LITERATURE REVIEW

Facial nerve paralysis

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ABSTRACT

The facial nerve, the seventh pair of cranial nerves, has an essential role in non-verbal communication through facial expression. Besides innervating the muscles involved in facial expression, the complex structure of the facial nerve contains sensory fibres involved in the perception of taste and parasympathetic fibres involved in the salivation and tearing processes. Damage to the facial nerve manifested by facial paralysis translates into a decrease or disappearance of mobility of normal facial expression.

Facial nerve palsy is one of the common causes of presenting to the Emergency Room. Most facial paralysis are idiopathic, followed by traumatic, infectious, tumor causes. A special place is occupied by the child's facial paralysis. Due to the multitude of factors that can determine or favour its appearance, it requires a multidisciplinary evaluation consisting of otorhinolaryngologist, neurologist, ophthalmologist, internist.

Early presentation to the doctor, accurate determination of the cause, correctly performed topographic diagnosis is the key to proper treatment and complete functional recovery.

KEYWORDS: facial nerve paralysis, Bell's palsy, tumors, Ramsay-Hunt syndrome, facial nerve.

INTRODUCTION

The facial nerve, the seventh pair of cranial nerves, has an essential role in non-verbal communication through facial expression. Besides innervating the muscles involved in facial expression, the complex structure of the facial nerve contains sensory fibres involved in the perception of taste and parasympathetic fibres involved in the salivation and tearing processes.

Damage to the facial nerve manifested by facial paralysis translates into a decrease or disappearance of mobility of normal facial expression.

Facial nerve palsy is one of the common causes of presenting to the Emergency Room. Facial nerve palsy can be congenital or acquired in general or regional pathological contexts, but especially traumatic[1,2]. Besides the cause, the key in establishing the treatment and prognosis of this pathology is to identify the topographic location of the nerve lesion. In this context, knowledge of the functional anatomy of the facial nerve (VII) becomes essential.

FUNCTIONAL ANATOMY OF THE FACIAL NERVE

From a functional point of view, the facial nerve provides 4 important functions: motor, sensitive, sensory, secretory. These functions are performed by different categories of afferent and efferent fibres, each of them having a certain nervous trajectory, a characteristic physiology and the possibility of being functionally explored individually or in context with the other three (Figure 1).

Anatomically, two types of efferent fibres are described – motor fibres and parasympathetic secretory fibres (Table 1). Both categories of fibres are quasi-constant in the facial nucleus[3]. Outside the facial nucleus, a series of motoneurons are distributed to the stapedius muscle and the tensor tympani muscle which are associated with the trigeminal motor nucleus.

Parasympathetic secretory efferent fibres are intended for the lacrimo-muconasal system (lacrimo-muconasal nucleus) and the superior salivatory nucleus. The latter serves the intermediate nerve (Wrisberg

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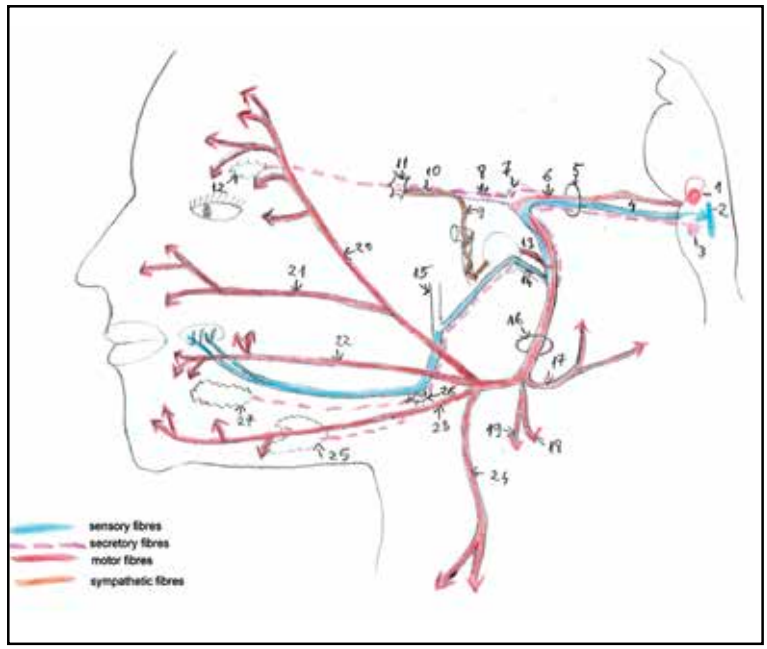

Figure 1. Schematic presentation of the facial nerve: 1. Facial nerve motor nucleus; 2. Solitary tract nucleus; 3. Superior salivatory nucleus; 4.The intermediate nerve; 5. Internal auditory canal; 6. Facial nerve; 7. Geniculate ganglion; 8. Greater petrosal nerve; 9. Deep petrosal nerve; 10. Vidian nerve; 11. Pterygopalatine ganglion; 12. Lacrimal gland; 13. Stapedius nerve; 14. Chorda tympani nerve; 15. Lingual nerve; 16. Stylomastoid foramen; 17. Posterior auricular nerve; 18. Digastric branch of the facial nerve; 19. Stylohyoid branch of the facial nerve; 20. Temporal branch of the facial nerve; 21. Zygomatic branch of the facial nerve; 22. Buccal branch of the facial nerve; 23. Marginal mandibular branch of the facial nerve; 24. Cervical branch of the facial nerve; 25. Submandibular salivary gland; 26. Submandibular lymph node; 27. Sublingual salivary gland.

nerve), the geniculate ganglion, the facial nerve, the chorda tympani nerve, the lingual nerve, the submaxillary and sublingual salivary glands[3,4].

Afferent pathways of the facial nerve are very numerous and are represented by cortico-pontine pathways (they reach the precentral motor cortex in the frontal lobe, located before the central cleft), indirect cortico-nuclear pathways (they are related to the reticulate substance), extrapyramidal pathways (partially distributed in the premotor areas of the orbito-frontal cortex) and afferent pathways in the subcortical areas[4]. Afferent fibres involve the reflex activity of the facial nerve: the blink reflex, the stapedial reflex and the sucking reflex.

The terminal motor branches of the facial nerve are

Table 1. Facial nucleus and efferent fibres.

MOTOR FIBRES	SECRETORY FIBRES	
Motor fibres ↓ Facial nucleus ↓ Facial nerve ↓ 1 2 3 4 1. Stapedius muscle 2. Stylohyoid muscle 3.Posterior digastric muscle 4. Platysma muscle	Lacrimo-muconasal system ↓ Facial nerve ↓ Geniculate ganglion ↓ Superficial greater petrosal nerve ↓ Pterygopalatine ganglion ↓ Sensitive innervation - lacrimal gland - nasal mucosa - palatal mucosa	Superior salivatory nucleus ↓ Wrisberg nerve ↓ Geniculate ganglion ↓ Facial nerve ↓ Chorda tympani nerve ↓ Lingual nerve ↓ - submaxillary salivary gland - sublingual salivary gland

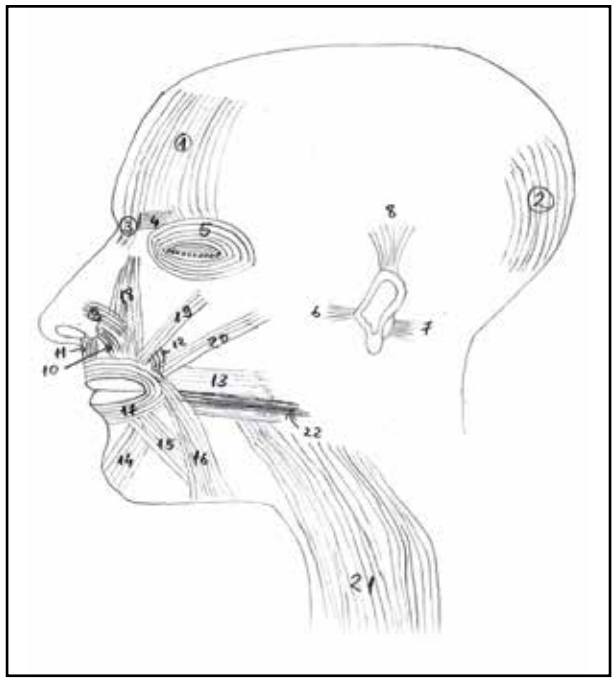

Figure 2. Schematic presentation of the motor facial nerve: 1. and 2. Occipitofrontalis muscle; 3. Pyramidal muscle; 4. Corrugator supercilii muscle; 5. Orbicularis oculi muscle; 6. Anterior auricular muscle; 7. Posterior auricular muscle; 8. Superior auricular muscle; 9. Transverse nasalis muscle; 10. Dilator naris muscle; 11. Depressor septi nasi muscle; 12. Levator anguli oris muscle; 13. Buccinator muscle; 14. Mentalis muscle; 15. Depressor anguli oris muscle; 16. Triangular muscle of the lips; 17. Orbicularis oris muscle; 18. Levator labii superioris alaeque nasi muscle; 19. Zygomaticus minor muscle; 20. Zygomaticus major muscle; 21. Platysma muscle; 22. Risorius muscle.

distributed to: eyelid and eyebrow muscles, auricular muscles, nose muscles, lip muscles and platysma muscle (Figure 2).

The secretory function of the facial nerve is performed by two preganglionic parasympathetic nuclei organized into two functional systems: the lacrimo-muconasal system (ensures parasympathetic innervation of the lacrimal glands, nasal mucosa and palatal velum) and the superior salivatory nucleus (Figure 3).

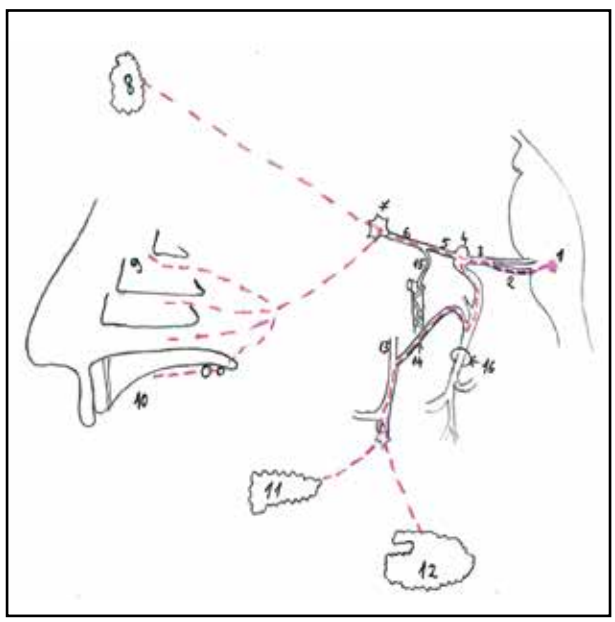

Figure 3. The secretory fibers of the facial nerve: 1. Superior salivatory nucleus; 2. Wrisberg nerve; 3. Facial nerve; 4. Geniculate ganglion; 5. Superficial great petrosal nerve; 6. Vidian nerve; 7. Pterygopalatine ganglion; 8. Lacrimal gland; 9. Nasal mucosa; 10. Palatal mucosa; 11. Sublingual salivary gland; 12. Submaxillary salivary gland; 13. Lingual nerve; 14. Chorda tympani nerve; 15. Deep petrosal nerve; 16. Stylomastoid foramen.

SYMPTOMS OF PERIPHERAL FACIAL NERVE PARALYSIS

Starting from the four functions of the facial nerve, peripheral facial nerve palsy is characterized by motor, sensory and visceral deficit in the affected hemiface.

The first and most significant sign is the facial asymmetry given by: the asymmetry of the oral commissure and flattening of the nasolabial fold (the lips are pulled towards the healthy side and there may be salivary leakage), the disappearance of expression wrinkles, the eyelid is partially or fully open (lagophthalmos), the lower eyelid droops and the orifice of the lacrimal gland becomes visible (epiphora). Subjectively, the patient reports a feeling of heaviness and swelling in the affected hemiface, eating disorders (difficult chewing and swallowing, food debris stagnates in the oral cavity after swallowing, loss or diminution of taste) and the inability to blink[2,5].

Voluntary movements are diminished or abolished on the affected side: closing the eyes, frowning, raising the eyebrow, swelling of the cheeks, whistling, while the smile becomes crooked or impossible[2,5].

Two essential signs are characteristic for the clinical diagnosis of peripheral facial paralysis: Charles Bell's phenomenon – upward and inward movements of the eyeball are disturbed, abolished eyelid occlusion; the Babinski sign – pulling down the affected labial commissure and a difficult forward movement of the mandible.

Depending on the signs and symptoms, the severity of facial nerve palsy can be assessed using the House-Brackmann Scale[6,7]:

- Grade I – Normal – normal facial functioning.
- Grade II – Mild dysfunction – slight weakness observed during a careful evaluation, slight asymmetry at the level of the mouth (visible when smiling), normal symmetry and tone at rest; the eye closes completely with minimal effort.
- Grade III – Moderate dysfunction – obvious weakness but without a noticeable difference between the two sides, normal symmetry and tone at rest; visible contracture or hemifacial spasm (but not severely); the eye closes completely, but with effort.
- Grade IV – Moderate-severe dysfunction – the patient cannot frown, asymmetry of the mouth, incomplete eye closure; normal symmetry and tone at rest.
- Grade V – Severe dysfunction – facial movement is hardly perceptible, incomplete eye closure, inability to frown, slight movement of the corner of the mouth; at rest, visible asymmetry.
- Grade VI – Total paralysis – absent movements and inertia at the level of the affected hemiface.

In central facial paralysis, upper facial motility (forehead, eyes) is preserved.

TOPOGRAPHIC DIAGNOSIS

Establishing a positive diagnosis of facial nerve palsy and especially the location of the lesion are two important steps in choosing treatment and evaluating the prognosis. Clinical examination provides data that can guide the topographic diagnosis and must be completed with an audiometric evaluation, Schirmer's test, salivation test, taste assessment[1,2,8].

Imaging and especially the electrophysiological evaluation of the facial nerve completes the battery of tests that help identify the lesion.

Imaging of the facial nerve is performed using the Computed Tomography (CT) and Magnetic Resonance Imaging (MRI)[9]. The CT examination requires high resolution systems that highlight nerve damage, especially at the level of the petrous pyramid, facilitating two-dimensional reconstructions of facial nerve segments (labyrinthine, tympanic or mastoid). Magnetic resonance imaging with gadolinium injection provides us with normal or pathological data on the entire trajectory of the facial nerve. The contrast agent is distributed in the perineural space around the facial nerve. At the level of the mastoid portion, for example, the facial nerve occupies about 40% of the fallopian lumen, the difference being occupied by arteriovenous plexuses.

For the accuracy of the diagnosis, the two imaging methods can be used complementarily.

Electrophysiological exploration of the facial nerve (the maximal and minimal stimulation test, electroneuronography, electromyography) consists in the fact that it shows the functionality of the nerve along its entire trajectory[1,10-13]. Various aspects of the conduction of the nerve influx are investigated (latency, amplitude), the sensorimotor reflex loops of the facial and trigeminal nerves, as well as the central-level connections of the trigeminal and facial nuclei.

The method provides essential data on the severity of axon or myelin damage – it has prognostic value, guides drug therapy and/or helps establish the surgical strategy when it is required.

According to the studies of Fraiss and collaborators, electrical investigation (electrodiagnosis) is made depending on the clinical aspect, evolution and the therapeutic strategy according to the following algorithm[14]: stimulation electrodiagnosis, the study of the blink reflex, detection electrodiag-

nosis (needle or surface electrodes), electroneurography, Hilger test (nerve stimulation), magnetic stimulation, antidromic stimulation.

In practice, surface electromyography (EMG) shows the extent of the paralysis. The stimulation electromyography performed on day 5-7 after onset indicates the extent of nerve degeneration. The results of this test can be correlated with the severity degrees of the House-Brackmann scale. Thus, neurapraxia determined by EMG corresponds to grade I of severity, having the highest rate of spontaneous recovery, axonotmesis corresponds to grades II and III (incomplete nerve degeneration), and neurotmesis to grade IV[15].

Needle detection electromyography, starting on the 20th day from the onset, indicates the potential for nerve recovery.

ETIOLOGY OF FACIAL NERVE PARALYSIS

Most facial paralysis are idiopathic, in about 70% of cases of facial paralysis the doctor being forced to make an exclusion diagnosis, since there is no obvious cause[1,16]. Traumatic (10-23%), infectious (up to 7% of cases, viral or bacterial), tumor (2.2-5%) causes follow[1,17]. A special place is occupied by the child's facial paralysis.

The most frequent are unilateral. The existence of bilateral facial paralysis (0.3-2%) often implies a manifestation of a general systemic disease (for instance, Lyme disease, diabetes, sarcoidosis, Parkinson's disease, multiple sclerosis, Guillain-Barre syndrome)[18].

The most commonly affected age group is between 15 and 45 years old, but there is no age or gender specificity[1].

Facial paralysis of the newborn and child

Paralysis of the facial nerve in the pediatric population is a relatively rare pathology, some authors reporting an incidence of 0.05-0.2%[19,20]. From an epidemiological point of view, they can be acquired (traumatic, infectious, inflammatory, neoplastic) or congenital (genetic or malformations, trauma at birth).

Prematurity, the use of forceps, cesarean section, macrosomia (weight greater than 3,500 grams) are part of the causes of facial paralysis in the newborn[21,22]. Recovery is complete within a few months after birth.

Malformative causes can be syndromic or non-syndromic. Among the syndromic ones, we mention the Moebius syndrome (facial diplegia, bilateral paralysis of the external oculomotor nerve) with a prevalence of 1/150,000 of live births, Goldenhar syndrome (oculo-auriculo-vertebral dysplasia), syringobulbia (congenital pseudobulbar paralysis), Arnold-Chiari syndrome or Franceschetti-Zwahlen syndrome (mandibulofacial dysostosis and auricular aplasia)[23-25].

Genetically, hereditary myopathies such as myasthenia or myotonic dystrophy with the identification of chromosomal loci 3q21-22 and 10q21.3-22.1 may be incriminated in the occurrence of facial paralysis in the newborn[1,2].

The most common causes of peripheral facial paralysis in preschool and school children are "à frigore", otitic, traumatic, viral facial paralysis or rare causes (lymphoma, hemopathy, metabolic diseases)[26]. The treatment of paralysis, as in adults, is that of the primary disorder.

Traumatic facial nerve paralysis

Traumatic causes are the second most common cause of paralysis of the facial nerve, and they can be represented by fractures of the temporal bone, ballistic traumas or nerve wounds in its extracranial trajectory. Temporal bone fractures involving the facial nerve can be of three categories, depending on the trajectory of the fracture line and the intercepted organs: longitudinal, transverse or mixed[27]. Longitudinal fractures, the most common (70-90%), occur in a plane that begins at the level of the petrous ridge, goes to the mastoid, labyrinth, then involving the anterior wall of the external auditory canal, the glenoid cavity, the second portion of the facial canal up to the geniculate ganglion[28]. Transverse fractures (10-15%) have a trajectory from the occipital hole to the labyrinthine portion of the facial nerve[29]. Oblique fractures are a combination of the two trajectories described above, situation that results in damage of the facial nerve in several areas of its trajectory.

Traumatic facial paralysis occurs in 70% of cases immediately after the trauma, and in 30% of cases occurs late, at 5-7 days, depending on the mechanism of production and the effectiveness of the treatment[30].

The early post-traumatic mechanisms are represented by: partial or total sectioning of the nerve, intra- or extra-nervous hematoma and/or edema, nerve compression, elongation of the nerve trunk, damage or destruction of the Schwann sheath. The mechanisms of facial paralysis appearing later after the trauma occur through ischemia and edema.

Clinically, facial paralysis in temporal bone fracture may be associated with retroauricular ecchymosis, otorrhea or hemotympanum, CSF fistula, hypoacusis (mixed hypoacusis occurs in the mixed fracture), trigeminal or oculomotor nerve palsy, taste disorders.

In order to establish the topographic diagnosis, it is necessary to perform a computed tomography completed, depending on the needs, by a cranial MRI[31]. The CT should assess the entire facial nerve pathway.

The electrophysiological examination of the facial nerve requires the Hilger test (the minimal excitability threshold of the facial nerve), the electroneurography – the Esslen test (denervation potentials) and the detection electromyography.

Immediate post-trauma treatment addresses the general symptomatic context and will include antiedematous, antihemorrhagic, anti-infective agents, while surgical treatment is performed depending on the trajectory of the fracture.

Depending on the topography of the lesion, surgical approach techniques vary, the surgeon being able to choose: the transmastoid route, the transmastoid extralabyrinthine route, the translabyrinthine route or the route of the middle cranial fossa. Usually, for longitudinal fractures, the approach is made through the middle cranial fossa. For transverse and mixed fractures, the approach through the route of the middle cranial fossa combined with the transmastoid route is used when hearing is present, or the translabyrinthine approach, when hearing is impaired[32].

Tumor causes of facial paralysis

A slow onset facial paralysis, with insidious evolution, may reveal the existence of a tumor (benign or malignant) on the nerve pathway (facial nerve neuromas), at the level of the cerebellopontine angle, of the petrous temporal bone or a cholesteatoma[1,33].

Cranial MRI examination with gadolinium contrast agent is decisive for establishing the diagnosis and announcing the therapeutic strategy. Facial neurinoma can develop in any part of its pathway (internal auditory canal, labyrinth, geniculate ganglion, tympanum, mastoid, tympanic cord, parotid). Angiomatous lesions are usually located in the geniculate ganglion area, while the jugulotympanic glomus extends very rarely to the facial nerve.

Otitic facial paralysis occur as a result of middle ear and mastoid disorders, neglected or ill-treated (necrotizing malignant otitis, otomastoiditis, cholesteatoma). They benefit from surgical treatment adapted to the underlying disease.

Facial paralysis of general causes

General diseases affect the facial nerve through an intrinsic mechanism unlike all other causes that are extrinsic. The mechanisms are complicated, sometimes unclear and specific to each type of general disease. Facial paralysis may occur in collagenoses, metabolic diseases, intoxications, deficiencies, or may have neurological causes. When it appears, facial paralysis is a secondary symptom within a symptomatic complex specific to the underlying condition. Bilateral involvement can be described most frequently in this group.

A special place is occupied by facial paralysis in Lyme disease. Lyme disease is caused by the spirochete Borrelia Burgdorferi inoculated by the tick bite and has three stages of evolution:

- Primary – with cutaneous manifestations strictly at the level of inoculation.
- Secondary – hematogenous dissemination with chronic multi-organ erythema migrans.
- Tertiary – months or years after inoculation, with rheumatic, dermatological or neurological manifestations.

Peripheral facial paralysis occurs in the second and third stages of Lyme disease[34].

In support of the diagnosis, generally revealed by the anamnesis and the staged evolution of the disease, come: CSF examination which reveals relevant elements of lymphocytic meningitis, ELISA serology antispirochete antibodies IgG and IgM[1,2,34].

The treatment is third-generation amoxicillin and cephalosporin antibiotic therapy.

Infectious facial nerve paralysis

Infectious causes of facial paralysis can be of viral or bacterial nature. The viral etiology is the most commonly incriminated, in 4.5 – 7% of cases[1], the activation of the Herpes Zoster virus being described with an increased incidence in both adults and children.

Facial paralysis due to the Herpes Zoster virus is installed by affecting the geniculate ganglion and is known as Ramsay-Hunt syndrome. The Herpes Zoster virus is a DNA molecule (viral genome) in a protein packaging. This viral envelope does not move, its encounter with the nerve cell being random. For each type of virus there is a specific type of host cell, where it can penetrate and develop to the detriment of the latter and of the tissue of which it is a part. For the zoster virus, the preferred host cells are those of the spinal or cranial ganglia, nerve cells and ectodermal cells in the skin.

The works of Hope and Simpson (1965) later confirmed by modern virology (Mahalingan in 1990) support the idea that in zoster infections, the primary infection is the varicella virus[35,36]. After generalized varicella infection, the virus remains dormant in the body, preferably grouped in the mentioned nervous areas, becoming uninfluenced by varicella antibodies and ready to manifest patho-

logically as soon as the host organism suffers from immunosuppression. At this moment, the virus, through a process of nucleocapsid pinocytosis, enters the nerve cell, modifying its metabolism and functionality for the benefit of its multiplication and destruction of nerve structures in this regard. The main impact virus – nerve tissue is made in the following areas: geniculate ganglion, nerve trunk (axonal myelin degeneration) and at the level of the neuromuscular junction. At the level of these vivo-neural impact areas, two types of histopathological lesions occur: nuclear damage and compressive, massive edema with cellular infiltration.

From a clinical point of view, one has described a number of features of facial paralysis with shingles, initially established by Ramsay-Hunt, and which are still valid today[37]:

- Simple auricular area involvement – otalgia – blisters at the eardrum and external auditory canal level; vesicular rash inside the sensitive area of the facial nerve; moderate general infectious context.
- Incomplete auricular area involvement has in addition to the first painful peripheral facial paralysis and positive serology.
- Complete auricular area involvement – high-frequency perceptual hearing loss; irreversibility of lesions; facial paralysis; accentuated general infectious context.
- Associated zoster facial paralysis – lingual area; ophthalmic area; occipitocervical area.

The treatment involves the mandatory combination of antivirals and cortisone anti-inflammatory drugs, the precocity of instituting the treatment being decisive.

Failure or partial inefficiency of corticosteroids and antiviral treatment requires surgical decompression of the facial nerve.

"À frigore" facial nerve paralysis

"À frigore" peripheral facial nerve paralysis or Bell's palsy is idiopathic, without being able to establish its precise etiology[38]. It appears outside a viral context, only 14% of cases occur in a seasonal viral context; the cold seems to be the main favouring and even triggering factor[39].

It represents up to 70% of all facial nerve palsies[1] and has an equal frequency in both sexes and a preferential incidence for the 25-60 age groups[39]. From an evolutionary and prognostic point of view, it is more severe in diabetics and pregnant women.

The evolution of the disease is favourable in the proportion of 75-85%, and recovery after a correct and complete treatment is complete after 6 weeks[39]. It is recurrent in varying proportions (geographical area, authors) between 10-14%[39,40].

Clinical symptoms are not specific and often inconsistent, and may be represented by: ear pain, mild flu syndrome, cutaneous hypoesthesia, painful hyperacusis, all paraclinical examinations being negative.

A very important role in the treatment and especially in the evaluation of the possibility of recovery is played by the topographic diagnosis. The location of the lesion is identified using the tear test (Schirmer), the stapedial reflex testing and electrogustometry.

Electrical tests – surface, stimulation, detection electromyography and especially Blink electromyography – reflexes have an evaluative and prognostic role in evolution and recovery.

Currently, there are three types of therapies in practice: corticotherapy – according to Stennert's protocol[41] (Table 2), antiviral therapy, surgical decompression of the facial nerve in the cases which do not respond to the mentioned treatment.

PROGNOSIS

Assessing the prognosis of facial nerve paralysis can be difficult, taking into account the possible causes and topography of the lesion.

In the literature, several factors are described that can be associated with a negative prognosis[1,42,43] :

√ Complete paralysis
√ Absence of stapedial reflex
√ Age over 50
√ Absence of signs of recovery 3 weeks after paralysis onset
√ Ramsay-Hunt syndrome
√ Poor response to electrophysiological tests.

SEQUELAE AND COMPLICATIONS OF FACIAL NERVE PARALYSIS

Being located at the level of the face, sequelae and complications of paralysis[1,15] of the facial nerve are embarrassing and generating depression with behavioural changes; they can even lead to loneliness tendencies.

Ocular complications can be represented by lagophthalmia, keratitis, conjunctivitis, eversion of the lower eyelid with tears running down the cheeks (epiphora)[44,45]. They occur during the evolution of a facial paralysis and require proper supervision and treatment (artificial tears). They disappear or reduce their intensity depending on the degree of recovery of the facial nerve.

The sequelae of facial paralysis are motor and sensory. Motor sequelae are total paralysis, homo- and

Table 2. Stennert's protocol[41].

Days of treatment	Cortisone (prednisolone – equivalent dose) (mg/day)			Dextran 40 with sorbitol or mannitol 5–10% (ml)	Pentoxifylline (Trental) (ml)
		<70 kg	>70 kg		
Hospitalized	Infusion	200	250	500	15
1				500	15
2		200	250	500	15
3		150		500	15
4		150		500	15
5		100		500	15
6		100		500	15
7		75		500	15
8		50		500	15
9	Oral circadian (6–8 a.m.)	40		500	15
10		20		500	15
Ambulatory care					
11		15			
12		12.5			
13		10			
14		7.5			
15		5			
16		2.5			
17		2.5			
18		2.5			

contralateral muscle contracture, synkineses, hemifacial spasm[1]. Besides having a negative prognosis, total paralysis leads over time to remarkable atrophy of the involved hemiface muscles, with severe chewing and eye disorders.

Muscle contracture can occur both at the level of the involved hemiface, and in the healthy area. Homolateral contracture is a manifestation occurring at a distance in time from the moment of paralysis onset; it evolves progressively, also causing in most cases a contracture to accompany the contralateral hemiface. The latter is a natural, spontaneous, uncontrolled attempt, which tries to obtain by repetition an as weak muscular activity as possible on the paralyzed side.

Synkineses manifest clinically through uncontrolled contractures of the oral commissure and of blinking on the affected side[1]. They set in a few months after the onset of facial paralysis and are due to aberrant nerve regeneration that extend beyond the nerve sheath and its territory of innervation (aberrant reinnervation).

Hemifacial spasm, also called post-paralytic spasm, must be detected early by electrical examination of the nerve and / or by testing the muscle tone of the facial muscles[1,46]. This test imagined by Georges Freyss consists in giving each facial muscle (10 lateral and medial muscle groups) a grade between 0-30 depending on its degree of motricity (0 – total paralysis, without muscle contracture; 30 – normal contraction)[44].

Symptomatic facial spasm is fundamentally different from essential hemifacial spasm. The latter is due to a neurovascular conflict located at the emergence of the facial nerve.

The secretory syndrome can be considered the correspondent of synkineses, but with afferent sensory circuit (tactile, olfactory, gustatory) having an effector, secretory agent (lacrimal gland, salivary gland), the impulse passing through the lacrimomuconasal nucleus.

Xerophthalmia, dry eye without tears, can lead to eye loss if, during the evolution of facial paralysis, it is not treated with artificial tears or blepharorrhaphy.

Frey's syndrome is characterized by sweating during feeding appeared at the level of the parotid area (common after parotidectomies) caused by anastomosis errors between the nerve threads of the facial nerve with the nerves of the sweat glands in the facial skin.

Crocodile tears syndrome is a classic complication of Bell's palsy (à frigore)[1,47]. The etiology and mechanisms of this complication are still obscure. A possible aberrant connection of parasympathetic fibres intended for the lacrimal gland with fibres

from the geniculate ganglion is considered.

The treatment of sequelae of facial nerve paralysis is primarily one of recovery through methods of relaxing physiotherapy of the facial muscles. An important role in these cases is played by transcutaneous neuromuscular electrical stimulation (NMES)[48,49]. NMES consists in the muscular and nervous stimulation with an electric current at a certain intensity (mA) to determine the contraction of the deep muscles in the area being worked. This intensity is called the motor threshold. Electrical stimulation acts on type II muscle fibres that are involved in strong, high-speed contractions. Type I muscle fibres are activated by traditional therapy for the recovery of a facial paralysis. Therefore, the combination of the two treatment methods involves the activation of both types of muscle fibres and, in this way, the positive results of the rehabilitation process can increase[50]. In our experience, using associated NMES and classical mime exercises (swollen cheeks, smile through gritted teeth, smile without showing teeth, slow closing of the eyes) the results are better in terms of recovery of these patients than in the case of NMES as single therapy. Voluntary muscle contraction associated with electrical stimulation seems to produce greater activation of the central nervous system than electrical stimulation alone[51]. During combination therapy (NMES and mime exercises), the so-called motor adaptation or recalibration occurs, which can last a long time after the cessation of treatment. The limit of NMES is the association of a demyelinating disease of the central nervous system, in which case recovery with this type of stimulation is partial.

In case of failure of physiotherapy, plastic surgery is used; nevertheless, the latter provides modest results (nerve graft, implant at the level of the upper eyelid, facelift, etc.)[52].

Botulinum toxin injection appears to have satisfactory results in synkineses and facial spasm[53].

CONCLUSIONS

Facial nerve palsy is a common pathology in medical practice. Due to the multitude of factors that can determine or favour its appearance, it requires a multidisciplinary evaluation consisting of otorhinolaryngologist, neurologist, ophthalmologist, internist.

Early presentation to the doctor, accurate determination of the cause, correctly performed topographic diagnosis is the key to proper treatment and complete functional recovery.

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