syndrome, and leukemia are several examples. In this article, the authors describe the cause, the initial diagnostic approach, and the management of long-term sequelae of bilateral paralysis that has evolved in the authors’ center over the past 13 years.

**Methods:** A chart review was performed to identify all patients diagnosed with bilateral paralysis at the authors’ center between January of 2002 and January of 2015. Demographics, signs and symptoms, diagnosis, initial medical treatment, interventions for facial reanimation, and outcomes were reviewed.

**Results:** Of the 2471 patients seen at the authors’ center, 68 patients (3 percent) with bilateral facial paralysis were identified. Ten patients (15 percent) presented with bilateral facial paralysis caused by Lyme disease, nine (13 percent) with Möbius syndrome, nine (13 percent) with neurofibromatosis type 2, five (7 percent) with bilateral facial palsy caused by brain tumor, four (6 percent) with Melkerson-Rosenthal syndrome, three (4 percent) with bilateral temporal bone fractures, two (3 percent) with Guillain-Barré syndrome, one (2 percent) with central nervous system lymphoma, one (2 percent) with human immunodeficiency virus infection, and 24 (35 percent) with presumed Bell palsy. Treatment included pharmacologic therapy, physical therapy, chemodenervation, and surgical interventions.

**Conclusions:** Bilateral facial palsy is a rare medical condition, and treatment often requires a multidisciplinary approach. The authors outline diagnostic and therapeutic algorithms of a tertiary care center to provide clinicians with a systematic approach to managing these complicated patients.

**Patients and Methods**

The institutional review board at the Massachusetts Eye and Ear Infirmary approved this study. A chart review encompassing all patients seen with bilateral facial palsy at our facial nerve center between January of 2002 and January of 2015 was performed. Demographics, symptoms, diagnosis, initial treatment, and subsequent interventions, including physical therapy, chemodenervation, and surgery, were reviewed. All patients were reviewed.

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with bilateral facial palsy, whether synchronous (involvement of the contralateral side within 30 days) or asynchronous (involvement of the contralateral side after 30 days), were included.4,10 Patients were classified according to the long-term state of each side of the face, either flaccid facial paralysis or nonflaccid facial palsy, characterized by hypertonicity and synkinesis.11,12

RESULTS

Of the 2471 patients seen at our center, 68 patients (3 percent) whose initial history, presentation, or clinical course involved bilateral facial palsy were identified. Nine patients (13 percent) presented with congenital disease; of the acquired cases, 21 patients (36 percent) had synchronous bilateral facial palsy and 38 patients (64 percent) had asynchronous bilateral facial palsy (Fig. 1). The female-to-male ratio was 1.44:1, with a mean age of 26 years (range, 0 to 69 years). The causes of bilateral facial palsy in this series are listed in Table 1.

Although bilateral involvement in Bell palsy is rare, it was the most common diagnosis among patients with bilateral facial palsy identified in this study (Fig. 2 and Table 2); importantly, all 24 cases presented in asynchronous fashion. Of these 24 patients, 20 had residual deficits from the original Bell palsy and developed nonflaccid facial palsy before experiencing the contralateral episode. Two patients completely recovered, and two patients were lost to follow-up. Most of these patients experienced typical prodromal symptoms, including facial numbness, otalgia, and retroauricular pain preceding the facial paralysis. Magnetic resonance imaging scans were obtained for 17 patients, and revealed typical abnormal enhancement along the distal meatal, labyrinthine, and proximal tympanic segments of the seventh cranial nerve in 10 patients. Nineteen patients were treated with a course of steroids and antiviral agents, four patients received steroids alone, and one patient refused steroidal treatment. Patients who went on to develop nonflaccid facial palsy were offered physical therapy

![Fig. 1](image-url). Causes for synchronous and asynchronous bilateral facial palsy (BFP). CNS, central nervous system.
and chemodenervation with botulinum toxin. The subset of patients with unsatisfactory results following a regimen of physical therapy and chemodenervation were offered static and dynamic surgical interventions. Facial nerve decompression was considered in patients with recurrent ipsilateral facial palsy or patients with severe nonflaccid facial palsy who presented acutely with flaccid facial paralysis on the contralateral side, and was successfully performed in three patients.

Ten patients presented with bilateral facial palsy caused by Lyme disease (Fig. 2 and Table 2). Nine patients developed synchronous facial paralysis affecting the contralateral side within 21 days. One patient developed facial palsy on the contralateral side 3 years after the disease. All patients were treated with appropriate antibiotics, and some also received steroids and/or antiviral agents. In all cases, the initially paralyzed side was slower to recover than the contralateral side. All patients went on to develop hypertonicity and synkinesis to varying degrees, which was worse on the side initially paralyzed. Initial treatment included physical therapy and chemodenervation, followed by static and dynamic surgical reanimation procedures as required.

The third most common diagnosis of bilateral facial palsy was Möbius syndrome, occurring in nine patients (Fig. 2 and Table 2). All patients were noted to have bilateral sixth and seventh cranial nerve deficits and presented with bilateral flaccid facial paralysis. On examination, the patients displayed the typical foreshortened upper lip (Fig. 3). For facial reanimation, patients were offered physical therapy, bilateral free gracilis transfer, and upper lip augmentation/elongation.

Nine patients (13 percent) in our series developed bilateral facial palsy following treatment for bilateral vestibular schwannomas resulting from neurofibromatosis type 2 (Fig. 2 and Table 2). Four patients had complete bilateral flaccid facial paralysis, three patients had bilateral nonflaccid facial palsy, and two patients demonstrated unilateral flaccid facial paralysis and contralateral nonflaccid facial

| Conditions                        | No. (%) |
|----------------------------------|---------|
| Congenital                       | 9 (13)  |
| Idiopathic                       |         |
| Möbius syndrome                  | 9 (13)  |
| Acquired                         | 59 (87) |
| Infectious                       |         |
| Viral                            |         |
| Bell palsy                       | 24 (35) |
| Human immunodeficiency virus     | 1 (2)   |
| Bacterial                        |         |
| Lyme disease                     | 10 (15) |
| Vascular malformation            |         |
| Brainstem cavernous hemangioma   | 4 (6)   |
| Benign neoplastic                |         |
| Bilateral vestibular schwannomas (NF2) | 9 (13) |
| Pilocytic astrocytoma            | 1 (2)   |
| Hematologic malignancy           |         |
| CNS lymphoma                     | 1 (2)   |
| Traumatic                        |         |
| Bilateral temporal bone fractures| 3 (4)   |
| Autoimmune                       |         |
| Guillain-Barré syndrome          | 2 (3)   |
| Idiopathic granulomatous         |         |
| Melkersson-Rosenthal syndrome    | 4 (6)   |

NF2, neurofibromatosis type 2; CNS, central nervous system.

**Table 1. Congenital and Acquired Conditions of Bilateral Facial Palsy**

![Fig. 2. Diagnoses of bilateral facial paralysis. CNS, central nervous system; HIV, human immunodeficiency virus.](image-url)
Table 2. Causes of Bilateral Facial Palsy

| Cause                     | Presenting Features/ Diagnostic Criteria                                                                 | Type of Paralysis                | Diagnostics and Management of Underlying Cause                                      | Management of FP                                                                 |
|---------------------------|----------------------------------------------------------------------------------------------------------|----------------------------------|---------------------------------------------------------------------------------------|----------------------------------------------------------------------------------|
| Bell palsy                | Rapid onset (within 72 hr) Uniform involvement with or without otalgia, facial numbness                   | Initially flaccid, then progresses to full recovery or NFFP | Steroids with or without antivirals, prednisone 60 mg orally for 5 days followed by 10 mg/day taper for 5 days within 72 hr; valacyclovir, 1 g orally twice daily for 7 days | Acutely: Corneal protection and eyelid stretching; NFFP: PT, botulinum toxin type A, surgery (e.g., platysmectomy, HSN) |
| Lyme disease              | CDC criteria (signs/symptoms and/or positive Lyme titer)                                                | Same as in Bell palsy            | Antibiotic, doxycycline 100 mg orally twice daily, amoxicillin or cefuroxime 500 mg orally three times per day for 28 days | Same as for Bell palsy                                                          |
| Guillain-Barré syndrome   | Migrating, symmetric, areflexic flaccid paralysis; BFP accompanied by weakness of the extremities and absence of tendon reflexes | NFFP or FFP                      | Nerve conduction studies and cerebrospinal fluid analysis                             | Normal full recovery if not based on presentation                                |
| HIV                       | Often during seroconversion, may include prodrome of systemic viral infection or aseptic meningitis      | Most of the time flaccid, some patients show some facial movement | ELISA confirm with Western blot, viral load and CD4 count -> HAART; steroids, prednisone 60 mg orally for 5 days followed by 10 mg/day taper for 5 days within 72 hr | Often spontaneous complete recovery                                               |
| Möbius syndrome           | Signs and symptoms Underdevelopment cranial nerves VII, VI (sometimes involvement of V, IX, X, and XII) Foreshortened upper lip and inability lateral gaze | Most of the time flaccid, some patients show some facial movement | Clinical history, patient examination, neurology consultation                          | Eye: weight/spring Midface: suspension Lip: elongation Smile: temporalis, free gracilis by V |
| Postsurgical (vestibular schwannoma resections in NF2) | Neurologic lesions Eye lesions Skin lesions | Flaccid (if not grafted or failed graft) NFFP (if successful graft) | Neurotology/neurosurgery consultation Stereotactic radiosurgery if brainstem compression Removal of skin lesions for aesthetics Targeted therapy (e.g., against VEGF) | Ipsilateral trigeminal (masseteric or deep temporal nerve transfer), one-stage gracilis free muscle transfer innervated by masseteric nerve, temporalis muscle or tendon transfer, and static reanimation techniques |
| Melkersson-Rosenthal syndrome | Episodes of facial palsy, lingua plicata, and orofacial edema                            | Progressive facial paralysis       | Patient history and examination, skin biopsy Glucocorticoids combined with minocycline, clofazimine, NSAIDs, and thalidomide | Nerve decompression Based on appearance of NFFP or FFP                           |

BFP, bilateral facial palsy; FP, facial palsy; NFFP, nonflaccid facial palsy; PT, physical therapy; FFP, flaccid facial palsy; HSN, highly selective neurorctomy; CDC, Centers for Disease Control and Prevention; ELISA, enzyme-linked immunosorbent assay; HAART, highly active antiretroviral therapy; VEGF, vascular endothelial growth factor; NSAIDs, nonsteroidal antiinflammatory drugs.

Palsy. Individualized treatment plans were offered, including physical therapy, chemodenervation, and surgical interventions, and were tailored to therapy of the primary disease. For example, cessation of bevacizumab therapy before surgical intervention was orchestrated with the neurooncology team.

Three patients had bilateral facial palsy caused by bilateral temporal bone fracture, two
occurring from motor vehicle accidents and one from a bitemporal head crush injury (Fig. 2). All were treated postoperatively with extensive physical therapy, with varying degrees of spontaneous recovery and further therapy dictated by their final recovery.

Four patients presented with Melkersson-Rosenthal syndrome (Fig. 1 and Table 1). Of these, one presented with the classic symptoms of facial paralysis, facial edema, and lip swelling, whereas three patients showed only the fissured pattern on the tongue with facial paralysis (Fig. 3). All patients reported recurrent episodes. Patients received dual therapy with glucocorticoids and antiviral agents. All patients had incomplete recovery resulting in bilateral nonflaccid facial palsy. Patients were offered facial nerve decompression in the acute setting and physical therapy and chemodenervation when there was evidence of any kind of movement, or when they presented with long-term sequelae of nonflaccid facial palsy. They were offered facial reanimation surgery when, after an exhaustive conservative management (chemodenervation and physical therapy), they remained without a meaningful smile. One patient chose to undergo facial nerve decompression surgery.

Two patients presented with bilateral nonflaccid facial palsy caused by Guillain-Barré syndrome; both were offered physical therapy and chemodenervation (Fig. 2 and Table 2). Four patients who experienced flaccidity for greater than 6 months, with no expectation of recovery after surgery for cavernous brainstem hemangioma, were offered static and dynamic surgical interventions. One patient presented with two episodes of bilateral flaccid facial paralysis and abducens palsy after aggressive chemotherapy with carboplatin and vincristine, because of recurrent pilocytic astrocytoma. Initial treatment included extensive physical therapy resulting in nearly complete recovery. One patient presented with bilateral flaccid facial paralysis and was diagnosed with central nervous system lymphoma after extensive diagnostic workup. The patient was initially treated with antiviral medications and corticosteroids for presumptive Bell palsy.

One human immunodeficiency virus–positive patient experienced acute onset of bilateral flaccid facial paralysis accompanied by fever and vomiting. Progression to bilateral nonflaccid facial palsy was noted after 2 months. Initial treatment included physical therapy and chemodenervation, followed by static and dynamic surgical reanimation procedures.

DISCUSSION

Bilateral facial paralysis remains a diagnostic challenge. Although the pathophysiology of asynchronous bilateral facial palsy is typically associated with localized inflammatory conditions believed to result in neural ischemia (e.g., viral reactivation, granulomatous disease), the pathophysiology of synchronous bilateral facial palsy typically involves a more systemic insult, including neurotoxic or autoimmune processes (e.g., Lyme disease, human immunodeficiency virus, Guillain-Barré syndrome) and massive trauma (e.g., bilateral temporal bone fractures) (Fig. 1). In contrast to unilateral facial palsy, bilateral facial palsy presents often as a manifestation of a serious systemic condition; as such, a comprehensive history and physical examination together with appropriate blood work and imaging studies necessary to establish the correct diagnosis in expedient fashion is critical. Synchronous bilateral facial palsy presenting in acute fashion (i.e., paralysis that develops rapidly) is frequently caused by trauma or infectious or autoimmune diseases (e.g., Lyme disease, human immunodeficiency
virus, Guillain-Barré syndrome), whereas slowly progressive bilateral facial palsy strongly suggests neoplastic disease such as central nervous system lymphoma. Diagnostic workup should include a complete blood cell count, fluorescent treponemal antibody test, human immunodeficiency virus test, fasting glucose level, erythrocyte sedimentation rate, Lyme titer, antinuclear antibody level, antineutrophil cytoplasmic antibodies, antiphospholipid antibody, and imaging with fine-cut contrast-enhanced magnetic resonance imaging or computed tomography of the temporal bones. In addition, lumbar puncture may be performed to evaluate cell counts and to establish bacterial or viral infection through enzyme-linked immunosorbent assay, Western blot, or polymerase chain reaction. Each disease has a unique pathophysiology, diagnostic management, and initial treatment, as listed in Table 2.

Bell palsy is an acute peripheral facial nerve palsy in patients for whom physical examination and history are otherwise unremarkable, consisting of deficits affecting all facial zones equally that fully evolve within 72 hours. To date, it remains a clinical diagnosis of exclusion. Strong evidence points to reactivation of herpes simplex virus type 1 within the geniculate ganglion as the underlying cause, with reactivation of herpes simplex virus type 1 resulting in destruction of ganglion cells, and infection of Schwann cells leading to demyelination, neural inflammation, and resultant ischemic compression at the level of the meatal foramen. Complete recovery to normal facial function occurs in approximately 70 percent of untreated cases, with permanently impaired facial function occurring to a minor degree in 13 percent and to a major degree in 16 percent of cases. Synchronous Bell palsy is exceedingly rare. In the acute phase, management options for Bell palsy include corneal protective measures, eyelid stretches, pharmacologic treatment with steroids and antivirals, and consideration of facial nerve surgical decompression; patients who go on to develop nonflaccid facial palsy may be offered physical therapy, chemodenervation, and static and dynamic reanimation procedures.

Lyme disease, a tick-borne disease caused by the spirochete Borrelia burgdorferi, is the most common vector-borne disease in the United States. Neuroborreliosis is a subacute manifestation of the disease, with Lyme disease–associated facial paralysis being its most common manifestation. Interestingly, in all cases, the side that was initially flaccidly paralyzed was last to recover, suggesting that the degree of insult relates to the rate of recovery. Long-term synkinetic sequelae are managed with physical therapy, chemodenervation, and surgical reanimation. Möbius syndrome is a congenital disease with facial paralysis and ocular abduction deficits because of underdevelopment of the facial (seventh) and abducens (sixth) nerves. Interventions include physical therapy to optimize any underlying movement, lip elongation surgery, and bilateral muscle transfer for smile reanimation.

Guillain-Barré syndrome is a rapidly progressing polyradiculoneuropathy, characterized by a migrating, symmetric (usually bilateral), areflexic flaccid paralysis. In 1956, a variant of Guillain-Barré syndrome was described by Miller Fisher, and involves a syndrome of ophthalmoplegia, ataxia, and areflexia, and can also include bilateral facial palsy. Most patients begin recovering within 4 weeks of onset without any treatment. Plasmapheresis or intravenous immunoglobulin therapy may hasten recovery in these patients. Patients who do not fully recover receive facial reanimation interventions either for flaccid facial paralysis or for nonflaccid facial palsy.

Melkerson-Rosenthal syndrome is a rare neuromyocutaneous granulomatous disorder of unknown cause and pathophysiology, characterized by lingua plicata (i.e., fissured tongue), orofacial edema, and recurrent and progressive episodes of facial palsy (often mischaracterized as Bell palsy). Symptoms may present in concurrent or asynchronous fashion. The therapy remains a challenge and is based on symptomatology, as the pathophysiology is unclear. Physical therapy may be used in acute flaccid facial paralysis and long-term nonflaccid facial palsy, together with static or dynamic surgical reanimation procedures. Facial nerve decompression has proven effective in preventing recurrence of facial palsy in Melkerson-Rosenthal syndrome.

Neurofibromatosis type 2 is an autosomal dominantly inherited neoplasia syndrome that predisposes to multiple tumors of the nervous system, including bilateral vestibular schwannomas. The management of neurofibromatosis type 2 is especially challenging because bilateral tumors place multiple cranial nerves at risk, including the facial and trigeminal nerves. Patients with unilateral facial paralysis may later develop bilateral facial palsy; therefore, contralateral facial nerve branches are not an ideal donor source for dynamic reanimation. Treatment options in neurofibromatosis type 2 patients include ipsilateral trigeminal (masseteric or deep temporal) nerve transfer, one-stage free gracilis muscle transfer.
Fig. 4. (Above) Patient photographs of central nervous system lymphoma presenting with multiple cranial neuropathies including bilateral flaccid facial paralysis (left to right: resting position, eye closure, smiling). (Center) Patient photographs of recurrent facial paralysis demonstrating bilateral nonflaccid facial palsy. (Below) Patient photographs of bilateral, asynchronous Bell palsy, demonstrating right nonflaccid facial palsy and left flaccid facial paralysis.
innervated by the masseteric nerve, temporalis muscle or tendon transfer, and static reanimation techniques. Rarely, progression of intracranial disease in neurofibromatosis type 2 patients will present with the clinical scenario of successful dynamic reanimation followed by decreased movement, as tumor growth impinges on the donor nerve.

Facial palsy is a condition that can be characterized by either flaccidity, hypertonicity and synkinesis, or a combination of both elements. Clinical sequelae include difficulty with eyebrow elevation, incomplete eye closure, poor corneal protection, difficulty expressing oneself nonverbally, and a wide spectrum of smile dysfunction. Lower lip abnormality, difficulty with speech and articulation, and oral competence can also be major factors. There are significant psychosocial implications of facial palsy as well. Patients with bilateral facial palsy are even more complex than those with unilateral sequelae, and sometimes intervention decisions need to be made independently for each side of the face. Patients with bilateral facial palsy may present with bilateral flaccid facial paralysis (flaccid facial paralysis/flaccid facial paralysis) (Fig. 4), bilateral nonflaccid facial palsy (nonflaccid facial palsy/nonflaccid facial palsy) (Fig. 4), and the highly disfiguring combination of flaccid facial paralysis on one side and nonflaccid facial palsy on the contralateral side (flaccid facial paralysis/nonflaccid facial palsy) (Fig. 4). These patients have a significantly decreased quality of life. Treatment strategies are varied and are based on type and severity of bilateral facial palsy (Fig. 4). Office procedures interventions include physical therapy\textsuperscript{11} and chemodenervation.\textsuperscript{3,19} Surgical interventions include highly selective neuroectomy for refractive synkinesis,\textsuperscript{20} nerve decompression based on Gantz criteria,\textsuperscript{30} platysmectomy,\textsuperscript{31} free gracilis transfer,\textsuperscript{3,32} eyelid weight,\textsuperscript{3,33} cross-face nerve graft,\textsuperscript{3,34} nasolabial fold modification, lip augmentation, asymmetric face lift, lid blepharoplasty, and external nasal valve correction.\textsuperscript{5} The role of facial nerve decompression in acute stages for patients with complete idiopathic or posttraumatic paralysis (e.g., in Bell palsy and Melkersson-Rosenthal syndrome) is gaining increasing importance. It is thought that the acute facial palsy results from a size mismatch between the nerve and the bony nerve canal caused by swelling of the nerve. Gantz et al.\textsuperscript{30} established diagnostic criteria for the consideration of neural decompression in unilateral Bell palsy using electroneurography and voluntary electromyography in the acute stage. If the electroneurography demonstrates more than 90 percent neural degeneration in comparison with the normal side and absent voluntary motor unit potentials, decompression should be considered within 14 days of symptom onset, giving the patient a greater chance for a better recovery.\textsuperscript{30,35,36} In cases of recurrent facial paralysis (i.e., relapsing-remitting) such as Melkersson-Rosenthal syndrome, facial nerve decompression may be offered in elective fashion to prevent future attacks.\textsuperscript{30,37} As neural decompression is not without risk, the ultimate decision lies with the patient in consultation with an experienced neuro-otologic surgeon.\textsuperscript{30,37,38} Further research is required to better define the role of facial nerve decompression in facial palsy.

**SUMMARY**

Bilateral facial palsy is a rare clinical entity caused by myriad disparate conditions requiring vastly different treatment paradigms. Synchronous bilateral facial palsy is often a manifestation of serious underlying disease, whereas asynchronous bilateral facial palsy warrants workup for granulomatous and autoimmune processes. In this article, we outline diagnostic and therapeutic algorithms of a tertiary care center, to share our maturing experience of managing this rare subset of facial paralysis patients.

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Volume 138, Number 4 • Bilateral Facial Paralysis

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