Facial Nerve Palsy

Etiology and Approach to Diagnosis and Treatment

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Abstract: Facial nerve palsy has a broad differential diagnosis and possible psychological and anatomical consequences. A thorough investigation must be performed to determine the cause of the palsy and to direct treatment. If no cause can be found, therapy with prednisone with or without an antiviral medication can be considered and begun as early as possible after onset of symptoms. Resolution and time to recovery vary with etiology, but overall prognosis is good.

Key Words: facial nerve palsy, Lyme disease, congenital palsy, Bell palsy

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TARGET AUDIENCE

This review article targets health care providers who see children and adolescents in acute care settings.

LEARNING OBJECTIVES

After completing this CME activity readers should be better able to:

1. Evaluate the epidemiology of acquired facial nerve palsy in children and its most common etiologies.
2. Design an approach to diagnosis of the likely etiology of facial nerve palsy in a child based on historical and physical examination findings.
3. Select the most appropriate treatment of acquired facial nerve palsy in children based on etiology.

All cases of acquired facial nerve palsy in children must be thoroughly evaluated to rule out serious pathology such as infection, oncological disorders, trauma, or hemorrhage. In addition, because of the deficits caused by injury to the nerve, consequences can be psychologically and anatomically debilitating. Early recognition and initiation of appropriate treatment are therefore essential. After reading this article, readers should be better able to evaluate the epidemiology of acquired facial nerve palsy in the pediatric population, diagnose the etiology of facial nerve palsy in children based on historical and physical examination findings, and select the best treatment approach.

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EPIDEMIOLOGY

Acquired facial nerve palsy is an uncommon problem, affecting 20 to 32 per 100,000 people per year in the general population.[1,2] Its incidence in children is even lower, estimated at 2.7 per 100,000 per year in children younger 10 years and 10.1 per 100,000 per year in children 10 to 20 years old.[2] A bimodal distribution of infectious and traumatic facial nerve palsy occurs, peaking at 1 to 3 years and 8 to 12 years old.[3] Males and females are affected equally, as are both sides of the face. Recurrence rates range from 3% to 11%.[4,5]

ANATOMY OF CRANIAL NERVE VII

Cranial nerve (CN) VII, also termed the facial nerve, has both sensory and motor components. It is responsible for portions of taste and for control of the salivary and lacrimal glands, and it serves as the motor nerve for both the stapedius muscle in the middle ear and for the muscles of facial expression. It has the longest intraosseous course of any CN, making it especially susceptible to injury or infection. It emerges from the brainstem at the cerebellopontine angle, along the caudal edge of the lateral pons, near the cerebellum. Its course then carries it over the posterior cranial fossa, through the internal acoustic meatus, where it then forms the geniculate ganglion. Shortly after the formation of the geniculate ganglion, the greater petrosal nerve branches off to supply the lacrimal glands. The nerve then travels through the facial canal in the petrous portion of the temporal bone, bringing it in close proximity to the medial portion of the inner ear and the mastoid cavity.

In its course through the temporal bone, the facial nerve first supplies the stapedius muscle. Later, additional branches form the chorda tympani, which provides innervation to the submandibular and sublingual salivary glands and taste to the anterior two thirds of the tongue. The facial nerve exits the skull through the stylomastoid foramen, between the mastoid and styloid processes. Following its emergence from the skull, the facial nerve gives off the posterior auricular nerve to provide sensation to the periauricular area, then runs anteriorly through the parotid gland and forms 5 terminal branches supplying motor function to the face.[6]

Because of crossing of motor fibers at the level of the motor nucleus of the facial nerve in the brainstem, motor function is supplied to the upper portion of the face from both sides of the brain, whereas the lower half of the face is controlled by fibers supplied solely by the contralateral side. Therefore, an important clinical sign distinguishing a central from a peripheral process affecting the facial nerve is the preservation of function of the muscles of the forehead.[7] A central nervous system lesion resulting in a facial palsy will present with normal tone and movement of both sides of the forehead. A peripheral facial nerve palsy will result in abnormal motor tone and movement to the affected side of the forehead.

PRESENTATION OF FACIAL NERVE PALSY

The typical presentation of facial nerve palsy is a rapid onset of partial to complete paralysis of the facial muscles with

preservation of facial sensation, although some patients may report a feeling of "numbness." In a Finnish study, 29% of adult and pediatric patients with idiopathic facial nerve palsy had associated systemic symptoms including fever, headache, sore throat, or neck stiffness.[8] Cases of facial nerve palsy are typically unilateral, although bilateral cases can occur, most often due to Lyme disease.[9]

On physical examination, the patient will be unable to raise the eyebrow or tightly close the eyelid on the affected side. The nasolabial fold is typically absent, and the mouth may be drawn toward the unaffected side. Patients may drool from the affected side because of inability to keep the mouth closed. Crying infants will demonstrate inability to close the eyelid on the affected side and pulling of the angle of the mouth toward the unaffected side. As discussed above, preservation of use of forehead musculature indicates a central lesion and should prompt evaluation for an intracranial process.

Because of the efferent parasympathetic branches to the lacrimal and salivary glands and the motor branch to the stapedius, additional symptoms such as hyperacusis, decreased lacrimation, or decreased taste can help in localization of the lesion along the course of the facial nerve.

DIFFERENTIAL DIAGNOSIS OF FACIAL NERVE PALSY

The etiologies of facial nerve palsy in children can be congenital or acquired and can be further classified as infectious, traumatic, malignancy associated, hypertension associated, and idiopathic (Table 1). Causes will also vary dramatically by geographic area and by season of the year.

Congenital Facial Nerve Palsy

Birth Trauma

Congenital facial nerve palsy occurs in 2.1 per 1000 children.[10] It is typically associated with birth trauma. A history of forceps delivery, prolonged labor, and/or a birth weight greater than 3500 g are risk factors.

TABLE 1. Etiologies of Facial Nerve Palsy

- Congenital causes
  - Birth trauma
  - Genetic syndromes
    - Melkersson-Rosenthal syndrome
    - Albers-Schönberg disease (osteopetrosis)
    - Möbius syndrome
    - Goldenhar syndrome (oculoauriculovertebral dysplasia)
- Acquired causes
  - Infectious
    - Lyme disease
    - Herpes simplex virus
    - Varicella virus
    - Otitis media
    - Other (human herpesvirus type 6, mumps, coxsackie virus, adenovirus)
  - Traumatic
  - Malignancy associated
  - Hypertension associated
  - Idiopathic (Bell palsy)

Genetic Syndromes

Patients with genetic syndromes often exhibit other associated physical anomalies that distinguish their facial nerve palsies from those associated with birth trauma.[11]

Melkersson-Rosenthal Syndrome

Melkersson-Rosenthal syndrome is an inherited disorder that presents with recurrent attacks of intermittent facial nerve palsy and facial swelling, with the presence of a fissured tongue. Patients may demonstrate the complete triad, suffer the findings in a sequential pattern, or have only portions of the syndrome. Greene and Rogers[12] report that facial nerve palsy is present in 30% to 50% and can become permanent.

Albers-Schönberg Disease (Osteopetrosis)

Osteopetrosis is an inherited bone disorder that results in gradually increasing bone density. Bone growth can extend into the CN foramina, impinging on the nerve and resulting in facial nerve palsy. Often paralysis can be bilateral.[13]

Möbius Syndrome

Patients with Möbius syndrome demonstrate bilateral facial nerve palsy often associated with other CN palsies (often CN VI).

Goldenhar Syndrome (Oculoauriculovertebral Dysplasia)

Patients with Goldenhar syndrome evidence facial nerve palsy, malar and maxillary hypoplasia, and hemifacial microsomia. Seventy percent of patients exhibit unilateral, predominantly right-sided anomalies.[14]

Acquired Facial Nerve Palsy

Infectious Causes

Among studies of facial nerve palsy seen at children's hospitals in the United States, infectious causes account for more than one third of cases.[3,13] In addition, children with acquired facial nerve palsy often have a history of preceding viral infection.[5,15]

Lyme Disease

Lyme disease, attributed to the spirochete *Borrelia burgdorferi*, is a common cause of acquired facial nerve palsy in endemic areas, accounting for up to 50% of cases.[8,9,16] The palsy can occur either as part of the first stage of disease with the classic erythema chronicum migrans rash, or as part of a later stage as the disease advances. In a series of 8 patients with acute facial nerve palsy diagnosed with Lyme disease, including 2 children, patients also exhibited swelling and erythema of the affected side of the face.[17] Up to 80% of pediatric patients demonstrate pronounced systemic symptoms including fatigue, nausea, abdominal pain, arthralgia, and headache.[9,18]

In a retrospective study of 313 pediatric patients presenting with peripheral facial nerve palsy, Nigrovic et al[16] found that independent predictors of Lyme disease were onset of symptoms in peak Lyme disease season, absence of previous herpetic lesions, presence of fever, and history of headaches. Patients with at least 3 of these predictors had a greater than 50% risk of having Lyme disease. Of patients further evaluated by lumbar puncture who demonstrated cerebrospinal fluid (CSF) pleocytosis, 98% had proven Lyme disease. Even without evidence of meningitis, headache and onset of symptoms during peak Lyme season remained independent predictors of Lyme.

The facial nerve palsy associated with Lyme disease may develop as a result of direct neural invasion by the spirochete.[19] In studies examining the CSF of children with Lyme-attributed facial nerve palsy, Belman et al[20] found that 68% had abnormal

CSF findings on lumbar puncture: 55% demonstrated a lymphocytic pleocytosis; 45% showed elevated protein; and 33% had evidence of both pleocytosis and elevated protein. Additionally, 82% had free *B. burgdorferi* antibodies present in the CSF.

Herpes Simplex Virus (HSV)

Recent studies of endoneurial fluid and saliva in patients with idiopathic facial nerve palsy in pediatric emergency departments in the northeastern United States, including those with negative Lyme titers, have demonstrated HSV in 79% of patients.[21,22] It is believed the virus leads to inflammation and demyelination, creating the peripheral palsy.

Varicella Zoster Virus (VZV)

Acute peripheral facial nerve palsy with documented lesions of the auricle or oral epithelium is known as Ramsay Hunt syndrome. In a Finnish study, more than one third of cases of facial nerve palsy in children were attributed to VZV.[8] A study of pediatric patients in Japan found Ramsay Hunt syndrome to be more prevalent among children 6 to 15 years old than in those younger than 5 years (53% vs 9%).[23] Patients may present without the classic lesions (known as zoster sine herpete), and VZV in such cases is found only through viral testing of serum or saliva.

Because varicella virus latency occurs in sensory rather than motor nerves, it is postulated that during a primary varicella infection, the virus may enter sensory branches of the facial nerve and travel to the sensory fibers of the geniculate ganglion to establish latency. Upon reactivation, the virus travels back along the sensory fibers, at the same time causing inflammation to the adjacent motor fibers of CN VII, thus leading to facial nerve palsy.

Otitis Media (OM)

Before the common use of antibiotics, OM was responsible for a greater proportion of facial nerve palsies. In the modern era, only 0.005% of patients with OM develop facial nerve palsy, as compared with 0.7% previously.[24] The etiology of facial nerve palsy associated with OM is unclear, but several hypotheses exist. In its early stages, OM may cause retrograde infection within the facial nerve canal or tympanic cavity ascending the chorda tympani, causing reactivation of latent viral infection. Another theory postulates that peripheral demyelination of the facial nerve occurs due to the presence of inflammatory bacterial toxins. In its later stages, if the inflammatory process of OM spreads beyond the tympanic cavity and mastoid and into the facial nerve canal (fallopian canal), it can then lead to accompanying inflammation or compression of the facial nerve. Finally, in chronic OM, a polyp or a cholesteatoma may cause erosion into the fallopian canal with direct extension of infection.[25]

Other

Other viruses have been associated with acquired facial nerve palsy, including human herpesvirus 6 and the mumps virus. Paun et al[26] found that in patients younger than 18 years admitted to a Romanian hospital with idiopathic facial nerve palsy, 24 (83%) of 29 had evidence of an active upper respiratory infection, and 18 (62%) of 29 had positive viral antigens on indirect immunofluorescence of nasopharyngeal exudate, with coxsackie and adenovirus being the most frequently detected.

Traumatic

Trauma to the face, temporal area, or skull can produce a temporary facial nerve palsy. Approximately 20% of cases of peripheral facial nerve palsy are attributed to trauma. In cases of blunt trauma, including that occurring in children, complete recovery is typical.[3,27]

Other Causes

Malignancy

In a retrospective study by Grundfast et al,[13] 12% of patients admitted to a children's hospital in 1990 with idiopathic facial nerve palsy were found to have malignancies, including leukemia, astrocytoma, and rhabdomyosarcoma. Clinical features such as gradual progression of paralysis lasting greater than 3 weeks, no return of function in 6 months, ipsilateral recurrence, hemifacial spasm, other associated cranial neuropathies, and pain and single-branch involvement should raise suspicion of a neoplastic etiology.

Hypertension

In a retrospective review of cases of noncongenital pediatric facial nerve palsy seen over a 10-year period at a children's hospital in London, 7 (8%) of 87 patients had a palsy attributable to hypertension. Among 3 of 35 patients treated for hypertension in this period, facial nerve palsy was the presenting symptom. The paralysis coincided with a rise in blood pressure and improved with control of the hypertension. All cases evidenced unilateral peripheral (ie, extracranial) facial nerve palsy.[28]

The pathology of the hypertension-associated facial nerve palsy is believed to be secondary to hemorrhage into the facial nerve canal, as evidenced on autopsy. Additionally, vascular lesions at any site along the course of the facial nerve can result in pressure on the nerve, causing paralysis.[28]

Idiopathic (Bell) Palsy

First described in the early 19th century by Sir Charles Bell, Bell palsy is an idiopathic palsy of the facial nerve, and as such is a diagnosis of exclusion. Prior studies have found Bell palsy to be responsible for up to 50% of cases of facial nerve palsy in children.[29,30] As diagnostic testing has improved, however, many cases once considered Bell palsy have now been found to have an infectious cause. Recent studies of pediatric patients with facial nerve palsy list Bell palsy as the diagnosis in 9% to 16% of cases of facial nerve palsy.[3,13]

DIAGNOSIS OF FACIAL NERVE PALSY

History and Physical Examination

A thorough history and physical examination with a focus on the otolaryngological and neurological examinations should be completed on all patients with a congenital or acquired facial nerve palsy. An algorithm for assisting in determining the possible etiology of the lesion is provided (Fig. 1).

If the palsy was noted at or shortly after birth, questions should be asked regarding a history of prolonged labor, forceps use, or facial and periauricular ecchymoses at delivery or shortly thereafter.[31] Beyond the neonatal period, historical clues such as age at onset, prior occurrences, recent illness and systemic symptoms (particularly headache or rash), and travel to areas endemic for Lyme should be elicited.

The patient should be examined for other stigmata of syndromes including craniofacial dysmorphism or multisystem pathology. If forehead musculature is spared, a central lesion is likely, and the patient requires immediate evaluation for stroke or other central processes.[32] A thorough neurological examination should be performed, and any abnormalities in addition to the facial palsy should lead to imaging studies of the brain.[33] The patient should be evaluated for decreased or absent tearing on the affected side. If developmentally appropriate, differences in taste should be assessed. Degree of deficit in the facial nerve can be scored using the House-Brackmann scoring system,

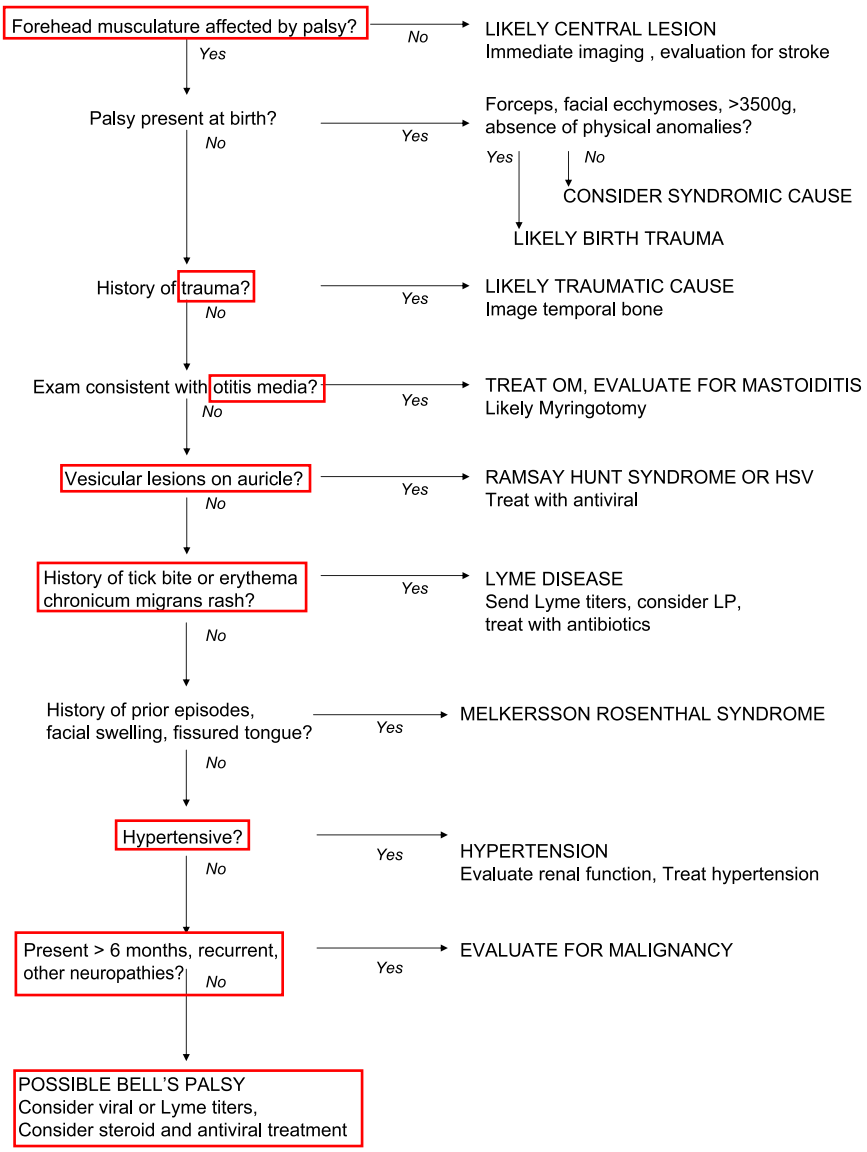

FIGURE 1. Algorithm for aid in diagnosing etiology of facial nerve palsy in children.

where 1 is normal, 2 is mild, 3 to 4 is moderate, 5 is severe, and 6 is total loss of function.[34]

Laboratory Testing

Laboratory testing should be directed based on history and physical examination findings. In general, specific titers should be sent to confirm specific diagnoses that have specific therapies (Lyme disease, for example). If the patient resides or has visited a Lyme disease–endemic area but has no evidence or history of rash, Lyme titers should still be strongly considered. The Centers for Disease Control defines Lyme infection as either evidence of erythema chronicum migrans rash or serological evidence of Lyme through enzyme-linked immunosorbent assay testing confirmed by Western blot. Nigrovic et al[16] suggest that children presenting with facial nerve palsy and more than 1 predictor for Lyme disease facial nerve palsy should have a Lyme titer sent.

In patients presenting with acquired facial nerve palsy, consideration is often given to performing a lumbar puncture to obtain CSF. Studies and recommendations regarding the utility of this test in cases of facial nerve palsy are controversial. In a study examining the CSF of 12 patients with idiopathic facial nerve palsy, 7 children and 1 adult had elevated mononuclear cells in their CSF (>5). All children showed an increased CSF/albumin ratio, indicating disruption of the blood-brain barrier. Routine blood studies were normal in all patients. Clinically, 9 of 12 patients had a history suggestive of viral infection, and only 1 child had signs and symptoms of meningitis. Based on these data, Sandstedt and colleagues[35] recommend lumbar puncture in all patients even without signs of meningitis. In contrast, Eshel et al[36] found that, in a retrospective review of all acquired facial nerve palsies in a 9-year period, only 2 of 28 children had abnormal CSF findings. Both patients were febrile and with leukocytosis and OM on examination.

Albisetti et al[37] showed that 25% of children with facial nerve palsy and lymphocytic pleocytosis produced intrathecal antibodies to *B. burgdorferi* within 4 to 6 days of neurological symptoms. They concluded that, in patients with acute facial nerve palsy due to neuroborreliosis, analysis of CSF for intrathecal antibody production can help to establish rapid diagnosis, thus allowing for prompt treatment.[37]

Recommendations made by the American Academy of Pediatrics in the *Red Book* indicate that lumbar puncture is not indicated for Lyme-associated facial nerve palsy without clinical signs of meningitis.[38]

Imaging

Imaging of the facial nerve for acquired facial nerve palsy, excluding trauma, is recommended only for atypical Bell palsy with peripheral palsy lasting greater than 2 months, or for recurrent or progressive palsies.[39] In congenital facial nerve palsy, temporal bone computed tomography should be used to assess the facial canal and to rule out middle- or inner-ear anomalies in conjunction with absence or dysplasia of the facial nerve. Magnetic resonance imaging can then be used to provide complementary information.[31]

When the facial nerve is imaged, the study must include images of the brain and pons, the cerebellopontine angle, the internal auditory canal, all 3 portions of the facial canal, the geniculate ganglion, the stylomastoid foramen, and the parotid gland.[39]

Among patients with facial nerve palsy undergoing gadolinium-enhanced magnetic resonance imaging evaluation, 43% of pediatric patients show facial nerve enhancement. In those without enhancement, mean time to recovery was significantly shorter (9.5 vs 19.3 weeks).[40]

Referral and Further Testing

Outpatient referrals to neurology, audiology, otolaryngology, and/or ophthalmology may be appropriate based on patient age, etiology, and presentation of disease. Further testing by consultants may be indicated. Audiological testing, for example, may be useful in localizing the lesion along the facial nerve. More urgent consultations may be necessary if the CN VII palsy does not appear peripheral or if other neurological signs or symptoms are present.

Electroneuronography assesses the extracranial portion of the facial nerve by applying a brief electrical stimulus to it transdermally near the stylomastoid foramen, with electrodes located at distal facial musculature sites, often nasolabial fold or perioral muscles. The compound action potential elicited is then measured. Testing can predict prognosis of recovery. In a survey of more than 1000 patients in the Netherlands including both adults and children, when electroneuronography was performed and excitability was normal, 80% of patients recover completely. When a slight paresis is present, only 25% completely recover function. When excitability is absent, almost no patients return to normal.[4]

TREATMENT OF FACIAL NERVE PALSY

Medical

In all cases of facial nerve palsy, prophylaxis of ophthalmologic complications should be initiated. To avoid exposure keratitis or corneal damage in cases with extensive paralysis and incomplete closure of the eye, ocular lubricants or artificial tears should be used during daytime hours. Patching or taping of the affected eye is discouraged because of the possibility of eyelid opening beneath the patch, thus coming into contact with the eye and causing corneal damage.[41]

In cases where the infectious etiology of facial nerve palsy is known or strongly suspected (OM, HSV, VZV, Lyme), antibiotic or antiviral therapy directed against the underlying cause should be initiated immediately. Even without associated signs or symptoms, if the patient is from or has visited a Lyme-endemic area (especially during peak season for Lyme), empiric oral treatment for *Borrelia* pending the results of titers should be strongly considered.[16] Based on recommendations by the American Academy of Pediatrics' *Red Book*, treatment should consist of oral doxycycline or amoxicillin (for patients <8 years old) for 21 to 28 days.[38]

Treatment of idiopathic facial nerve palsy, however, is controversial. In 2 prospective placebo-controlled randomized clinical trials, Unuvar et al[42] and Wolf et al,[43] oral corticosteroids for the treatment of idiopathic facial nerve palsy, no statistically significant difference was shown in recovery time or residual weakness. In contrast, another randomized double-blind controlled study using placebo versus prednisone for idiopathic facial nerve palsy in adults showed no significant difference in time to recovery between treatment and placebo, but grade of paralysis at recovery was significantly better in the prednisone group.[44] Based on these data, the authors recommend that patients with House-Brackmann grade V or VI at onset receive prednisone to decrease their chance of developing denervation and subsequent hemifacial spasm. Additionally, Ramsey et al[45] conducted a meta-analysis of 47 prospective controlled trials using early initiation of corticosteroid treatment for complete idiopathic facial nerve palsy. The 2 trials meeting inclusion criteria in the analysis indicated that oral steroid treatment significantly improves complete facial motor recovery (difference, 17%).[45]

Because of the high incidence of viral infection that is seen with facial nerve palsy (especially HSV and VZV), antiviral treatment in combination with oral steroids has been considered as a possible treatment. In a recent randomized placebo-controlled trial in Scandinavian adults comparing placebo to prednisone with or without an antiviral medication, a 40% shorter time to recovery was seen among patients taking oral steroids. There was no difference seen between patients who received antiviral medication and those who did not, regardless of concurrent treatment with steroids.[46]

In other trials including both adults and children comparing combination treatment of acyclovir and prednisone to prednisone alone for the treatment of idiopathic facial nerve palsy, patients treated with a combination of acyclovir and prednisone had significantly better outcomes compared with those treated with prednisone alone or with prednisone and placebo.[47,48] All patients were begun on treatment within 3 days of onset of symptoms. Finally, 3 meta-analyses of randomized controlled trials examining the effect of steroids versus steroids plus antiviral medication on idiopathic facial nerve palsy have been published recently. Two of the analyses indicate no increased benefit from the addition of an antiviral medication.[49,50] The third meta-analysis by de Almeida et al[51] indicates a 12% reduced risk of unsatisfactory recovery with the addition of an antiviral agent to a corticosteroid regimen.

No conclusive recommendations can be made based on these data, particularly for pediatric patients as many of the randomized controlled trials used in the meta-analyses did not include them. Although the prognosis for full recovery in Bell palsy in children is excellent, and most studies do not support the empiric use of acyclovir, some individuals may choose to administer this agent. Patients with Ramsay-Hunt syndrome (with viral ulcers in the external ear canal) or active herpetic lesions on the face or in the oral cavity should receive antiviral treatment.

Surgical

In patients with facial nerve palsy secondary to OM, myringotomy with or without tube insertion[25] should be performed. If mastoiditis is suspected, mastoidectomy is indicated. Surgical treatment of facial nerve palsy should also be considered in cases of trauma or congenital palsy. Several techniques exist using both static and reanimation techniques, using muscle flaps,[31] but are beyond the scope of this review.

PROGNOSIS OF FACIAL NERVE PALSY

Prognosis and median time to recovery from facial nerve palsy vary based on the etiology of the disease. Congenital traumatic facial nerve palsy has been shown to resolve spontaneously in 90% of patients within 4 weeks.[11] Evidence shows 80% to 90% recovery rates in adult and pediatric patients with blunt temporal trauma,[27] although time to recovery was longer than in patients with an infectious etiology (10 vs <1 month).[3]

Recovery rates of idiopathic facial nerve palsy in children range from 70% to 90%.[5,15,33] In a 2-year follow-up of facial nerve palsy in children, Skogman et al[52] found a 78% recovery rate. There was no correlation between sequelae and age, sex, presence or absence of Lyme disease, or treatment.

SUMMARY

Facial nerve palsy has a broad differential diagnosis and possible psychological and anatomical consequences. A thorough investigation must be performed to determine the cause of the palsy and to direct treatment. If no cause can be found, therapy with prednisone with or without an antiviral medication can be considered and begun as early as possible after onset of symptoms. Resolution and time to recovery vary with etiology, but overall prognosis is good. After reading this article, readers should be better able to diagnose the etiology of facial nerve palsy in children based on historical and physical examination findings, and select the best treatment approach.

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CME EXAMINATION
OCTOBER 2010

Please mark your answers on the ANSWER SHEET.

Facial Nerve Palsy, *Lorch and Teach*.

1. Which of the following should be the first step in the evaluation of a patient with facial nerve palsy sparing the forehead musculature?
   A. Lumbar puncture
   B. Neuroimaging
   C. Lyme titers
   D. Complete blood count
   E. Otologic examination

2. In a Lyme-endemic region, facial nerve palsy associated with the presence of which of the following indicates a high (>50%) risk of having Lyme disease?
   A. Recurrent facial nerve palsy, fatigue, arthralgia
   B. Bilateral facial nerve palsy, facial swelling, facial erythema
   C. Absence of vesicular lesions, vomiting
   D. Facial nerve palsy occurring during peak Lyme disease season, absence of previous herpetic lesions, history of headaches
   E. Bilateral facial nerve palsy, cerebrospinal fluid pleocytosis, arthralgia

3. In a patient with an acquired idiopathic facial nerve palsy of House-Brackmann grade V, prompt treatment should be considered using which of the following?
   A. Prednisone and cefuroxime
   B. Prednisone and surgical decompression
   C. Observation only
   D. Prednisone and antivirals
   E. Surgical decompression alone

4. A recurrent ipsilateral acquired painful facial nerve palsy lasting greater than 3 weeks should prompt evaluation for which of the following?
   A. Malignancy
   B. Melkersson-Rosenthal syndrome
   C. Lyme disease
   D. Hypertension
   E. Herpes simplex virus

5. An 8-year-old with an acquired idiopathic facial nerve palsy has electroneuronography performed. Test results indicate normal excitability. Parents can be told which of the following?
   A. No recovery is likely.
   B. There is a low likelihood of recovery.
   C. One quarter of patients with these results will evidence complete recovery.
   D. The patient will definitely recover completely.
   E. More than 3 quarters of patients with these results will evidence complete recovery

ANSWER SHEET FOR THE *PEDIATRIC EMERGENCY CARE* CME PROGRAM EXAM October 2010

Please answer the questions on page 770 by filling in the appropriate circles on the answer sheet below. Please mark the one best answer and fill in the circle until the letter is no longer visible. To process your exam, you must also provide the following information:

Name (please print): _____

Street Address _____

City/State/Zip _____

Daytime Phone _____

Specialty _____

1. (A) (B) (C) (D) (E)
2. (A) (B) (C) (D) (E)
3. (A) (B) (C) (D) (E)
4. (A) (B) (C) (D) (E)
5. (A) (B) (C) (D) (E)

Your evaluation of this CME activity will help guide future planning. Please respond to the following questions below.

Please rate these activities (1 – minimally, 5 – completely)	1	2	3	4	5
These activities were effective in meeting the educational objectives	○	○	○	○	○
These activities were appropriately evidence-based	○	○	○	○	○
These activities were relevant to my practice	○	○	○	○	○

How many of your patients are likely to be impacted by what you learned from these activities?
○ <20% ○ 20%–40% ○ 40%–60% ○ 60%–80% ○ >80%

Do you expect that these activities will help you improve your skill or judgment within the next 6 months? (1 – definitely will not change, 5 – definitely will change)	1	2	3	4	5
	○	○	○	○	○

How will you apply what you learned from these activities (mark all that apply):

In diagnosing patients ○
In monitoring patients ○
In educating students and colleagues ○
As part of a quality or peformance improvement project ○
For maintenance of board certification ○
In making treatment decisions ○
As a foundation to learn more ○
In educating patients and their caregivers ○
To confirm current practice ○
For maintenance of licensure ○

How committed are you to applying these activities to your practice in the ways you indicated above? (1 – minimally, 5 – completely)	1	2	3	4	5
	○	○	○	○	○

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CME EXAM ANSWERS

Answers for the Pediatric Emergency Care CME Program Exam

Below you will find the answers to the examination covering the review article in the July 2010 issue. All participants whose examinations were received by September 15, 2010 and who achieved a score of 80% or greater will receive a certificate from Lippincott CME Institute, Inc.

EXAM ANSWERS
July 2010

1. A
2. E
3. C
4. C
5. E

TABLE 2. *Regional House-Brackmann facial nerve grading system*

	Grade	Description
Forehead	1	Normal forehead movement
	2	Slight weakness in forehead movement
	3	Obvious but not disfiguring asymmetry with motion, symmetric at rest
	4	Obvious weakness of disfiguring asymmetry with motion, symmetric at rest
	5	Barely perceptible motion in forehead, asymmetric at rest
	6	No movement
Eye	1	Normal eye closure
	2	Mild weakness in eye closure
	3	Obvious weakness but able to close eyes
	4	Unable to close eye with maximal effort
	5	Barely perceptible eyelid movement
	6	No movement
Midface	1	Normal midface movement
	2	Slight weakness in midface movement
	3	Obvious but not disfiguring weakness, symmetric at rest
	4	Obvious weakness and disfiguring asymmetry with motion, symmetric at rest
	5	Barely perceptible motion in midface, asymmetric at rest
	6	No movement
Mouth	1	Normal corner of mouth movement
	2	Slight weakness of corner of mouth movement
	3	Obvious but not disfiguring weakness, symmetric at rest
	4	Obvious weakness and disfiguring asymmetry with motion, symmetric at rest
	5	Barely perceptible corner of mouth movement, asymmetric at rest
	6	No movement
Synkinesis	None	None
	Mild	Obvious but not disfiguring
	Severe	Disfiguring or interferes with function

TABLE 1. *House-Brackmann facial nerve grading system*

	Grade	Defined by
1	Normal	Normal facial function in all areas.
2	Mild dysfunction	Slight weakness noticeable only on close inspection. At rest: normal symmetry of forehead, ability to close eye with minimal effort and slight asymmetry, ability to move corners of mouth with maximal effort and slight asymmetry. No synkinesis, contracture, or hemifacial spasm.
3	Moderate dysfunction	Obvious but not disfiguring difference between two sides, no functional impairment; noticeable but not severe synkinesis, contracture, and/or hemifacial spasm. At rest: normal symmetry and tone. Motion: slight to no movement of forehead, ability to close eye with maximal effort and obvious asymmetry, ability to move corners of mouth with maximal effort and obvious asymmetry. Patients who have obvious but not disfiguring synkinesis, contracture, and/or hemifacial spasm are grade III regardless of degree of motor activity.
4	Moderately severe dysfunction	Obvious weakness and/or disfiguring asymmetry. At rest: normal symmetry and tone. Motion: no movement of forehead; inability to close eye completely with maximal effort. Patients with synkinesis, mass action, and/or hemifacial spasm severe enough to interfere with function are grade IV regardless of motor activity.
5	Severe dysfunction	Only barely perceptible motion. At rest: possible asymmetry with droop of corner of mouth and decreased or absence of nasal labial fold. Motion: no movement of forehead, incomplete closure of eye and only slight movement of lid with maximal effort, slight movement of corner of mouth. Synkinesis, contracture, and hemifacial spasm usually absent.
6	Total paralysis	Loss of tone; asymmetry; no motion; no synkinesis, contracture, or hemifacial spasm.