Facial nerve paralysis in children

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

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

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

Received: May 20, 2015
Peer-review started: May 21, 2015
First decision: August 16, 2015
Revised: September 26, 2015
Accepted: October 12, 2015
Article in press: October 13, 2015
Published online: December 16, 2015

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,
**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) (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. 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.

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.

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. 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. 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. 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.

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.

Acquired facial paralysis can frequently be due to viral infections. The reactivation of Herpes Varicella-Zoster virus is usually associated to other cranial nerves paralysis; “Bell’s palsy”; “children”; “seventh nerve”; “therapy.”

**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.”
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 an 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[9,13].

Facial nerve palsy may also be present as a complication of several diseases such as acute and chronic otitis media, cholesteatoma, mastoiditis and meningitis[10,17].

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[9,9].

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[1,5,20].

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[9,14,18-21].

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[20].

The audiological evaluation is important in order to assess the presence of stapedial reflexes (topodiagnosis) 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)[15,9,12,21].

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
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 otorhinolaryngologists, 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\(^{[9,13,36]}\).

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 neurorhaphy 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...
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 emi facial 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

---

**Table 2 Therapeutic approaches to facial nerve palsy in childhood**

| Therapy of facial nerve palsy | Outcome¹ |
|------------------------------|----------|
| **Drugs**                    |          |
| Bell’s palsy                 | 70% recovery after 3 wk [23] |
| Oral steroids within 3 d of onset |          |
| 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 [36,40] |
| Anti-viral agents            |          |
| Other conditions            |          |
| **Protective measures**     |          |
| Targeted therapies for specific diseases | N/A |
| Eye protection              | N/A      |
| Artificial tears            | N/A      |
| Tarsorrhaphy                | N/A      |
| Eyelid weight implant       | N/A      |
| Nutritional support         | N/A      |
| **Surgery**                 |          |
| Traumatic palsy             | N/A      |
| 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 [36] |
| Gracilis muscle microvascular free flap | 89% recovery within 4-6 mo [41] |
| Sural nerve grafting        | N/A      |
| Cross-facial nerve grafting | 83% recovery within 1 yr [42] |
| **Rehabilitation approaches** | 100% recovery (temporary) [24] |
| Botulinum toxin             | N/A      |
| Physiotherapy               | N/A      |
| Biofeedback therapy         | N/A      |
| **Regenerative therapy**    |          |
| Bioelectrical interface/electrode | N/A |
| Stem cells and bio-scaffolds | N/A |

¹When available.
certify the real efficacy\textsuperscript{[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 stimuli 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\textsuperscript{[43]}.

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\textsuperscript{[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\textsuperscript{[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 workflow 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.

**REFERENCES**

1. Zandian A, Osiro S, Hudson R, Ali IM, Matusz P, Tubbs SR, Loukas M. The neurologist’s dilemma: a comprehensive clinical review of Bell’s palsy, with emphasis on current management trends. *Med Sci Monit* 2014; 20: 83-90 [PMID: 24441932 DOI: 10.12659/ MSM.889876]

2. Özkalı Y, Erol I, Saygın S, Yılmaz İ. Overview of pediatric peripheral facial nerve paralysis: analysis of 40 patients. *J Child Neurol* 2015; 30: 193-199 [PMID: 24810082 DOI: 1177/0883073814530497]

3. Barr JS, Katz KA, Hazen A. Surgical management of facial nerve paralysis in the pediatric population. *J Pediatr Surg* 2011; 46: 2168-2176 [PMID: 22075352 DOI: 10.1016/j.jpedsurg.2011.06.036]

4. Lunan R, Nagarajan L. Bell’s palsy: a guideline proposal following a review of practice. *J Paediatr Child Health* 2008; 44: 219-220 [PMID: 18469672 DOI: 10.1111/j.1440-1754.2007.01245.x]

5. Tiemstra JD, Khathate N. Bell’s palsy: diagnosis and management. *Am Fam Physician* 2007; 76: 997-1002 [PMID: 17956069]

6. Stew B, Williams H. Modern management of facial palsy: a review of current literature. *Br J Gen Pract* 2013; 63: 109-110 [PMID: 23561689 DOI: 10.3399/bjgp13X663262]

7. Shargorodsky J, Lin HW, Gopen Q. Facial nerve palsy in the pediatric population. *Clin Pediatr* (Phila) 2010; 49: 411-417 [PMID: 20394127 DOI: 10.1177/0009922809377978]

8. Al Tawil K, Saleem N, Kadri H, Rifae MT, Tawakol H. Traumatic facial nerve palsy in newborns: is it always iatrogenic? *Am J Perinatol* 2010; 27: 711-713 [PMID: 20387190 DOI: 10.1055/ s-0030-1253097]

9. Pavlou E, Gkampeta A, Arampatzi M. Facial nerve palsy in childhood. *Brain Dev* 2011; 33: 644-650 [PMID: 21144684 DOI: 10.1016/j.braindev.2010.11.001]

10. Morales-Chávez M, Ortíz-Rincónes MA, Suárez-Gorrín F. Surgical techniques for smile restoration in patients with Möbius syndrome. *J Clin Exp Dent* 2013; 5: e203-e207 [PMID: 24455082 DOI: 10.4317/ jced.51116]

11. Berker N, Acaroğlu G, Soykan E. Goldenhar’s Syndrome (oculo-auriculo-vertebral dysplasia) with congenital facial nerve palsy. *Yonsei Med J* 2004; 45: 157-160 [PMID: 15004885]

12. Pilon A, Rhee P, Newman T, Messner L. Bilateral abducens palsies and facial weakness as initial manifestations of a Chiari I malformation. *Optom Vis Sci* 2007; 84: 936-940 [PMID: 18049357]

13. Clark GD, Nordli DR, Dashe JF. Facial nerve palsy in children. Wolters Kluwer UpToDate®. 2014. Available from: URL: http://www.uptodate.com/contents/facial-nerve-palsy-in-children

14. Derin S, Derin H, Saham M, Caksen H. A pediatric case of Ramsay Hunt syndrome. *Case Rep Otolaryngol* 2014; 2014: 469565 [PMID: 25276457 DOI: 10.1155/2014/469565]

15. Finstuter J. Management of peripheral facial nerve palsy. *Eur Arch Otorhinolaryngol* 2008; 265: 743-752 [PMID: 18368417 DOI: 10.1007/s00405-008-0646-4]

16. Di Martino E, Sellhaus B, Haensel J, Schlegel JG, Westhofen M, Prescher A. Fallopian canal dehiscences: a survey of clinical and anatomical findings. *Eur Arch Otorhinolaryngol* 2005; 262: 120-126 [PMID: 15592859 DOI: 10.1007/s00405-004-0867-0]

17. Yamazaki K, Sato H, Murai K, Ogawa K. Infantile congenital petrosal cholestastoma: a case report and literature review. *Int J Pediatr Otorhinolaryngol* 2005; 69: 1703-1707 [PMID: 15979163 DOI: 10.1016/j.ijporl.2005.04.027]

18. Zielinski R, Kobos J, Zakrzewska A. Parotid gland tumors in children - pre- and postoperative diagnostic difficulties. *Pol J Pathol* 2014; 65: 130-134 [PMID: 25119173 DOI: 10.1141/pjp.2014.43963]

19. Kang HM, Kim MG, Hong SM, Lee HY, Kim TH, Yeo SG. Comparison of temporal bone fractures in children and adults. *Acta Otolaryngol* 2013; 133: 469-474 [PMID: 23294200 DOI: 10.3109/00017529.2012.754995]

20. Tsai HS, Chang LY, Lu CY, Lee PI, Chen JM, Lee CY, Huang LM. Epidemiology and treatment of Bell’s palsy in children in northern Taiwan. *J Microbiol Immunol Infect* 2009; 42: 351-356 [PMID: 19949760]

21. Dushyant B. Management of facial nerve palsy in newborn period. UK: Nottingham Neonatal Service-Clinical Guidelines (No. F16), 2013

22. Mitre EI, Lazarini PR, Dolci JE. Objective method for facial motricity grading in healthy individuals and in patients with unilateral peripheral facial palsy. *Am J Otolaryngol* 2008; 29: 51-57 [PMID: 18061833]

23. Biebl A, Luchner E, Hroncek K, Preisinger A, Eisenköbl A, Schmitt K, Forthner D. Facial nerve paralysis in children: is it as benign as supposed? *Pediatr Neurol* 2013; 49: 178-181 [PMID: 23831251 DOI: 10.1016/j.pediatrneurol.2013.03.013]

24. Terzis JK. Karypidis D. Therapeutic strategies in post-facial paralysis
Facial palsy in children

Synkinesis in pediatric patients. J Plast Reconstr Aesthet Surg 2012; 65: 1009-1018 [PMID: 22483723 DOI: 10.1016/j.bjps.2012.03.026]

Pourmomeny AA, Asadi S. Management of synkinesis and asymmetry in facial nerve palsy: a review article. Iran J Otorhinolaryngol 2014; 26: 251-256 [PMID: 25320703]

McCaul JA, Cascarini L, Godden D, Coombes D, Brennan PA, Kerawala CJ. Evidence based management of Bell’s palsy. Br J Oral Maxillofac Surg 2014; 52: 387-391 [PMID: 24685475 DOI: 10.1016/j.bjoms.2014.03.001]

de Almeida JR, Guyatt GH, Sud S, Dorion J, Hill MD, Kolber MR, Lea J, Reg SL, Somogyi BK, Westerberg BD, White C, Chen JM. Management of Bell palsy: clinical practice guideline. CMAJ 2014; 186: 917-922 [PMID: 24934895 DOI: 10.1503/cmaj.131801]

Shahidullah M, Haque A, Islam MR, Rizvi AN, Sultana N, Mia BA, Hussain MA. Comparative study between combination of famciclovir and prednisolone with prednisolone alone in acute Bell’s palsy. Mymsningsh Med J 2011; 20: 605-613 [PMID: 22081178]

Goudakos JK, Markou KD. Corticosteroids vs corticosteroids plus antiviral agents in the treatment of Bell palsy: a systematic review and meta-analysis. Arch Otolaryngol Head Neck Surg 2009; 135: 558-564 [PMID: 19526803 DOI: 10.1001/archoto.2009.44]

Numthavaj P, Thakkinstian A, Dejthevaporn C, Attia J. Corticosteroid and antiviral therapy for Bell’s palsy: a network meta-analysis. BMC Neurol 2011; 11: 1 [PMID: 21208452 DOI: 10.1186/1471-2377-11-1]

Pitaro J, Waissbluth S, Daniel SJ. Do children with Bell’s palsy benefit from steroid treatment? A systematic review. Int J Pediatr Otorhinolaryngol 2012; 76: 921-926 [PMID: 22530409 DOI: 10.1016/j.ijpedit.2012.02.044]

Chen WX, Wong V. Prognosis of Bell’s palsy in children—analysis of 29 cases. Brain Dev 2005; 27: 504-508 [PMID: 16198208]

Wang CH, Chang YC, Shih HM, Chen CY, Chen JC. Facial palsy in children: emergency department management and outcome. Pediatr Emerg Care 2010; 26: 121-125 [PMID: 20093994 DOI: 10.1097/PEC.0b013e3181d01840]

Duval M, Daniel SJ. Facial nerve palsy in neonates secondary to forces use. Arch Otolaryngol Head Neck Surg 2009; 135: 634-636 [PMID: 19620581 DOI: 10.1001/archoto.2009.69]

Shih WH, Tseng FY, Yeh TH, Hsu CJ, Chen YS. Outcomes of facial palsy in children. Acta Otolaryngol 2009; 129: 915-920 [PMID: 18923943 DOI: 10.1080/00016480802468179]

Kansu L, Yilmaz I. Herpes zoster oticus (Ramsay Hunt syndrome) in children: case report and literature review. Int J Pediatr Otorhinolaryngol 2012; 76: 772-776 [PMID: 22445801 DOI: 10.1016/j.jpedit.2012.03.003]

Banks CA, Hadlock TA. Pediatric facial nerve rehabilitation. Facial Plast Surg Clin North Am 2014; 22: 487-502 [PMID: 25444723 DOI: 10.1016/j.fsc.2014.07.006]

Leboulanger N, Maldent JB, Glynn F, Charrier JB, Monteil JP, Garabedian EN. Rehabilitation of congenital facial palsy with temporalis flap—case series and literature review. Int J Pediatr Otorhinolaryngol 2012; 76: 1205-1210 [PMID: 22658449 DOI: 10.1016/j.ijpedit.2012.05.007]

Terzis JK, Karypidis D. The outcomes of dynamic procedures for blink restoration in pediatric facial paralysis. Plast Reconstr Surg 2010; 125: 629-644 [PMID: 20124848 DOI: 10.1097/PRS.0b013e3181e91899]

Petersson RS, Sampson DE, Sidman JD. Dynamic facial reanimation with orthodromic temporalis tendon transfer in children. JAMA Facial Plast Surg 2014; 16: 432-436 [PMID: 25255818 DOI: 10.1001/jamafacial.2014.651]

Laughals NB, Urbanchek MG, Ray A, Brenner MJ. Update in facial nerve paralysis: tissue engineering and new technologies. Curr Opin Otolaryngol Head Neck Surg 2014; 22: 291-299 [PMID: 24979369 DOI: 10.1097/MOO.0000000000000062]

Fairbairn NG, Meppelinck AM, Ng-Glazier J, Randolph MA, Winograd JM. Augmenting peripheral nerve regeneration using stem cells: A review of current opinion. World J Stem Cells 2015; 7: 11-26 [PMID: 25621102 DOI: 10.4252/wjsc.v7.i1.11]

Ricks CB, Shin SS, Becker C, Grandhi R. Extracellular matrices, artificial neural scaffolds and the promise of neural regeneration. Neural Regen Res 2014; 9: 1573-1577 [PMID: 25368641 DOI: 10.4103/1673-5374.141778]

Volk GF, Klingner C, Finkensieper M, Witte OW, Guntinas-Lichius O. Prognostication of recovery time after acute peripheral facial palsy: a prospective cohort study. BMJ Open 2013; 3: pii: e003007 [PMID: 23794548 DOI: 10.1136/bmjopen-2013-003007]

Hadlock TA, Malo JS, Cheney ML, Henstrom DK. Free gracilis transfer for smile in children: the Massachusetts Eye and Ear Infirmary Experience in excursion and quality-of-life changes. Arch Facial Plast Surg 2011; 13: 190-194 [PMID: 21576665 DOI: 10.1016/j.archfacial.2011.29]

Malik S, Bhandekar HS, Korday CS. Traumatic peripheral neuroapraxia in neonates: a case series. J Clin Diagn Res 2014; 8: PD10-PD12 [PMID: 25478423 DOI: 10.7860/JCDR/2014/9205.5059]
