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## **Introduction**

From its etiology and pathogenesis to its appropriate management, much has been written over the past century regarding Bell's palsy, yet little has been accepted. Medical management with steroids and antivirals continues to be controversial. Surgical management has been even more unsettled with extremes of opinion continuing to range for those who believe surgical decompression is mismanagement to those who advocate early decompression for all patients with a complete facial paralysis (1). Thus, the clinician is often confused regarding the appropriate management of a patient presenting with an episode of acute facial paralysis due to Bell's palsy.

The term Bell's palsy has been used to describe a facial paralysis of acute onset and limited duration, the etiology of which was considered idiopathic in the past. The clinical presentation is typically defined by the rapid onset of the facial palsy, minimal associated symptoms, and spontaneous recovery. The diagnosis of Bell's palsy is made after the exclusion of all other possibilities. Even with this strict criteria, Bell's palsy remains the most common diagnosis given to patients with acute facial paralysis (2).

Fortunately, the etiology of acute facial paralysis due to Bell's palsy is becoming clearer. Historically, there have been two prevailing theories: 1) vascular congestion with secondary ischemia to the nerve and 2) viral polycranioneuropathy. McGovern (3) postulated autonomic vascular instability with spasm of the nutrient arterioles. This vasospasm would lead to ischemia, nerve edema, and secondary compression within the fallopian canal. The mechanisms responsible for such insults included cold temperatures or psychosomatic causes (3).

An alternative theory by Antoni (4) in 1919 proposed the term *acute infectious polyneuritis cerebri acusticofacialis*. McCormick (5) subsequently postulated the etiologic agent to be the herpes simplex virus in 1972. Recent studies by Murakami (6) strongly suggest that herpes simplex virus type 1 (HSV-1) is active in cases of Bell's palsy. In this study, endoneural fluid from 11 of 14 patients undergoing transmastoid decompression during the acute phase of the disease displayed DNA fragments of HSV-1 via the polymerase chain reaction. None from this surgical group displayed evidence of herpes zoster or Epstein Barr virus in the obtained samples. None of the affected controls undergoing surgery for temporal bone trauma or tumors displayed either HSV-1 or herpes zoster in their fluid samples either. Of the patients undergoing decompression for Ramsay Hunt syndrome, none exhibited DNA fragments of HSV-1 in their samples while all had herpes zoster. Indeed, Schirm and Mulkins (7) stated that the Murakami study was so well controlled that "it provides conclusive evidence that reactivation of HSV genomes from the geniculate ganglia is the most important cause of Bell's palsy."

Even more compelling was the finding of HSV-1 DNA in a temporal bone section of a patient dying six days after developing Bell's palsy (8). An animal model for Bell's palsy has been developed in which animals inoculated with HSV-1 demonstrate transient facial paresis (9), thus further supporting the idea that Bell's palsy is the result of a viral inflammatory response that induces edema within the facial nerve.

The natural history of Bell's palsy has been examined in an article by Peiterson (10) who evaluated the outcomes of 1011 patients with Bell's palsy who were not medically or surgically treated. It was found that this condition occurs in every decade of life with a mean age between 40-44 years. It is less common before the age of 15 and after the age of 60 years. The incidence in men and women is similar. Approximately 6-9% develop recurrent Bell's palsy. Facial paresis alone occurred in 31% of patients, while the remainder had unilateral complete paralysis. Of those experiencing only a paresis, over 95% recover without sequelae. Of the remaining 69% with complete paralysis, some return of facial function is evident within 3 weeks in 85% and an estimated 71% of patients achieve a House-Brackmann grade 1 and 13% a House-Brackmann grade 2. The remaining 16% in this complete paralysis group have a fair to poor recovery (House-Brackmann grades 3-5). This subset of patients is the controversial group of patients that may benefit the most from medical or surgical intervention.

Thus, of all patients with either a complete or partial facial paralysis, approximately 85% recover to normal within one year without treatment. It was noted that in patients that experienced delayed recovery over 3 months, all developed sequelae such as diminished function or contracture with associated movements. Return of at least some facial function was noted in all patients.

Many reports exist in the literature that attempt to document the outcomes in Bell's palsy. Unfortunately, there was a lack of an accepted facial function recovery reporting system until the American Academy of Head and Neck Surgery adopted the House-Brackmann facial nerve grading scale in 1984 (11).

## **Evaluation of Acute Facial Paralysis**

A careful history of the patient's illness narrows the scope of the differential diagnosis and reduces the number of subsequent radiographic and serologic studies that are necessary. Most palsies of the face are sudden in onset and frequently evolve over 2-3 weeks after onset. This evolution results in either a complete degeneration of nerve fibers or an incomplete degeneration with evidence of recovery as demonstrated by either clinical or electrophysiologic means. Any palsy progression over 3 weeks should be evaluated for a neoplasm. Evidence of trauma is usually obvious as is acute or chronic otitis media with or without cholesteatoma. Herpes zoster oticus (Ramsay-Hunt syndrome) is manifest by a facial paresis or paralysis with a vesicular eruption over a distribution of a cranial nerve. Sensorineural hearing loss and vertigo may also be present in up to 20% of cases. Recurrent facial palsy may be seen in cases of Bell's or Melkersson-Rosenthal syndrome. Bilateral facial palsy is most commonly seen in Guillain-Barre syndrome, as a manifestation of Lyme disease, or with intracranial tumors or infections (2).

The physical examination includes a complete head and neck examination with microscopic evaluation of the ear and a thorough cranial nerve examination. The face is examined and is assessed for paresis (an incomplete paralysis) or a complete paralysis.

Audiometry should be obtained in all cases to assess for possible involvement of the eighth cranial nerve in some disorders. Formulation of a treatment plan is dependent on the identification of the 15% of patients with Bell's palsy who do not fully recover. Both imaging studies and electrophysiologic studies of the facial nerve have been used in this aim. The site of the lesion is best determined with either CT or MRI and prognosis is best determined with electrophysiologic testing. High resolution CT (HRCT) may be obtained in cases to examine the fallopian canal in temporal bone trauma, mastoiditis, or cholesteatomas. HRCT is unable to demonstrate subtle facial nerve inflammation typically seen in Bell's palsy. Unfortunately, MRI assessment of the facial nerve has been disappointing as well. MRI may demonstrate enhancement of the facial nerve in cases of Bell's palsy or Ramsay-Hunt syndrome, however no correlation with the site or degree of enhancement has been made. The primary role of MRI in cases of suspected Bell's palsy is to exclude the possibility of other mass lesions that can lead to facial nerve paralysis (12). Topographic testing including the Schirmer test, stapedial reflex assessment, electrogustometry, and salivary flow has become obsolete due to the inability to predict clinical outcome (2). If the diagnosis remains uncertain, serologic studies can be considered to evaluate for Lyme disease, autoimmune disorders, or other central nervous system diseases.

## **Anatomy**

Knowledge of the anatomy of the facial nerve is important in understanding the pathophysiology and management of acute facial paralysis of Bell's type. The intracranial segment of the facial nerve and the nervus intermedius exit the brainstem adjacent to the pons, cross the cerebellopontine angle, and enter the internal auditory canal. The meatal segment of the facial nerve and the nervus intermedius remain in the anterior-superior quadrant of the IAC and enter the fallopian canal at the meatal foramen, superior to the transverse crest and anterior to the vertical crest (Bill's bar). Ge and Spector (14) have determined that the narrowest portion of the fallopian canal is located at the meatal foramen (mean diameter .68 mm). Thus, the size of

the foramen coupled with a tight arachnoid band located at this segment contributes to the constriction of the facial nerve at this point in Bell's palsy. The labyrinthine segment of the nerve, encased within the narrowest portion of the fallopian canal, courses 2-4 mm to the geniculate ganglion, where the nerve takes an acute turn at the external genu to enter the middle ear. The tympanic segment (horizontal segment) courses a total of 11 mm slightly above the cochleariform process and oval window and turns into the second turn (pyramidal turn) inferior to the lateral semicircular canal. The mastoid segment (vertical portion) then descends 13 mm to the stylomastoid foramen (2).

## **Electrophysiology**

Bell's palsy induces a wide range of facial movement dysfunction from mild paresis to total paralysis. The pathophysiology associated with this injury is suspected to be due to edema within the facial nerve induced by HSV reactivation. Several electrodiagnostic studies have been devised to evaluate the neural damage following acute facial nerve paralysis and can be used for prognostic purposes. These tests attempt to measure the amount of neural degeneration that has occurred distal to the site of the injury by measuring the muscle response to an electrically evoked stimulus. These tests rely on the physiological premise of neural injuries as described by Seddon (13). Injuries that produce only a conduction block within the nerve and do not disrupt axonal continuity are termed neuropraxia. In these cases, the nerve does not sustain permanent damage and no Wallerian degeneration occurs. All electrophysiologic studies (NET, MST, and ENoG) will be within normal limits. EMG will fail to demonstrate voluntary motor action potentials, as these cannot be conducted past the blockade. With more severe injuries, axoplasmic disruption (axonotmesis) or neural tubule disruption (neurotmesis) occurs. Axonotmesis describes a state of Wallerian degeneration distal to the lesion characterized by the preservation of endoneurial sheaths of the motor axons. Electrically, the NET, MST, and ENoG will indicate rapid and complete degeneration (if pure axonotmesis). The EMG will not demonstrate any voluntary motor units, and after 10-14 days, myogenic fibrillation potentials become evident. The axons will regenerate through the intact neural tubules, allowing complete return of motor function to the muscle fiber innervated by that nerve fiber. Neurotmesis describes the destruction of the axon and surrounding support cells. The lesion leads to Wallerian degeneration distal to the site of injury with the electrophysiologic tests being similar to that of axonotmesis. The outcome in this injury, however, is less predictable. The nerve fibers may be unable to regenerate successfully which might result in a misdirection of fibers resulting in synkinesis and incomplete facial function. Electrodiagnostic tests can be used to differentiate nerve fibers that have undergone Wallerian degeneration, but these tests are unable to differentiate axonotmesis from neurotmesis.

The currently popular tests used to establish prognosis in Bell's palsy include the NET (nerve excitability test), MST (maximum stimulation test), ENoG (electroneurography), and EMG (electromyography). The NET, MST, and ENoG are most applicable in the evaluation of acute paralysis (during the degeneration phase). Hilger first described the NET in 1964. It compares the current thresholds required to elicit minimal muscle contraction on the normal side of the face to those of the paralyzed side. During degeneration, the NET will show increasing side-to-side threshold differences. A difference of 3.5 mA is considered significant and suggests

more severe degeneration and a higher likelihood of a poor outcome. The maximum stimulation test is similar to the NET except that it uses maximum rather than minimum stimulation and responses are judged as a difference in facial movement. The MST will show greater degrees of facial weakness with worsening degeneration of the nerve. Unlike the NET and MST, the ENoG provides a quantitative analysis of the extent of degeneration without being dependent on observer qualification. It is the most accurate of all the diagnostic tests available and has proven predictive power. For this test, there is a recording of the summation potential (compound action potential) for the involved and uninvolved sides. The sides are compared with one another and the degree of degeneration within the nerve is directly proportional to the amplitudes of the measured summation potentials. Thus, with ongoing Wallerian degeneration, the ENoG will demonstrate lower percentages of intact motor axons able to propagate a summation potential (2).

Nerve degeneration in Bell's palsy typically occurs over a three-week period and the NET, MST, and ENoG will provide the most accurate information. Within the first three days after the onset of complete paralysis, the results of NET, MST, and ENoG will be inaccurate, as Wallerian degeneration has not yet occurred. The results during this time will be near normal. Because of this limitation, the prognosis cannot be established reliably until the fourth or fifth day when Wallerian degeneration of damaged nerve fibers is likely ongoing. Comparing ENoG results during the course of the degeneration is important for prognosis. Esslen (14) found that if there is greater than a 90% degeneration of the amplitude of the ENoG waveform on the affected side, the prognosis worsens. This study established that those patients with 90-97% degeneration, 30% recovered fully; of those patients with 98-99% degeneration, 14% recovered fully; and those with 100% degeneration, none recovered fully.

To further quantify patient outcomes using ENoG, Fisch (15) found that all patients having less than 90% maximal degeneration of facial nerve fibers within three weeks of onset of the palsy reach a satisfactory return of facial motion without any form of treatment (comparable to a House-Brackmann stage 1-2). On the other hand, 50% of the patients with a 95-100% maximal degeneration within two weeks of onset of the palsy have a permanent unsatisfactory recovery of function (House-Brackmann grades 3,4, or 5). He also found that if the nerve did reach 90% degeneration, there was a high likelihood of degenerating further to the critical 95% degeneration point. Thus, in these patients, the likelihood of normal to near normal recovery remains 50%.

EMG testing in the acute phase is primarily a complementary test as it is unable to distinguish a totally neuropraxic injury from a completely degenerated nerve from a regenerating nerve in the acute phase. For example, it is not uncommon for an ENoG test to record no response with early recovery in Bell's palsy. This is because regenerating nerve fibers fire at different rates, which does not allow a complete summation potential to be recorded on ENoG. In these cases, EMG is essential to assess if motor units are observed with voluntary contraction. EMG is also essential in the long-term evaluation of facial nerve paralysis. The presence of myogenic fibrillation potentials and the absence of voluntary motor units denotes complete degeneration while the coexistence of both defibrillation potentials and motor units indicates an incomplete lesion and the appearance polyphasic motor units signifies a regenerating nerve.

## Medical Management of Bell's Palsy

It is difficult to prove that medical management influences the course of recovery in patients with Bell's palsy. The most widely evaluated intervention is steroid therapy. In 1987, Stankiewicz (16) reviewed the literature on steroid use in Bell's palsy and found no irrefutable study showing the efficacy of steroid therapy. In 1993, however, Austin (17) conducted a randomized, double blind, placebo controlled study that revealed an improvement in grade of recovery with the use of prednisone. All patients treated with prednisone were in the good outcome group of grades 1-2 on the House-Brackmann scale. However, 17% of patients not treated with prednisone fell into grade 3 recovery. These results were statistically significant. A trend towards the prevention of denervation was noted but did not reach clinical significance. The authors felt that prednisone treatment was most likely to be beneficial in patients with more severe paralysis at initial presentation.

Given the likely association of Bell's palsy with HSV reactivation, Adour (18) conducted a double blind study comparing prednisone and placebo and prednisone and acyclovir. The drugs were initiated within three days of onset of the paralysis. Only 20% progressed to complete paralysis clinically within 14 days of onset and these were evenly distributed between the groups. The acyclovir treated patients demonstrated less degrees of facial weakness (and presumably less degeneration) on MST testing and a lower incidence of unsatisfactory recovery (House-Brackmann grade 3-5).

Given these results, most authors recommend a medical regime consisting of both steroids and antivirals, with most recommending initiation within three days of onset. Protection from corneal irritation is critical with hourly saline eye drops and nightly ophthalmic ointment in the eyes. Regular, scheduled assessment of facial function is important so that appropriate prognostic electrophysiologic tests can be administered in the event of complete paralysis.

## Surgical Management of Bell's Palsy

There is spirited debate among clinicians concerning the appropriate surgical management of the Bell's palsy patient since it was first reported in 1932 by Balance and Duel (4). Fowler (4) commented in 1939 that there was great controversy regarding the indications for such a procedure and that the procedure be done only by experts. Facial nerve surgery was greatly influenced in the 1960's with the advances in electrodiagnosis. During this decade, the site of the lesion was felt to be the tympanic segment. Thus, most cases of decompression were focused at that site. In the 1970's, McNeill (4) reviewed his patients treated with postauricular decompression from the geniculate to the stylomastoid foramen and found no appreciable benefit. All of these patients were operated on after two weeks. In 1972, Fisch and Esslen (19) published revolutionary data in the surgical management of Bell's palsy. They reported 12 patients who had undergone total facial nerve decompression via a middle cranial fossa and transmastoid approach. Using intraoperative electrical stimulation in three patients, they found a conduction block in the region proximal to the geniculate ganglion (in the area of the meatal foramen and labyrinthine segment), consistent with their gross anatomic observations. Fisch (15) later went on to conclude that decompression of the facial nerve in patients with 90% reduction of the compound action potential should be done within two weeks for maximum

benefit. He found that there was a statistically significant benefit to decompressing the meatal foramen in this selected group of patients. May (20) initially reported that transmastoid decompression to the labyrinthine segment was beneficial in selected patients (decreased salivary flow, decreased Shirmer test, and MST reduced to 25% of normal). In a subsequent publication (21), he recanted these ideas by finding that no patients benefited from transmastoid decompression performed within 14 days of onset. Thus, two schools of thought developed in the 1980's and 1990's; those who adopted the idea of early intervention debated the ideal approach while those who opposed surgical management were quite emphatic (4).

In order to help define which patients would benefit from surgical decompression and to corroborate the original findings of Fisch (15), Rubenstein and Gantz (1) undertook a retrospective study in 1999 to assess whether patients with severe degeneration (greater than 90% on ENoG) would benefit from decompression of the facial nerve. In this study, the meatal foramen, labyrinthine segment, geniculate ganglion, and tympanic section were decompressed through a middle cranial fossa approach. This route was chosen because of strong evidence that the conductive block occurs in and around the meatal foramen in 94% of patients (using intraoperative EMG) and that electrical impulse conduction improves after decompression of that area. If the conduction block was not identified during this approach using intraoperative EMG, a transmastoid decompression was added. They reported in a multi-institutional review that 92% of patients who exhibited greater than 90% degeneration on ENoG within 14 days recovered to a House-Brackmann grade 1 or 2. In patients who were treated only with steroids, only 45% recovered to a House-Brackmann grade 1-2.

They concluded that facial nerve decompression for Bell's palsy is useful in a selected group of patients—those with greater than 90% compound action potential reduction within 14 days on ENoG and lack of volitional activity on EMG.

## Conclusion

It is clear that the management of acute facial paralysis is evolving. Many unresolved issues remain. Hopefully, through diligent scientific inquiry, superior diagnostic and therapeutic methods will be discovered for the treatment of Bell's palsy.

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