REVIEW
FACIAL PLASTIC AND RECONSTRUCTIVE SURGERY
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Guidelines for the Initial Management of Acute Facial Nerve Palsy

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ABSTRACT

The aim of this study was to provide a concise review of international standards in the initial management of facial palsy. This culminates in guidelines and indications for referral to a tertiary facial nerve center. Facial nerve palsy is a relatively rare condition, and most practitioners outside specialized centers will only see a few cases per year. While most patients will ultimately show spontaneous and full recovery of facial nerve function, the importance of accurate diagnosis and early treatment cannot be underestimated. Incorrect diagnosis can be harmful to patients, resulting in worse facial function outcomes as a result of delay in diagnosis of occult neoplasms and failure to treat patients within a timely fashion. Management of facial palsy is complex and requires a multidisciplinary board with specific focus.

Keywords: Bell's palsy, facial movement disorder, facial paresis, facial weakness, guideline

Introduction

Facial palsy (FP) includes the entire spectrum of facial movement disorders, including flaccid facial palsy (FFP), facial paresis, and postparalytic facial palsy. Facial palsy is a severe condition with a serious impact on both functional and cosmetic outcomes, resulting in a significant loss in quality of life (QOL).[1,2] Reversible Bell's type palsy is by far the most common etiology in acute unilateral FP, accounting for 60-80% of cases.[3] Timely diagnosis and treatment is key in recovery of facial nerve function. However, the diagnosis of Bell's palsy (BP) should be made with caution: literature suggests misdiagnosis rates may be as high as 20%. Moreover, these misdiagnoses are populated by benign and malignant tumors that require intervention.[4-7]

This article serves as a concise review of international standards in the initial management of FP and culminates in guidelines and indications for referral to a tertiary facial nerve center. The late management options for FP will be discussed in a subsequent article.

History and Physical Examination

Performing a thorough history is of utmost importance in evaluating patients with acute FP, with particular attention paid to time course of onset, progression, recurrence, associated symptoms, oncological history, head trauma, subjective hearing loss, and recent travel. Differential diagnosis of acute FP is provided in Table 1.

The time course of onset of facial weakness is critical. Facial weakness usually develops over a period of a few hours to 2 days, although some etiologies cause paralysis over weeks to months. Bell's palsy typically presents with a prodrome but fully evolves over 1- 3 days. Start of recovery in BP is expected to occur within 6 months after onset. If the onset of facial weakness exceeds over 72 hours, or there are no signs of recovery after 6 months, the diagnosis of BP is highly unlikely and alternative diagnoses should be considered. A history of infection or exposure to ticks may suggest infectious etiology while otovestibular symptoms such as hearing loss or vertigo indicate otitis or temporal bone trauma. Inflammatory conditions such

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Table 1. Causes of facial palsy

Infectious	Bell's palsy, Ramsay Hunt syndrome, acute otitis media, malignant otitis externa, cholesteatoma, skull base osteomyelitis, Lyme, and HIV
Developmental	Mobius, hemifacial macrosomia, pontine malformation, or anomaly
Benign tumor	Facial nerve schwannoma
Malignancy	Parotid carcinoma, head & neck carcinoma, and leptomeningeal carcinomatosis
Trauma	Temporal bone fracture, penetrating trauma, and birth trauma
Iatrogenic	Parotid, soft tissue, otologic, or orthognathic surgery
Systemic/autoimmune	Sarcoidosis, Melkersson–Rosenthal syndrome, Guillain–Barre, multiple sclerosis, amyloidosis, granulomatosis with polyangiitis, Sjögren, systemic lupus erythematosus, Behçet
Metabolic	Hypothyroidism, pregnancy

as uveitis or parotitis and known autoimmune conditions may be important, especially in bilateral FP.

A complete head and neck examination is essential in the workup of FP. Palpation of the neck and parotid area to rule out neck or parotid masses, assessment of all cranial nerves to evaluate related nerve deficit, and skin examination may show vesicles (Zoster), a rash (systemic, autoimmune), malignant skin lesions or scars from prior surgery for skin cancer. Microotoscopy should be performed to rule out any vesicles in the ear canal and audiometry or tuning fork test can rule out other otologic causes.

Then the facial nerve function will be observed in rest[1] and motion and compared with the contralateral side. This examination will start superior to inferior: first, the patient is asked to raise the eyebrows to assess the action of the frontalis muscle.[2] When the upper third of the face is spared, a central cause should be excluded. Next, the examiner asks the patient to close the eyes softly and relax,[3] then close the eyes as hard as possible.[4] The inability to close the eye with minor effort is known as lagophthalmos, though often the eye can be closed with effort. By holding the eyelids open when the patient is asked to close them, the clinician can often notice the eye rolling upward. This is known as Bell's phenomenon. Severe lagophthalmos and the absence of Bell's phenomenon should be noted as they confer a higher risk of corneal drying exposure keratopathy, particularly when the patient is asleep. The patient is asked to smile without[5] and with showing teeth[6] to assess zygomaticus major muscle function. Finally, the patient is asked to pucker and press the lips together to evaluate orbicularis oris muscle function[7] and then to evert the lower lip to examine the lower lip depressors.[8] During these facial movements, it is important to check for synkinesis movements, for example, closing of the eye with lip pucker. The phenomenon of synkinesis is caused by aberrant reinnervation among facial nerve fibers in the late recovery of FP. Documentation of these facial movements using video- or photography at presentation and relevant follow-up visits is useful in evaluating recovery or interventions (Figure 1).

The House–Brackmann (HB) scale is one of the most commonly used tools for the clinical evaluation of facial nerve function. It is useful in the initial assessment and decision-making of FP. However, it is not sensitive to detect small changes and there is no specific evaluation of synkinesis. Therefore, HB scale is inadequate in evaluating rehabilitation or interventions. We prefer to use the eFACE, a clinician-graded facial function scale based on videography of dynamic movements.[8] It is a validated clinician objective assessment of facial paralysis, and in contrast to the widely used HB scale, it is sensitive for synkinesis and flaccid facial paralysis and facial reanimation interventions. We therefore recommend its use in staging, during follow-up, and after facial reanimation procedures.

Main Points

- Bell's palsy is a tentative diagnosis and should be made with caution.
- Facial nerve palsy requires close follow-up to confirm a time course consistent with BP.
- In traumatic or iatrogenic FP, urgent referral is important as treatment delay is a negative predictive factor.

Management of Facial Palsy

Guidance on the management of FP is summarized in the flowchart (Figure 2). An essential item in the initial assessment of FP is differentiation between an incomplete and complete paralysis of peripheral or central origin. Urgent referrals are critical not only to rule out stroke in the case of central paralysis but also for the potential surgical management of complete peripheral FP.

Trauma

The first and most urgent variable to be determined is whether the facial weakness resulted from trauma.

- In the case of facial weakness after penetrating trauma (with complete and immediate paralysis), surgical nerve repair by coaptation should be performed as soon as possible. Distal nerve axons undergo Wallerian degeneration (WD) over 3 days, so it is important to explore the wound in an expedient fashion. If the exploration is performed within 3 days, the distal nerve stumps can still be stimulated. This allows efficient localization of severed distal nerve stumps facilitating nerve co-adaptation.
- Iatrogenic FP can often occur after parotidectomy for benign lesions. It is important to bear in mind that anesthetic infiltration of the external auditory canal or retroauricular region can cause a temporary paralysis of the

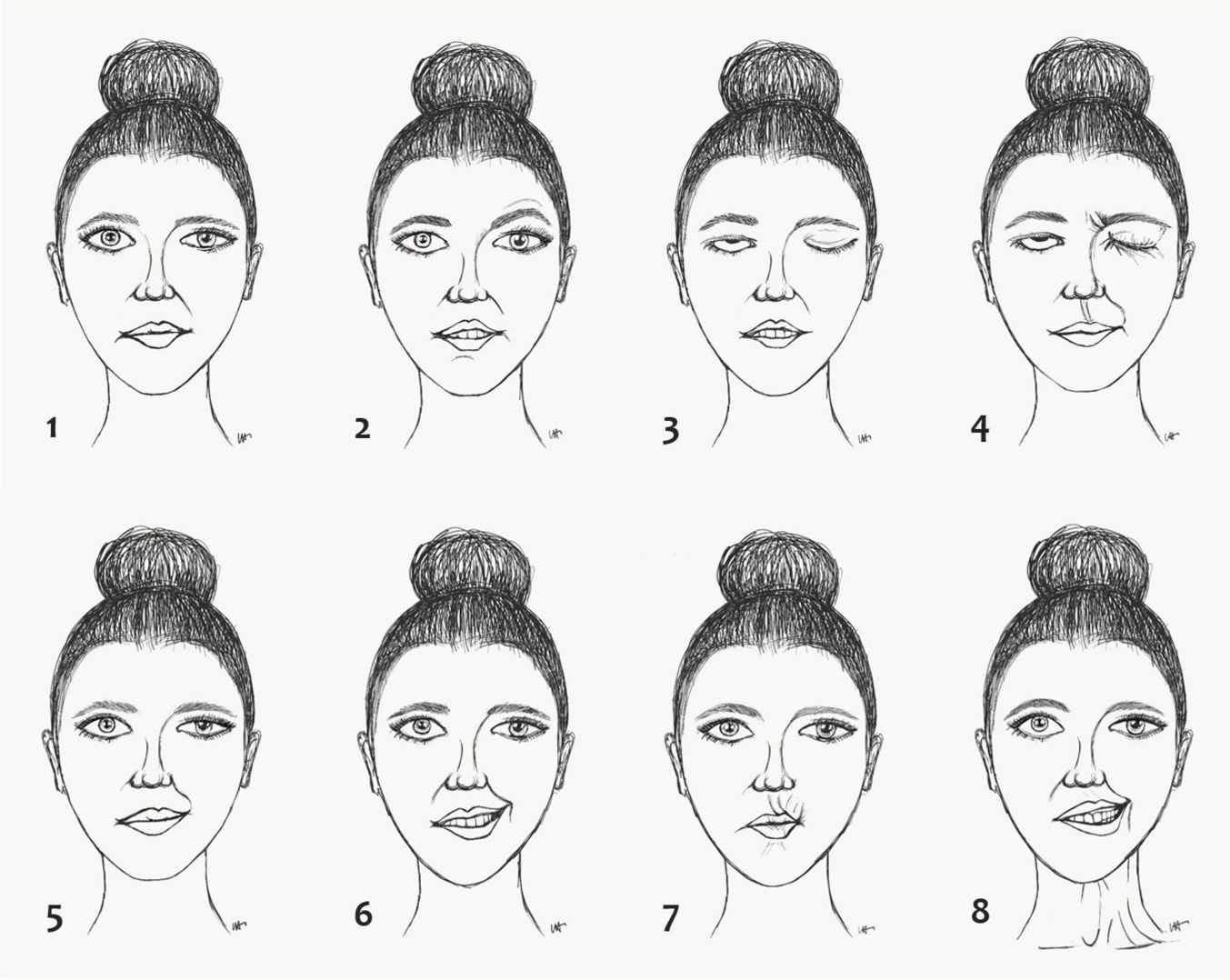


Figure 1. Facial movement examination.

extratemporal portion of the facial nerve. Lidocaine can last for 4-5 hours, while marcaine can last over 12 hours. If the paralysis persists longer, it is important to discuss the case with the operating surgeon to understand if the facial nerve was put at significant risk of inadvertent division—revision parotid—main trunk not found—lumpectomy. If so, early exploration can be considered. However, the vast majority of cases are related to traction of the nerve. If the surgeon is sure of identification and preservation of the main trunk and branches through nerve stimulation, it is reasonable to administer oral steroids and await spontaneous recovery.

- In case of blunt head trauma, a computed tomography (CT) scan of the ipsilateral mastoid should be executed. If CT scan shows a fracture through the facial nerve, we would recommend urgent referral for consideration of decompression.
- If CT scan is normal, our policy depends on the HB grade of facial weakness. With an FP HB grade of maximum 4, we would recommend a conservative treatment and watchful waiting. In case of flaccid FP—HB grade 5-6—after blunt head trauma, we recommend urgent referral to a tertiary facial nerve center for consideration of decompression of the compromised segment. In this case, we recommend a gadolinium-enhanced MRI of the course of the facial nerve for localization of the affected segment to further guide surgical decompression if needed.

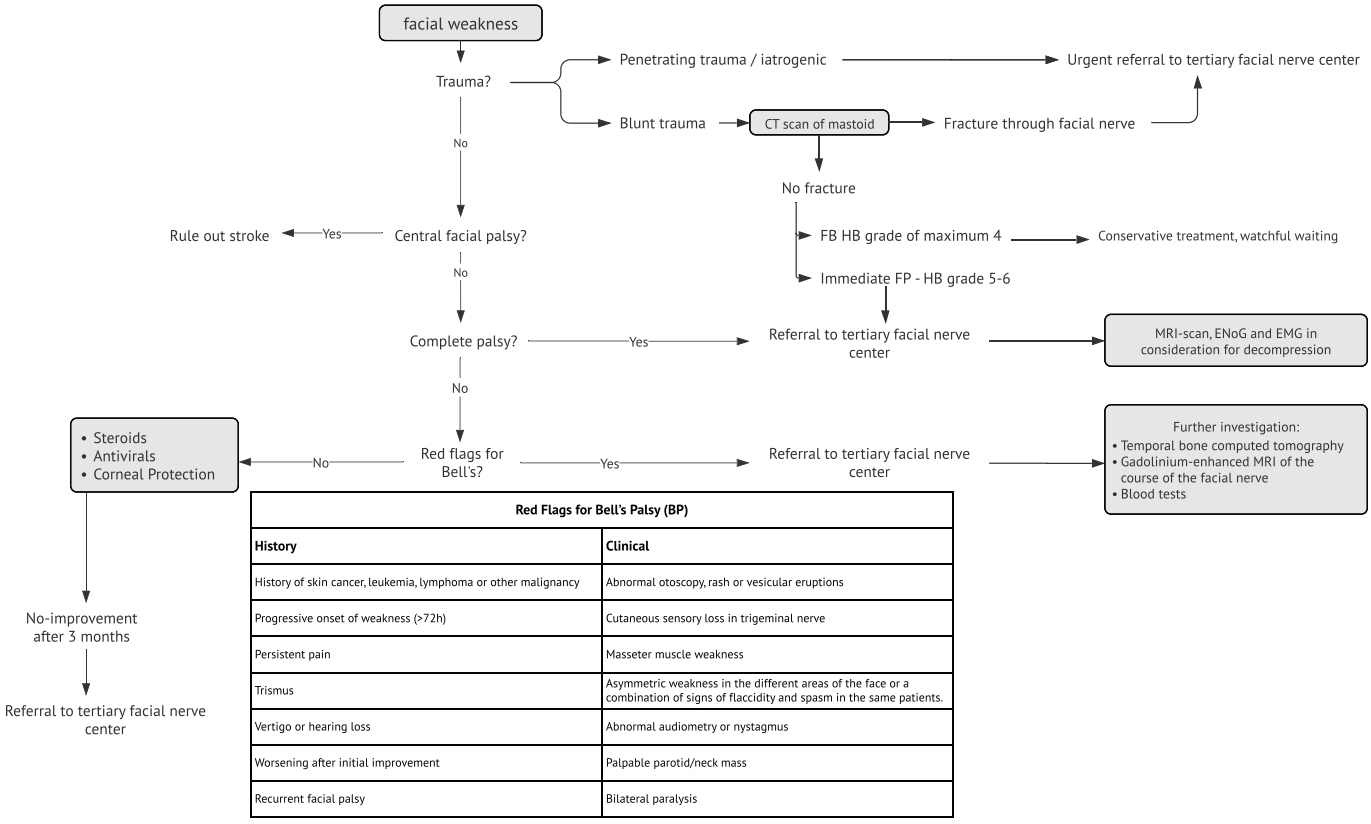
Central Facial Palsy

It is of vital importance to rule out a central FP that is characterized by palsy of the lower half of the face and non-involvement of the forehead. If this is the case, the patient should be referred urgently to rule out stroke.

Bell's Palsy

All initial diagnoses of Bell's are tentative. This point cannot be stressed strongly enough.

All patients with symptoms and signs consistent with BP must be closely followed to confirm signs of recovery. In BP, some recovery should be seen within 4 months and certainly within 6 months. The patient's recovery of facial function should then proceed either to full recovery or to a hypertonic/synkinetic postparalysis FP state by 12 months, which is roughly the expected time for facial nerve function to recover. Persistent signs of facial flaccidity after 12 months



Red Flags for Bell's Palsy (BP)	
History	**Clinical**
History of skin cancer, leukemia, lymphoma or other malignancy	Abnormal otoscopy, rash or vesicular eruptions
Progressive onset of weakness (>72h)	Cutaneous sensory loss in trigeminal nerve
Persistent pain	Masseter muscle weakness
Trismus	Asymmetric weakness in the different areas of the face or a combination of signs of flaccidity and spasm in the same patients.
Vertigo or hearing loss	Abnormal audiometry or nystagmus
Worsening after initial improvement	Palpable parotid/neck mass
Recurrent facial palsy	Bilateral paralysis

Figure 2. Flowchart giving guidance on the management of acute facial palsy.
FP: facial palsy, HB: House Brackmann, CT: computed tomography, MRI: Magnetic resonance imaging, ENoG: ectroneuronography and EMG: electromyography.

also effectively rule out the diagnosis of Bell's palsy. Making a definitive clinical diagnosis of BP at the first consultation therefore is dangerous and should be strongly discouraged for various reasons.

Once a diagnosis of BP is made, the patient is often then placed in a watch-and-wait holding pattern/scenario with no further active investigations being performed. Other specialist clinicians and GPs assume a benign diagnosis that will improve over some period of unspecified time. It is not infrequent in a facial nerve center setting to find patients being referred with "Bell's palsy or atypical Bells" following 3-4 years of monitoring with non-resolving flaccid facial paralysis. This is a disaster for the patient as the optimal therapeutic window has often passed.

It is recommended to use the term *Bell's-type picture* until the facial function can be measured at the above critical time points, to confirm a time course and a facial function recovery pattern consistent with Bells. Failure of recovery at 6 months should lead to a reappraisal of the diagnosis. The disease course is either that of a Bell's type picture or it is not. If it is not, then diagnosis remains unknown and requires active investigation. Recommended terminology is listed below:

1. Acute flaccid facial paralysis 1-6 months (Bell's-type picture or not—if other associated signs or symptoms that are inconsistent with Bell's palsy);
2. Persistent flaccid facial paralysis 6 months to 2 years (by definition cannot be of a Bell's-type picture)—cause unknown;
3. Chronic flaccid facial paralysis >2 years (by definition cannot be of a Bell's-type picture)—cause unknown.

It is not unsurprising that the literature suggests misdiagnosis rates as high as 20%. What is of more concern is that many of these misdiagnoses are populated by benign and malignant tumors.[4-7]

In cases where the history and physical examination are consistent with Bell's-type picture, no further investigations are mandatory. When history and physical examination are inconsistent with a Bell's-type picture (i.e., red flags for BP), imaging studies—preferably a gadolinium-enhanced MRI of the course of the facial nerve including the entire parotid gland and computed tomography of temporal bone and neck—are performed to rule out neoplastic or infectious causes like osteomyelitis. Blood tests are indicated in recurrent cases with suspicion for autoimmune or infectious etiologies like HIV. Serology tests are justified in endemic areas for Lyme. Consequently, underlying pathologies causing the FP should be managed accordingly.

Management of Incomplete Bell's Palsy

Peitersen[9] described spontaneous full recovery of BP in about 70% of all patients. Full recovery was significantly associated with less severity of weakness, early first signs of recovery, and young age. This study also stated that facial function recovered fully in 85% of patients within 3 weeks and in the remaining 15% after 3-5 months.

For medical treatment, the single most important factor affecting the outcome is delay in treatment, so early diagnosis and treatment are key. The following guidelines for medical management are specific for BP and were developed in accordance with the methods proposed by the GRADE Working Group.[10]

● Corticosteroids

Literature review comparing oral steroids versus placebo shows strong evidence for the use of oral steroids in early FP.[11] Corticosteroids reduce the risk of unsatisfactory facial recovery and this risk reduction is more powerful in patients with severe FP. Oral steroids should be started in the first 72 hours after the onset of symptoms. After 14 days of onset of symptoms, no treatment will have any impact on recovery. Most studies used prednisolone 60 mg per day for the first 5 days followed by a 5-day taper, usually this short-term dosing is well tolerated. Intravenous delivery of steroids was not shown to be superior to oral.

● Antivirals

There is strong evidence that monotherapy with antivirals has no role in BP.[12] However, a Cochrane review showed that combining steroids with antivirals was beneficial compared to monotherapy with steroids.[13] Antivirals are contraindicated in pregnancy and liver or kidney failure, and valacyclovir or famciclovir are preferred over acyclovir because of higher oral bioavailability. There is no consensus on dosage, but we recommend valacyclovir 3 g/day over 5 days or famciclovir 1 g/day over 5 days. The benefit of antivirals after 72 hours after onset of symptoms is arguable except in immunocompromised cases where it is recommended.

● Corneal protection

Closure of the eye is compromised, resulting in lagophthalmos, because normal retraction of the levator palpebrae superior muscle, innervated by the oculomotor nerve, is unopposed due to the paralysis of the orbicularis oculi muscle. This may result in corneal epithelial lesions, exposure keratopathy, ulceration, and in extreme cases, corneal perforation. In addition, proximal facial nerve lesions or BP can impair the parasympathetic innervation of the lacrimal gland compounding the risk of corneal dryness and lesions. Patients are encouraged to use protective glasses and regular use of natural tears during the day and a thicker ointment at night. Night-time requires particular attention as there is no active tear production at night, and the cornea can easily dry and ulcerate, particularly in the absence of the Bell's phenomenon. Careful taping of the eyelids may help in protecting against exposure keratopathy. Again, clinical examination for associated cranial nerve dysfunction is essential, and exposure keratitis in patients with combined facial and trigeminal dysfunction can be painless; therefore, we recommend urgent referral of these patients to a tertiary referral facial nerve center as these patients are particularly vulnerable to corneal blindness.

● Physiotherapy/electrical stimulation/acupuncture

There is no evidence for adding physiotherapy or electrical stimulation in the early management of FP.[14] It does not provide any benefit, to the contrary, it may facilitate aberrant reinnervation and excess motor unit recruitment, favoring abnormal movement patterns, synkinesis, mass movements, and hypertonic areas. Systematic reviews by Pereira et al[15] and La Touche et al[16] suggest that physical therapy might prevent and decrease synkinesis in longstanding facial paralysis. However, a Cochrane analysis by Teixeira et al[17] was unable to find a significant benefit or harm from any physical therapy.

Complete Facial Palsy

In case of sudden and complete FP or sudden flaccid facial paralysis, we recommend referral to a tertiary facial nerve center for further investigation to consider urgent surgical decompression. In the absence of any red flags or underlying pathology, electrodiagnostic testing is indicated to stratify those patients for nonsurgical versus surgical management.

Electrodiagnostic Testing

Electroneuronography (ENoG) and electromyography (EMG) are the 2 most reliable electro-physiological tests currently. These tests provide prognostic information for likelihood of recovery in patients with complete paralysis and help identify those who might benefit from surgical decompression in the acute setting. In patients with incomplete paralysis or early recovery, electrodiagnostic testing is not indicated.

Electroneuronography measures muscle action potentials evoked by supramaximal stimulation of the facial nerve by placing a stimulating electrode at the stylomastoid foramen and a recording electrode at the nasolabial groove.[18,19] The amplitude of the reaction is compared between the affected and normal sides. The result is expressed as a percentage, estimating the relative proportion of nerve fibers that have undergone WD. Electroneuronography is not performed before the fourth day of paralysis because WD does not occur in the first 3 days after the pathological event. Patients with degeneration of more than 90% on ENoG in the first 2 weeks after onset of FFP without recovery showed better outcome after surgical decompression.[20] After 2 weeks of paralysis, however, ENoG is not indicated because patients who fail to reach degeneration threshold of more than >90% in the first 2 weeks have a good prognosis for recovery.

Facial nerve EMG evaluates motor activity by measuring electrical action potentials generated by spontaneous and voluntary muscle contraction. This is measured by needle electrodes placed in orbicularis oculi and oris muscles and asking the patient to make forceful contractions. Positive waves and fibrillation potentials are signs of denervation, while polyphasic motor unit potentials indicate active reinnervation. Electromyography is mainly helpful in more longstanding FP, the appearance of polyphasic potentials on EMG indicates nerve regeneration and often precedes functional recovery. In acute FP setting, EMG is mainly helpful in patients with complete palsy where ENoG demonstrates >90% degeneration of the facial nerve. If a subsequent EMG investigation shows voluntary action potentials, the prognosis of facial nerve recovery is nevertheless excellent, and there is no indication for surgical decompression. This is caused by the asynchronous discharge of regenerating nerve fibers that fail to produce measurable potential on ENoG and is called "early de-blocking phenomenon."[21]

Surgical Decompression

The following criteria for surgical decompression were validated by Gantz et al[22]:

- Idiopathic and posttraumatic complete FP;
- ENoG: > 90% degeneration;

- o EMG: no voluntary motor action potentials;
- o Decompression must occur within 14 days of onset of complete FP.

Intra-operative electrical testing suggests that surgical decompression in BP should involve the entire labyrinthine segment and the bony canal proximal to the geniculate ganglion.[23] Recovery of facial nerve function should not be expected in the first weeks after surgery.[24] A systematic review by Casazza et al reported significantly better facial nerve outcomes for middle cranial fossa decompression (MFD) performed less than 14 days versus more than 14 days after onset of symptoms. Regarding transmastoid decompression (TMD), there was no significant better outcome regarding facial nerve function when compared to medical controls.[25] To be concise, there is evidence for performing MFD in Bell's palsy patients within 14 days of symptom onset, if ENoG shows degeneration greater than 90% and EMG shows no voluntary potentials.[26] There is no robust evidence to support TMD at any stage.

Conclusion

In conclusion, facial nerve palsy is a relatively rare condition, and most practitioners outside specialized centers will only see a few cases per year. While most patients will ultimately show spontaneous and full recovery of facial nerve function, the importance of accurate diagnosis and early treatment cannot be underestimated. Incorrect diagnosis can be harmful to patients, resulting in worse facial function outcomes as a result of delay in diagnosis of occult neoplasms and failure to treat patients within a timely fashion. Management of FP is complex and requires a multidisciplinary board with specific focus (facial plastic reconstructive surgery, opthalmology, neurotology, neurology, neurosurgery, and speech language therapy).

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