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EDITORIAL

Facial nerve paralysis in children

Andrea Ciorba, Virginia Corazzi, Veronica Conz, Chiara Bianchini, Claudia Aimoni

Andrea Ciorba, Virginia Corazzi, Veronica Conz, Chiara Bianchini, Claudia Aimoni, ENT and Audiology Department, University Hospital of Ferrara, 44100 Ferrara, Italy

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Correspondence to: Andrea Ciorba, MD, PhD, ENT and Audiology Department, University Hospital of Ferrara, Via A Moro 8, loc Cona, 44100 Ferrara, Italy. andrea.ciorba@unife.it
Telephone: +39-532-239746
Fax: +39-532-237447

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Abstract

Facial nerve palsy is a condition with several implications, particularly when occurring in childhood. It represents a serious clinical problem as it causes significant concerns in doctors because of its etiology, its treatment options and its outcome, as well as in little patients and their parents, because of functional and aesthetic outcomes. There are several described causes of facial nerve paralysis in children, as it can be congenital (due to delivery traumas and genetic or malformative diseases) or acquired (due to infective, inflammatory, neoplastic, traumatic or iatrogenic causes). Nonetheless, in approximately 40%-75% of the cases, the cause of unilateral facial paralysis still remains idiopathic. A careful diagnostic workout and differential diagnosis are particularly recommended in case of pediatric facial nerve palsy, in order to establish the most appropriate treatment, as the therapeutic approach differs in relation to the etiology.

Key words: Facial paralysis; Seventh cranial nerve; Children; Bell's palsy; Therapy

Core tip: Pediatric facial nerve palsy can be congenital or acquired and its etiology can remain unknown. Bell's palsy is the most frequent form of facial paralysis also in children; about 70% of these cases has a favorable prognosis with spontaneous resolution. An accurate differential diagnosis is necessary to assess the prognosis and the therapeutic options. In Bell's palsy, the use of oral corticosteroids is recommended also in children, preferably within 3 d from onset. In children presenting a permanent congenital or acquired facial palsy, the therapeutic strategy consists in surgical techniques associated to rehabilitative approaches.

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INTRODUCTION

Pediatric facial nerve palsy can be congenital or acquired. Despite efforts to define its etiology, the cause of paralysis can often remain unknown. Idiopathic facial paralysis, even in childhood, is commonly known as Bell's palsy,

Table 1 Possible causes of facial nerve palsy in children

Causes of facial nerve palsy in childhood	
Idiopathic	Bell's palsy
Congenital	Delivery traumas:
	Primiparity
	Birth weight > 3500 g
	Forceps
	Cesarean section
	Prematurity
	Syndromic malformative:
	Möbius syndrome
	Goldenhar syndrome
	Syringobulbia
	Arnold-Chiari syndrome
	Genetic:
	Hereditary myopathies
	3q21-22 and *10q21.3-22.1* mutations
Acquired	Infectious:
	Ramsay Hunt syndrome
	Epstein-Barr virus
	Haemophilus influenzae
	Tubercolosis
	Lyme disease
	Cytomegalovirus
	Adenovirus
	Rubella
	Mumps
	Mycoplasma pneumoniae
	Human immunodeficiency virus
	Acute otitis media
	Chronic otitis media/cholesteatoma
	Inflammatory:
	Henoch-Schönlein porpora
	Kawasaki syndrome
	Neoplastic:
	Schwannomas of the Ⅶ c.n.
	Hemangiomas
	Rhabdomyosarcoma
	Temporal bone histiocytosis
	Leukemia
	Parotid gland tumors
	Traumatic:
	Temporal bone fractures
	Iatrogenic

named after the Scottish surgeon Sir Charles Bell, who, in 1821, firstly described a "weakness" of the facial nerve[1].

Aim of this paper is to describe the most common causes of facial nerve palsy in children and therefore the most appropriate available treatments.

STUDY METHODS

Narrative review. PubMed database was searched up to April 2015, for meta-analysis, systematic reviews, and controlled trials, going back for 10 years. The search was conducted independently and was restricted to children. Full text articles were required when the title, abstract or keywords indicated that the study could be suitable for this review. Additional papers were also identified from the references in the chosen literature.

The medical subject heading used included "facial paralysis"; "Bell's palsy"; "children"; "seventh nerve"; "therapy".

EPIDEMIOLOGY AND ETIOPATHOGENESIS

There are many possible causes of facial nerve paralysis in children. These can be classified as congenital (traumatic, syndromic and non-syndromic malformations, genetic) or acquired (infectious, inflammatory, neoplastic, traumatic)[2,3] (Table 1).

Unfortunately, in about 50% of the cases, the etiology remains unknown: these forms are classified as Bell's palsy. In children, Bell's palsy has an estimated incidence of about 6.1 cases per year per 100000 in those aged between 1 and 15 years[2,3]. It is believed that it can be caused by viruses such as Herpes simplex 1. About 70% of Bell's palsy has a favorable prognosis with spontaneous resolution within 3 mo, without sequelae. The paralysis severity at onset can influence the degree of recovery: a severe paralysis hardly obtains a complete recovery of nerve function[4-7].

Congenital facial paralysis can result from developmental defects or delivery traumas. Perinatal traumas are the most frequent causes of congenital paralysis. The main reported risk factors associated to traumatic facial paralysis are: mother's first child, birth weight greater than 3500 g, use of forceps, cesarean birth and prematurity. These cases have usually a favorable prognosis, with infants recovering the full functionality of the seventh cranial nerve within few months without sequelae[8,9].

A congenital facial nerve paralysis, although other cranial nerves such as the Ⅲ, Ⅳ, Ⅴ, Ⅷ can be involved, is presented within the Möbius syndrome. The reported prevalence of this syndrome is about 1/150000 live births[9-12]. It is reported to be due to hypoplasia of the motor nuclei of the cranial nerves within the brainstem, probably due to a hypoxic-ischemic encephalopathy[10]. Those affected by Goldenhar syndrome (hemifacial microsomia, with a spectrum of congenital malformations involving the structures derived from the first and second branchial arch) can also present a congenital facial paralysis[11]. Congenital pseudobulbar palsy (Syringobulbia) is a condition that clinically manifests with facial paralysis, dysphagia and speech difficulties, while in the Arnold-Chiari syndrome, congenital facial paralysis is usually associated to other cranial nerves paralysis (especially the Ⅵ one) due to malformations of the posterior fossa that allow herniation of brain structures through the foramen magnum[12].

Genetic causes of facial nerve paralysis includes hereditary myopathies, such as myotonic dystrophy and myasthenia. Also two loci responsible for isolated hereditary forms of facial paralysis (chromosome 3q21-22 and 10q21.3-22.1) have been identified[9,10,13].

Acquired facial paralysis can frequently be due to viral infections. The reactivation of Herpes Varicella-Zoster

may be responsible, even in children, of Ramsay Hunt syndrome (zoster oticus); in this case, facial palsy can be associated to the presence of vesicular lesions of the external auditory canal and/or of the auricular concha. The incidence of this syndrome under 10 years of age is reported to be 2.7/100000[9,11,14]. Not frequently, a bilateral facial nerve palsy may be the onset of a Epstein-Barr virus, Haemophilus influenza, tuberculosis or Borrelia burgdorferi infection. Lyme disease has become the most common cause of acute facial paralysis in children in those areas where Borrelia Burgdorferi infection is endemic[9,15]. Other agents that may cause facial nerve palsy in children are cytomegalovirus, adenovirus, rubella, mumps, Mycoplasma pneumoniae and HIV[3,13].

Facial nerve palsy may also be present as a complication of several diseases such as acute and chronic otitis media, cholesteatoma, mastoiditis and meningitis[16,17].

Other inflammatory diseases such as vasculitis and Henoch-Schönlein porpora or Kawasaki syndrome can also occur with facial nerve palsy[9].

Rarely, in children, facial nerve paralysis can be due to tumors such as schwannomas or hemangiomas of the seventh nerve or bone tumors such as rhabdomyosarcoma and histiocytosis.

Pediatric facial nerve paralysis has been also described associated to leukemia (in many cases bilateral) or to parotid gland tumors[9,18].

Finally, traumas such as temporal bone fractures (longitudinal, transverse and oblique) can cause facial nerve palsy in children[19], while iatrogenic paralysis can occur after surgery of the parotid gland, middle ear or mastoid[8,9].

CLINICAL FEATURES

The peripheral paralysis of the seventh cranial nerve is characterized by motorial, sensorial and visceral deficits of the hemi-face involved. There is a facial asymmetry at the examination of the face: the facial creases and the nasolabial fold disappear; the affected side also presents a dropping mouth rim (with possible saliva leakage), eyelid widening and lagophthalmos (static signs). Dynamic signs are represented by the inability to whistle, puffing cheeks, frown, close the eyelid. Signs of Bell and Nigro can be present. Hyperacusis, due to paralysis of the stapedius muscle, can be present, too[1,5,15].

The little patient may also report paresthesias or pain of the pinna or of the concha. Lacrimal and salivary production can be reduced (visceral deficit). Lagophthalmos can promote corneal irritation; furthermore the child may complain of a metallic taste in the mouth due to the taste alteration of the anterior 2/3 of the tongue[1,5,10,20].

In a very young children and in newborns, the unilateral facial paralysis can be suspected when, in absence of front and nasolabial groove motility, there is also asymmetry of the face with buccal deviation when crying. In cases of severe paralysis, the child cannot close the eye due to a complete absence of movement on the affected side and there is an asymmetry of the face at rest. In newborns, this condition can also hamper breastfeeding[10,21].

In all the cases, the occurrence of facial nerve palsy in children represents a serious clinical problem also due to the functional and aesthetic outcomes affecting the quality of life; this feature is cause of significant concern in the little patients and their parents as well as in doctors.

DIAGNOSIS

A comprehensive history evaluation is always important for the correct diagnosis. It is necessary to investigate about the onset and the time course of the paralysis and its eventual progression (*e.g.,* a gradual onset, > 3 wk, may suggest a neoplastic etiology). All the associated symptoms should be identified, as well as any other comorbidities affecting the child[14,15,18-21].

During the ENT examination, particular attention should be given to the inspection of the external auditory canal, the eardrum and the mastoid region. The facial nerve evaluation, in terms of facial movements and spontaneous expressions, should be classified according to House-Brackmann grading system, whenever the child is cooperative. Both the eye and palpebral region as well as the lower face should be careful observed at rest and at movement, eventually documenting the asymmetry using a camera or a video-camera. Computer systems can also provide tools for measuring the facial asymmetry[22].

The audiological evaluation is important in order to assess the presence of stapedial reflexes (topo-diagnosis) and eventually to evidence the presence of hearing loss[15,21].

Blood pressure and blood count should be verified in all cases of pediatric paralysis. Particularly, in children it has been described that high blood pressure levels can be associated to recurrent facial palsy[14,21]. Furthermore, a moderate increase of monocytes and lymphocytes is compatible with Bell's palsy, as far as this analysis does not place definitive diagnosis nor exclude an inflammatory process. The lumbar puncture is performed only when suspecting a meningitis (severe headache, fever, papilledema, neck stiffness) or a Guillain-Barré syndrome: in this last case, the analysis of the cerebrospinal fluid shows a characteristic increase in protein not accompanied by a consensual cells increasing (albumin-cytological dissociation)[1,5,9,13,21].

Specific laboratory and imaging tests are not routinely indicated, but are recommended for patients with recurrent paralysis or when there has been no improvement after 3 wk of therapy. With the purpose of diagnose the Ramsey Hunt syndrome in children, an ELISA serum searching for IgM and IgG antibody titer against Herpes Varicella-Zoster is recommended[14]. Serologic tests for Lyme disease should be carried

out when the history of the patient suggests a possible exposure, while in case of clinical suspicion of a neoplastic etiology, the computed tomography of petrous bone and the brainstem magnetic resonance imaging must be performed. Radiological images are required even when the child shows other neurological manifestations or in suspected chronic otitis media, acute mastoiditis or temporal bone fracture[14,15,18-21].

Electrophysiological studies can be useful to identify the cause of the paralysis, to define the prognosis and follow-up of functional recovery, but they are still not considered necessary in all pediatric patients[14,15,18-21].

PROGNOSIS

To assess the prognosis of facial paralysis can be difficult, especially in children, even if the possibility of a complete functional recovery is greater in pediatric cases than in adult ones.

The degree of paralysis represents a prognostic element: patients with partial paralysis have a better prognosis. Actually, the Ⅱ degree according to House-Brackmann scale has a good outcome, while the Ⅲ and the Ⅳ degrees are associated to moderate residual dysfunctions. The Ⅴ and the Ⅵ degrees, instead, have poor possibility of recovery[1,23].

Perinatal traumatic forms usually have a good prognosis, with a possible spontaneous resolution within 4 mo of life[9,13,21]. Bell's palsy has a generally optimal functional recovery in a short period of time; a favorable prognostic indicator is represented by a clinical improvement within 3 wk by the onset[1,13,23]. Ramsey Hunt syndrome has a worse prognosis compared to Bell's palsy: only 10% of severe paralysis due to reactivation of Herpes Varicella-Zoster have a full recovery[14].

The prognosis of facial paralysis caused by tumors is of course related to the type and stage of the tumor and the treatment performed[8,9,13,15,21].

It has been reported that in about 5% of cases, the affected side may develop residual sequelae like contractures, spasms, synkinesis[1,15]. The latter, in particular, affect the symmetry and facial expressiveness and usually recognize three possible pathogenetic mechanisms: an aberrant axonal regeneration, an aberrant nerve impulse transmission and a hyperexcitability of the nucleus of the facial nerve. The most common synkinesis affects the eye and mouth muscles: during a voluntary movement of the mouth, for example a smile, there could be an involuntary eye closure and *vice versa*. Less frequently, involuntary movements of the chin can be seen during voluntary movements of the mouth or the voluntary eye closure[24]. A similar phenomenon can occur with the autonomic fibers: for example, when eating, the activation of salivation causes also lachrymation (phenomenon known as "crocodile tears")[1,15,25].

THERAPY

The treatment of facial palsy is related to the etiology and the severity of the palsy itself. When a specific cause is identified, treatment is aimed to resolve the underlying cause. The therapeutic approach in children often involves a multidisciplinary team, comprehending otolaryngologists, pediatricians, neurologists, ophthalmologists, maxillofacial surgeons, plastic surgeons, physiotherapists (Table 2).

Drug therapy

In the idiopathic cases of facial palsy, the main limitations regarding drug therapy in children concern the lack of controlled clinical trials on children with Bell's palsy and its favorable natural history[9,13]. Since most of these forms in childhood recover spontaneously, aim of the drug therapy is to minimize the possibility of incomplete resolutions and reduce the risk of sequelae, such synkinesis, autonomic dysfunctions (*e.g.,* crocodile tears), facial spasms[26]. When Bell's palsy occurs in adults, it is well known that glucocorticoids in combination with antiviral therapy (acyclovir or valacyclovir) are recommended[27-30]. In children, the use of oral corticosteroids is recommended preferably within 3 d from onset of symptoms (the suggested treatment regimen is prednisone 1-2 mg/kg per day for 10 d, gradually decreasing the dose)[13,31] as the majority of patients improves in the first three weeks[32], although several studies did not find significant differences between the outcomes of children treated with corticosteroids and not[20,33-35]. The Ramsay Hunt syndrome, instead, should be treated as soon as possible with intravenous steroid associated with antivirals in children older than 2 years (*e.g.*, acyclovir 80 mg/kg per day every 6 h for 5 d or, in children older than 12 years, valacyclovir 20 mg/kg three times per day, up to a maximum of 1000 mg three times daily), in order to obtain a full recovery in 75% of cases if treated within the first three days from onset[5,13,36].

The majority of children has a spontaneous recovery, but for both congenital and acquired forms, particular attention should be paid to the corneal protection, resorting to the use of protective devices and lubrication with artificial tears to prevent irreversible corneal lesions. Rarely, persistent paralysis with an important lagophthalmos may require a tarsorrhaphy or the implantation of a temporary weight in the upper eyelid. Moreover, in infants with difficulty in suction due to mouth muscles involvement, it is essential to provide an alternative nutritional support[1,9,13,15,21,37].

Children with persistent severe paralysis require a long follow-up. The absence of signs of functional recovery after six weeks requires a comprehensive reassessment of the diagnostic-therapeutic approach[13].

Infants with congenital paralysis for perinatal trauma, usually have a good prognosis even without treatment. For those presenting a neural damage, there are surgical solutions in combination with steroid therapy, depending on the severity of the case[9]. The direct neurorrhaphy has an excellent prognosis, due to the large neuronal plasticity and the excellent regenerative capacity in the childhood. Alternatively the use of a nerve graft is

Table 2 Therapeutic approaches to facial nerve palsy in childhood

Therapy of facial nerve palsy		Outcome[1]
Drugs	Bell's palsy	
	Oral steroids within 3 d of onset	70% recovery after 3 wk[32]
	Ramsay Hunt syndrome	
	Intravenous steroids as soon as possible	75% recovery at 6 mo if treated within 3 d from onset; 30% recovery at 6 mo if treated after 7 d from onset[36,44]
	Antiviral agents	
	Other conditions	
	Targeted therapies for specific diseases	N/A
Protective measures	Eye protection	N/A
	Artificial tears	N/A
	Tarsorrhaphy	N/A
	Eyelid weight implant	N/A
	Nutritional support	N/A
Surgery	Traumatic palsy	
	Neurorrhaphy within 72 h	N/A
	Nerve grafting within 72 h	N/A
	Other conditions	
	Dynamic facial reanimation	
	Temporalis elongation mioplasty	80% recovery within 1 mo[38]
	Gracilis muscle microvascular free flap	89% recovery within 4-6 mo[45]
	Sural nerve grafting	N/A
	Cross-facial nerve grafting	83% recovery within 1 yr[46]
Rehabilitation approaches	Botulinum toxin	100% recovery (temporary)[24]
	Physiotherapy	N/A
	Biofeedback therapy	N/A
Regenerative therapy	Bioelectrical interface/electrode	N/A
	Stem cells and bio-scaffolds	N/A

[1]When available.

also described, with discrete functional and aesthetic results[3,9]. In both cases, the repair of the nerve should be completed within 72 h from the trauma onset[3,13].

Surgical therapy

In the pediatric population, the surgical decompression of the facial nerve in its labyrinthine segment is not recommended[3,9], primarily due to the lack of systematic clinical studies demonstrating its real effectiveness and secondly due to the risk of sensorineural hearing loss occurrence. In children presenting a permanent congenital or acquired facial palsy, surgical techniques of dynamic facial reanimation can be considered in order to tentatively restore a static and dynamic facial symmetry. Among these, the most performed are locoregional muscles transfers and muscle and nerve grafts[10,13]. In particular, a frequently performed intervention is the temporalis elongation myoplasty: the tendon of the temporal muscle is moved from the mandibular coronoid process to the lips, with 80% of children regaining a sufficient symmetry within a month[38]. A similar intervention is the bilateral anterior third of the masseter muscle transfer above the corners of the mouth. Also the employment of microvascular free flaps of gracilis muscle has been proposed[10,13]. Another microsurgical technique consists of nerve grafts (usually sural nerve) between the branches of the facial nerve of the healthy side of the face and those of the injured side (cross facial nerve grafting). This practice allows the healthy facial nerve to send a symmetrical and synchronous pulse to the paralyzed side[3,10,24,37,39,40]. Children have the best chances of success with this type of surgery[10,15]. When it is not possible to perform a cruciate graft, a neural transposition from a donor site of the same side of the facial paralysis can be proposed (*e.g.*, the hypoglossal nerve or the trigeminal motor branch): the nerve is partly or completely dissected and anastomosed to the distal part of the paralyzed facial nerve[10,15].

Rehabilitation approaches

Among the proposed treatments for synkinesis and emifacial spasms, the botulinum toxin has been proposed also in childhood[15,24]. Unfortunately, the toxin has a temporary effect, making necessary to repeat the injections. Moreover, the periods of relief from synkinesis become more and more short. Better results have been reported with the use of botulinum toxin after a cross facial nerve grafting[15,24]. Although with less evidences, other rehabilitative approaches, such as physical therapy, biofeedback therapy, relaxation exercises with massages therapy, coordination and facial expression exercises, can reduce muscle stiffness, facilitating facial movements. Relatively to acupuncture and electrical nerve stimulation (in order to accelerate healing by stimulating muscle), there are still not enough data in the literature in order to

certify the real efficacy[15,24-27].

Regenerative therapy

In the recent years, innovative technologies are improving the possibilities for facial reanimation with bioelectrical interfaces by using tissue-engineered constructs. An emerging strategy in order to restore a symmetrical smile is a direct neural interface: Inputs of the interfaced nerves induce stimuluses in the injured facial nerve. Regenerative electrodes are used in case of traumatic injuries of the nerve: These could be implanted at the end of the facial nerve and could allow its regrowth through the construct[41].

Among regenerative therapy, peripheral nerve regenerative strategies are clinically not available yet. Experimental procedures described in the literature have shown different achievements and consist of a combination of stem cells and bio-scaffolds.

Different types of stem cells have been proposed in order restore neuronal integrity; among these, embryonic stem cells, nerve and mesenchymal stem cells, adipose and bone marrow derived stem cells and also other types have been proposed[42].

Bio-scaffolds aim to maintain cell feasibility, but should also sustain proliferation and allow intercellular communication and cellular growth. Carbon nanotubes, hyaluronic acid-based scaffolds, polymeric scaffolds and other similar solutions have been proposed with the aim of piloting the neuronal/assonal regrowth[43].

Nonetheless, this therapeutic strategy is indeed complex; if it will become available, hopefully, it could offer new potential approaches for future treatments.

CONCLUSION

Pediatric facial nerve palsy is a condition with several implications, particularly when occurring in childhood. It causes significant concerns in doctors and in parents as well, mainly due to the functional and aesthetic outcomes.

The causes of paralysis in children are many, however idiopathic facial paralysis, or Bell's palsy, is the most frequent form of facial paralysis in children too. A careful diagnostic workout and differential diagnosis are always recommended, in order to establish the most appropriate treatment. Hopefully, in the future, regenerative medicine could offer new options for the treatment of this condition.

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