

Facial Palsy, a Disorder Belonging to Influential Neurological Dynasty: Review of Literature

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Abstract

Facial paralysis is one of the common problem leading to facial deformation. Bell's palsy (BP) is defined as a lower motor neuron palsy of acute onset and idiopathic origin. BP is regarded as a benign common neurological disorder of unknown cause. It has an acute onset and is almost always a mononeuritis. The facial nerve is a mixed cranial nerve with a predominant motor component, which supplies all muscles concerned with unilateral facial expression. Knowledge of its course is vital for anatomic localization and clinical correlation. BP accounts for approximately 72% of facial palsies. Almost a century later, the management and etiology of BP is still a subject of controversy. Here, we present a review of literature on this neurologically significant entity.

Keywords: Bell's palsy, corticosteroids, facial palsy, neurological disorder

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Introduction

Facial paralysis is a disfiguring disorder that has a considerable impact on the patient because it results in the loss of facial expression; it is most commonly caused by a benign self-limiting inflammatory condition known as Bell's palsy (BP).^[1] BP is one of the most common neurologic disorders affecting the cranial nerves, and is the most common cause of facial paralysis worldwide. It is thought to account for approximately 60–75% cases of acute unilateral facial paralysis. It is an idiopathic, acute peripheral-nerve palsy involving the facial nerve, which supplies all the muscles of facial expression. The facial nerve also contains parasympathetic fibers to the lacrimal and salivary glands, as well as limited sensory fibers supplying taste to the anterior two-thirds of the tongue.^[2]

Historical Aspect

It was first reported by Nicolas A Friedrich two century ago in 1798. BP is named after Sir Charles Bell (1774–1842), who was a Scottish anatomist and Edinburgh graduate. He first described the syndrome along with the anatomy and function of the facial nerve in a lecture "On the nerves: Giving an account of some experiments on their structure and functions, which lead to a new arrangement of the system" to the Royal Society of London in 1821.^[3]

Epidemiology

The annual incidence of BP is 15 to 30 per 100,000 persons, with equal numbers of men and women affected. There

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is no predilection for either side of the face. BP has been described in patients of all ages, with peak incidence noted in the 40s. It occurs more commonly in patients with diabetes and in pregnant women. Patients who have had one episode of BP have an 8% risk of recurrence.^[4,5]

Etiology and Pathogenesis

Controversy surrounds the etiology and treatment of BP.^[6] The causes of the paresis still remain unknown even if the viral etiology has been discussed by several authors. Herpes viruses seemed to be the most plausible infective agent, determining inflammation and swelling of the nerve with subsequent blockage of the neural activity.^[7,8] The natural history of BP is encouraging for the patients because a total recovery of facial function is expected in 70–85% of the patients, and a higher percentage of recovery is achieved if corticosteroid therapy is administered,^[9,10] and early physical rehabilitation is performed in severe grades of paresis.^[11] The causes include microcirculatory failure of the vasonervorum, viral infection, ischemic neuropathy, autoimmune reactions, surgical procedures such as local anesthesia, tooth extraction, infections, osteotomies, preprosthetic procedures, excision of tumors or cysts, surgery of temporomandibular joint, and surgical treatment of facial fractures and cleft lip/palate.^[12]

The literature also reports three mechanisms in which a dental procedure could damage a nervous structure, that are, direct trauma to nerve from a needle, intraneural hematoma formation or compression, and local anesthetic toxicity. Direct trauma seems unlikely because many patients report experiencing trauma to the nerve when they feel the electric shock sensation on injection of the needle. However, virtually, all these symptoms resolve completely with no residual nerve damage.^[13]

Clinical Features

Facial paralyzes are divided into two types, namely, supranuclear and infranuclear system. The neurones supplying the lower face receive upper motor neurones (UMN) from the contralateral motor cortex, whereas the neurones to the upper face receive bilateral UMN innervation. An UMN lesion, therefore, causes unilateral facial palsy with some sparing of the frontalis and orbicularis oculi muscles because of the bilateral cortical representation. Furthermore, although voluntary facial movements are impaired, the face may still move with emotional responses, for example on laughing. Paresis of the ipsilateral arm (monoparesis) or arm and leg (hemiparesis), or dysphasia may be associated because of more extensive cerebrocortical damage.

Lower motor neurone (LMN) facial palsy is characterized by unilateral paralysis of all muscles of facial expression for both voluntary and emotional responses. The forehead is unfurrowed and the patient is unable to close the eye on that side. Attempted closure causes the eye to roll upwards (Bell's sign). Tears tend to overflow on to the cheek (epiphora), the corner of the mouth droops, and the nasolabial fold is obliterated. Saliva may dribble from the commissure and may cause angular stomatitis. Food collects in the vestibule and plaque accumulates on the teeth on the affected side. Depending on the site of the lesion, other defects such as loss of taste or hyperacusis may be associated.^[14]

Differential Diagnosis

Many conditions can produce isolated facial nerve palsy identical to BP. Structural lesions in the ear or parotid gland (e.g., cholesteatoma, salivary tumors) can produce facial nerve compression and paralysis. Other causes of peripheral nerve palsies include Guillain-Barré syndrome, Lyme disease, otitis media, Ramsay Hunt syndrome (an outbreak of herpes zoster in the facial nerve distribution), sarcoidosis, and some influenza vaccines. Although these conditions can present as isolated facial nerve palsies, they usually have additional features that distinguish them from BP. Patients with Lyme disease often have a history of tick exposure, rash, or arthralgias. Facial nerve palsies from acute and chronic otitis media have a more gradual onset, with accompanying ear pain and fever. Patients with Ramsay Hunt syndrome have a pronounced prodrome of pain and often develop a vesicular eruption in the ear canal and pharynx, although cases without the vesicular eruption (i.e., zoster sine herpete) have been reported. Polyneuropathies (e.g., Guillain-Barré syndrome, sarcoidosis) more often affect both facial nerves. Tumors present with a more insidious onset of symptoms over weeks or months. Central nervous system lesions (e.g., multiple sclerosis, stroke, tumor) can also cause facial nerve palsy. However, some motor neurons to the forehead cross sides at the level of the brainstem, and hence, the fibers in the facial nerve going to the forehead come from both cerebral hemispheres. Supranuclear (central) lesions affecting the facial nerve do not paralyze the forehead on the affected side, resulting in a unilateral facial paralysis with forehead sparing. Often, there is at least some weakness of extremities on the affected side as well. Influenza vaccines in the past have been associated with peripheral neuropathies.^[4,15-17]

Diagnostic Evaluation

Determining whether facial nerve paralysis is peripheral or central is a key step in the diagnosis. In facial palsy,

facial weakness is demonstrated by asking the patient to:

- Close the eyes against resistance
- Raise the eyebrows
- Raise the lips to show the teeth
- Try to whistle.

Full neurological examination is needed, looking particularly for signs suggesting a central lesion, such as hemiparesis, tremor, loss of balance, involvement of the Vth, VIth, or VIIIth cranial nerves. The following investigations may be indicated [Table 1].

Management and Prognosis

Treatment of BP should be conservative and guided by the severity and probable prognosis in each particular case. Studies have shown the benefit of high dose corticosteroids for acute BP. Although antiviral treatment has been used in recent years, evidence is now available indicating that it may not be useful.^[18-22] Because of acyclovir's relatively poor bioavailability (15 to 30%), newer drugs in its class are being trialed. Better bioavailability, dosing regimens, and clinical effectiveness in treating shingles have been shown with valaciclovir (prodrug of aciclovir), famciclovir (prodrug of penciclovir), and sorivudine. A recent study showed a significantly better outcome in patients with BP treated with valacyclovir and prednisone as compared with patients who were given no medical treatment [Table 2]. If presentation is early, most clinicians give prednisolone for 5 days, the aim being to reduce neuronal edema. Most recent studies, therefore, advocate prednisolone as the predominant medical treatment, especially if begun within the first 72 hours; it is less effective after 7 days. It has been shown to improve the primary outcome, i.e., recovery of facial function, as rated on the House-Brackmann scale, and secondary outcomes such as the quality of life, appearance, and pain.^[10,22-24]

Additional measures include eye protection, physiotherapy, acupuncture, botulinum toxin, or possibly surgery. An eye patch is of value to protect the cornea. Topical ocular therapy is useful in most cases, with the exception of those in which the condition is severe or prolonged. In these cases, surgical management is best. Surgical techniques [Table 3] aimed at improving function and cosmetics include nerve repair, graft, or transposition. Attachment of the distal end of the affected facial nerve to another afferent cranial nerve trunk, for example, the contralateral facial nerve, or a hypoglossal jump graft was a part of nerve transposition. This technique must be undertaken within 2 years of paralysis. Several procedures are aimed at protecting the cornea from exposure and achieving facial symmetry. These procedures reduce the need for constant use of

Table 1: Investigations required to rule out the diagnosis of facial palsy

Imaging with MRI or CT of the internal auditory meatus, cerebellopontine angle, and mastoid may be needed to exclude an organic lesion such as a tumor-particularly in progressive facial palsy
Study of evoked potentials to assess the degree of nerve damage. Facial nerve stimulation or needle electromyography may be useful, along with electrogustometry, nerve excitability tests, electromyography, and electroneuronography
Blood pressure measurement (to exclude hypertension)
Blood tests that may include:
Fasting blood sugar levels (to exclude diabetes)
Tests for HSV or other virus infections such as HIV may need to be considered
Serum angiotensin converting enzyme levels as a screen for sarcoidosis
Serum antinuclear antibodies to exclude connective tissue disease
In some areas, Lyme disease (tick-borne infection with <i>Borrelia burgdorferii</i>) should be excluded by ELISA test
Schirmer's test for lacrimation, performed by gently placing a strip of filter paper on the lower conjunctival sac and comparing the wetting of the paper with that on the other side
Test for loss of hearing
Test for taste loss by applying sugar, salt, lemon juice, or vinegar on the tongue and asking the patient to identify each of them
Aural examination for discharge and other signs of middle ear disease
Lumbar puncture occasionally

Table 2: Medicinal treatment

Medication	Dose
Acyclovir	Adults: 400 mg 5 times daily for 7 days Children older than 2 years: 80 mg per kg daily divided every 6 hours for 5 days, with a maximal dose of 3200 mg daily
Prednisone or prednisolone	Adults: 60 mg daily for 5 days, then 40 mg daily for 5 days Children: 2 mg per kg daily for 7-10 days

lubrication drops or ointments, may improve cosmesis, and may be needed to preserve vision on the affected side. These include the occlusion of eyelids with tape or patch in case of corneal erosions;^[25] external eyelid weights to improve mechanical blink; injection of hyaluronic acid gel in the prelevator aponeurosis region and/or pretarsal region in patients who are poor surgical candidates and/or as a temporary measure;^[26] tarsorrhaphy with suture or cyanoacrylate glue as a temporary/permanent measure;^[27] taping to prevent lower lid ectropion, botulinum toxin injected at upper border of tarsus to produce temporary ptosis, and Botulinum toxin^[28] for synkinetic eyelid movements secondary to aberrant regeneration in which there are eye movements such as blinking associated with oral movements.^[29,30] Several physical therapies, including massage and

Table 3: Surgical procedure

	Indications	Procedures
Neural procedures		
Nerve decompression	Indicated for decompression of nerve	
Neurorrhaphy	Primary repair of nerve following complete severance	Epineural repair under microscopic magnification
Nerve graft	Facial nerve excision following malignant parotid gland tumor excision	
Facial nerve crossover grafting	Unique motor fibers which not only provides voluntary motor action but also emotional expression, therefore, reinnervation can produce normal mimetic function	The long nerve graft are put across the face, and donor nerve are selected from the normal facial nerve
Facial nerve cross over and free muscle transfer	If the paralyzed muscles are atrophied, then these nerve fibers are planted in newly transferred new muscle mass by free tissue transfer	Free microvascular muscle transfers are gracilis muscles, pectoralis minor muscles, or rarely, rectus abdominis muscle
Nerve transfer procedure	When facial nerve cross over is not possible, such as in old age, associated morbidity, or the patient does not want long procedure	Donor nerves are hypoglossal and accessory nerve
Muscle procedures		
Temporali transfer		Muscle is divided in different slips and reflected back across the zygomatic arch to various region. The anterior slip goes to eyelids, the middle one to upper lip elevator and most posterior fibers for angle of mouth and lower lip
Masseter muscle transfer		Muscle is exposed either through Risdon incision or intraoral incision. The anterior half of muscle is erased
Static procedures		
Static sling surgeries for commissure		Slings are prepared either by tensor fascia graft or cortex or any other synthetic grafts One end is fixed to temporal fascia and other end is divided into two or three division
Gold weight for upper eyelid	To improve the position of upper eyelid	The upper eyelid is made heavier by insertion of gold piece which by gravity becomes heavier, and elevator finds it difficult to elevate the eyelid
Tarsorrhaphy	In elderly patients when there is hypotonia of lower lid and drooping of lower eyelid	Lateral 4-5mm of lid's edges are made raw and stitched together; it reduces the size of palpebral fissure and helps to protect the eye ball

facial exercises, are recommended to patients, however, there are few controlled clinical trials reporting their effectiveness. Some recent evidence supports facial retraining (mime therapy) with biofeedback. Prognosis of BP is fair with complete recovery in approximately 80% of the cases, 15% experience some kind of permanent nerve damage and 5% remain with severe sequelae.^[31]

A multidisciplinary team approach (general practitioners, otolaryngologists, ophthalmologists, plastic surgeons, dental surgeons, physiotherapists, and psychologists) is essential when there is no prospect of further recovery of facial nerve function. Facial reanimation may be possible by a combination of static and dynamic surgical techniques and may result in functional as well as cosmetic improvements.^[32,33] The most common complication of surgery is postoperative hearing loss, which affects 3–15% of patients. Based on the significant potential for harms and the paucity of data supporting

benefit, the American Academy of Neurology does not currently recommend surgical decompression for BP.^[34]

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Conflicts of interest

There are no conflicts of interest.

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