IJCNP, Volume 3, Issue 9, 2025 ISSN: 2995-536X
https://medicaljournals.eu/index.php/IJCNP

INTERNATIONAL JOURNAL OF COGNITIVE NEUROSCIENCE AND PSYCHOLOGY

DIAGNOSTIC APPROACHES AND CLINICAL CORRELATES OF FACIAL NERVE NEUROPATHY IN CHILDREN

Bobomurodova Maftuna Xikmatillo qizi
Master's Program in Pediatric Neurology, Samarkand State Medical University

Abstract: Facial nerve neuropathy in children is an uncommon but clinically significant condition that can result in facial asymmetry, impaired speech, and long-term psychosocial consequences. Despite its relatively low incidence compared to adults, pediatric cases present unique diagnostic and therapeutic challenges. This review article aims to analyze diagnostic approaches and clinical correlates of facial nerve neuropathy in children based on current literature. Key etiologies include idiopathic (Bell's palsy), infectious, traumatic, congenital, and neoplastic causes, each with distinct clinical manifestations and prognostic implications. Diagnostic strategies encompass neurological examination, electrophysiological studies such as electromyography (EMG) and electroneurography (ENoG), neuroimaging techniques including MRI and CT, and selected laboratory investigations. Prognosis is closely linked to underlying etiology: while idiopathic palsy generally resolves in up to 80% of pediatric patients, infectious or traumatic etiologies often demonstrate less favorable outcomes. The article highlights that early, algorithm-based diagnostic evaluation combined with individualized management improves recovery rates and reduces the risk of complications. Current evidence also underscores the necessity for standardized pediatric-specific protocols, as most existing guidelines are extrapolated from adult studies. Future research should focus on large-scale pediatric trials, refinement of electrodiagnostic techniques for children, and long-term follow-up studies to better define prognostic indicators.

Key words: children, facial nerve neuropathy, Bell's palsy, diagnosis, electromyography, neuroimaging, prognosis.

Introduction

Facial nerve neuropathy (or facial palsy) in children is an important neurological condition that can substantially affect function and quality of life. The facial nerve (cranial nerve VII) controls facial expression, eye closure, salivation and lacrimation, so its impairment may lead to asymmetry, eye dryness, difficulty eating and psychosocial distress. Although less common in children than adults, pediatric facial palsy is a frequent emergency presentationhttps://www.physio-pedia.com/Facial_Nerve_Paralysis_in_Children - :~:text=Facial%20Palsy%20is%20one%20of,2. The annual incidence of childhood facial palsy is relatively low – on the order of 1–10 cases per 100,000 children depending on age – but it can reach up to 10 per 100,000 in adolescents. For example, one retrospective study found a mean incidence of 1.73 cases per 100,000 pediatric population per year. Bell's palsy (idiopathic facial paralysis) accounts for roughly 60–80% of these cases in children. The prognosis is generally favorable: most children recover facial function completely, with 80–90% regaining normal movement within six months. Nevertheless, any facial nerve neuropathy requires timely recognition and evaluation, as it may signify treatable infections, trauma, or neoplasms. Early diagnosis is important to initiate appropriate therapy (e.g., steroids or antibiotics) and to prevent complications such as corneal ulceration or persistent facial paralysis.

Etiology and Classification

Facial nerve neuropathy in children can arise from a variety of causes. It is broadly classified as congenital or acquired, and as central (upper motor neuron) versus peripheral (lower motor neuron) in terms of localization. The vast majority of pediatric cases are peripheral (lower motor neuron) palsies involving the facial nerve itself. Among acquired peripheral causes, Bell's palsy (idiopathic facial

paralysis) is the most common, seen in about 60–80% of children with facial palsy. When a cause is found, common etiologies include infections (viral or bacterial), trauma, and neoplasms.

Congenital facial palsy is usually apparent at birth or in infancy and often results from birth trauma (e.g. forceps delivery, difficult labor), syndromic or developmental abnormalities (e.g. Möbius syndrome, Goldenhar syndrome, LCHAD/Arnold-Chiari malformation), or genetic conditions (hereditary myopathies and rare chromosomal abnormalities). Congenital causes may be unilateral or bilateral and often require neuroimaging to identify structural anomalies.

Infectious causes include viral neuritis and bacterial otitis. Herpesviridae are implicated in many cases: for instance, varicella zoster virus can cause Ramsey Hunt syndrome (facial palsy with ear vesicles and severe pain), and herpes simplex virus is thought to underlie many idiopathic cases. Lyme disease (Borrelia burgdorferi) is a notable cause in endemic regions, often presenting with facial palsy that may be bilateral and recurrent. Other infections associated with facial palsy include Epstein–Barr virus, cytomegalovirus, enteroviruses, and neuroborreliosis. Acute otitis media and mastoiditis can infrequently cause facial paralysis, likely via inflammation and compression of the nerve in its bony canal. (The incidence of facial palsy as a complication of acute otitis media is very low – on the order of 0.005% in the antibiotic era – but should be considered in a child with ear infection and facial weakness.)

Traumatic causes include birth trauma (leading to congenital palsy), blunt trauma to the temporal bone (e.g. skull fracture), or surgical injury (especially to the parotid or ear). Traumatic palsies often have immediate onset after injury and may be accompanied by hearing loss or hemotympanum if the temporal bone is involved. Direct nerve injuries are a small fraction of cases (~2%). **Neoplastic causes** are rare in children but include cerebellopontine-angle tumors (e.g. schwannomas, medulloblastomas) and leukemias/lymphomas with cranial nerve infiltration. A slowly progressive facial palsy, especially with other cranial nerve signs, should raise suspicion for a tumor.

Idiopathic (Bell's) palsy remains a diagnosis of exclusion. In this category, no specific cause is identified, though many theories propose viral inflammation (e.g. reactivation of herpes simplex) leading to nerve edema. Bell's palsy is usually unilateral, of sudden onset, and typically affects all ipsilateral facial muscles (involving the forehead). Studies report that idiopathic facial palsy accounts for about 60–80% of pediatric facial palsy cases. These cases are often managed empirically with corticosteroids and sometimes antivirals, although the evidence for treatment in children is limited.

Other categories include autoimmune or inflammatory disorders (e.g. Guillain–Barré syndrome can present with bilateral facial weakness, sarcoidosis), metabolic causes, or medications (though rare in this age group). Bilateral facial palsy is unusual (0.3–2% of cases) and suggests a systemic process; for example, Lyme disease accounts for about 35% of bilateral palsies in children. Upper motor neuron lesions (central palsy) due to stroke or brain tumor must also be considered; these typically spare the forehead muscles and often have other neurologic findings. Overall, a careful history and examination usually allow classification into congenital vs acquired and point toward likely etiology, guiding further evaluation.

Diagnostic Approaches

A structured diagnostic approach is essential to identify the cause of facial nerve neuropathy in children and to plan management. Key components include a detailed clinical evaluation, electrophysiological studies, imaging, and selective laboratory tests.

Neurological examination. The physical exam should assess facial nerve function systematically. Eliciting wrinkles on the forehead, forceful eye closure, smiling, and puffing out cheeks tests the temporal, zygomatic, buccal, mandibular, and cervical branches. The degree of paralysis is often graded using the House–Brackmann (H-B) scale: Grade I (normal) through Grade VI (complete paralysis). The exam also distinguishes upper vs lower motor neuron lesions: an UMN (central) lesion will spare the forehead due to bilateral cortical innervation, whereas an LMN (peripheral) lesion produces weakness of the entire ipsilateral face. A thorough cranial nerve exam is performed to check

for additional deficits (e.g. hearing loss or vestibular signs if vestibulocochlear nerve is affected, or decreased lacrimation/salivation if the proximal facial nerve is involved). Otoscopic inspection for ear infection or vesicles (suggestive of herpes zoster) is important. A general neurological exam should look for other findings (e.g. ataxia, reflex changes) that might indicate Guillain–Barré syndrome or central pathology. The H-B grade at presentation often correlates with prognosis: in general, incomplete (lower-grade) paralysis is associated with better recovery.

Electrophysiological studies. Electrophysiology can document nerve injury and help prognosticate. Electroneuronography (ENoG) and electromyography (EMG) are the principal tests. ENoG, performed 3–14 days after onset, measures compound muscle action potentials of facial muscles and can quantify the percentage of nerve degeneration. It is particularly useful if denervation is severe (>90% degeneration), which indicates a poorer prognosis and may influence surgical decisions. EMG, usually done 3–4 weeks after onset, detects spontaneous fibrillation potentials in facial muscles, confirming axonal injury. Both tests are resource-intensive and require patient cooperation or anesthesia in young children, so they are not routinely done in all cases. As StatPearls notes, "electrophysiological tests are suitable for prognosis; however, they are expensive, time-consuming, and have a short time to be useful (less than three weeks after symptom onset)". In practice, EMG/ENoG are often reserved for severe palsy, delayed recovery, or when planning decompression surgery.

Electroneuromyography can also track recovery: the reappearance of voluntary motor unit potentials indicates reinnervation. In addition, simple bedside tests (e.g. the corneal reflex/blink test, Schirmer test for lacrimation, stapedial reflex) may provide functional information about the nerve segments involved. However, these are rarely needed in typical Bell's palsy workup.

Neuroimaging. Magnetic resonance imaging (MRI) and computed tomography (CT) play complementary roles. MRI with gadolinium contrast is the modality of choice to visualize the facial nerve along its entire course, detect enhancement (indicating inflammation or neoplasm), and identify lesions in the brainstem or cerebellopontine angle. It is particularly useful if the history or exam suggest an alternative cause (e.g. insidious onset, recurrence, associated neurological deficits, or unilateral ear symptoms). StatPearls emphasizes that "MRI scanning is useful for detection of intratemporal lesions that may be resulting in compression of the facial nerve and particularly useful for imaging the cerebellopontine angle. MRI scans may also identify enhancement of the facial nerve around the geniculate ganglion". CT scanning is indicated mainly when a bony abnormality is suspected, such as temporal bone fracture or cholesteatoma. For example, a high-resolution CT of the temporal bone can reveal canal dehiscence or fracture lines in traumatic palsy. CT is also indicated if there are symptoms of chronic otitis media or mastoiditis. In isolated, uncomplicated Bell's palsy, routine imaging is not universally recommended – one pediatric review stated that in acute isolated cases, no further investigation is needed. However, imaging should be obtained if the palsy does not improve as expected or if "red flag" features are present (see below).

Laboratory tests. There is no single blood test for facial palsy, but targeted tests can identify specific etiologies. A complete blood count and inflammatory markers (CRP, ESR) may be checked if infection is suspected. Serologies for *Borrelia burgdorferi* (Lyme) IgM/IgG are indicated in endemic areas or if there is a history of tick exposure or erythema migrans. Varicella-zoster virus antibody titers (or PCR from vesicle fluid) can confirm Ramsay Hunt syndrome. Testing for herpes simplex, cytomegalovirus or Epstein–Barr virus is occasionally done in research settings, but not routinely. Screening for diabetes or hypertension is more relevant in older adults than children. Cerebrospinal fluid analysis (via lumbar puncture) is reserved for cases with meningeal signs or concern for Guillain–Barré syndrome (which can mimic facial neuropathy). Hearing evaluation with audiometry and tympanometry is often recommended to assess for concomitant conductive or sensorineural hearing loss (which may accompany otomastoiditis or acoustic neuroma). One case series advised that "an audiogram and tympanogram should be conducted in all children with acute facial nerve paralysis". In summary, laboratory tests should be guided by clinical suspicion: for a child with typical Bell's palsy and no other symptoms, extensive labs are usually unnecessary. In contrast, a child with

systemic signs or bilateral palsy warrants a broader workup (e.g. Lyme serology, CSF exam for GBS markers, etc.).

Together, these diagnostic approaches – focused history and exam, selective electrophysiology, imaging, and lab studies – allow clinicians to identify the cause of facial nerve neuropathy in most cases. The workup is tailored to each child's presentation: **isolated, acute, idiopathic** palsies often require minimal testing, whereas **atypical or complicated** cases demand a thorough search for underlying disease.

Clinical Correlates

Understanding how clinical features correlate with etiology and outcome can guide diagnosis and counseling. Certain clues in the presentation suggest specific causes, and the prognosis varies accordingly.

Symptomatic clues and etiology: Idiopathic (Bell's) palsy typically presents with acute onset of unilateral facial paralysis, often noticed overnight. There is usually no rash, fever, or identifiable infection. Children may report a prior viral illness (e.g. upper respiratory infection) but often have no obvious trigger. Bell's palsy in kids tends to be less painful than Ramsay Hunt and is usually limited to the face. By contrast, **Ramsay Hunt syndrome** involves severe ear pain and vesicular rash in the ear canal or tympanic membrane, and often results in more profound weakness and slower recovery. **Lyme-related palsy** may be suggested by exposure history or systemic findings (rash, arthralgias). Importantly, Lyme often causes bilateral or recurrent facial palsy: any child with a facial palsy and known tick exposure should have Lyme serology. **Infectious palsy** from otitis media or mastoiditis is usually accompanied by ear pain, otorrhea, and fever. **Neoplastic palsy** is typically insidious: children may report months of gradually progressive weakness, and exam may reveal involvement of other cranial nerves (e.g. hearing loss from acoustic neuroma, or long tract signs in a brainstem glioma). **Traumatic palsy** has immediate onset after an injury or surgery. A history of temporal bone fracture or prior ear surgery is a strong indicator.

Prognostic factors: In general, children have an excellent prognosis. Most series report recovery rates of 80–100%. In fact, one study noted that by six months follow-up, the majority of pediatric patients had complete resolution of palsy. Factors associated with better recovery include younger age and milder initial paralysis. Yoo et al. found that a lower initial H-B grade (II–IV) strongly predicted complete recovery by six months. Conversely, a higher H-B grade (V–VI) at onset suggests axonal loss and a longer or incomplete recovery. The electrophysiological findings also correlate: if ENoG shows >90% degeneration by day 7–14, the risk of permanent weakness is higher. The pediatric review by Wohrer et al. emphasized that in **isolated acute cases** (presumed Bell's palsy), children "have a very good recovery rate" regardless of treatment.

Different etiologies carry different prognoses. Idiopathic Bell's palsy in children is almost always benign, with rapid recovery (often complete by 3–6 months). Viral cases such as Ramsay Hunt often have a slower recovery; only about 50–75% fully recover motor function in Ramsay Hunt. Lyme-related palsy typically improves fully with antibiotic treatment, but if missed or untreated, neurological sequelae may occur. Traumatic palsy outcome depends on the nature of injury: if the nerve is completely severed, spontaneous recovery is unlikely without surgical repair; if the nerve is only stretched or mildly compressed, partial to full recovery can occur over months. Congenital palsy (e.g. birth trauma) often shows slow gradual improvement, but if the nerve was avulsed, recovery may be poor.

In addition to motor function, associated symptoms correlate with etiology. For instance, decreased tearing on the affected side may indicate a lesion at the geniculate ganglion or proximal nerve (which carries parasympathetic fibers), as seen in Bell's palsy or Ramsay Hunt. Loss of taste on the anterior two-thirds of the tongue suggests chorda tympani involvement (often in proximal lesions). Hearing assessment can reveal conductive loss in otitis media cases or sensorineural loss with CPA tumors.

Psychosocial impact is also a consideration: even transient facial asymmetry can affect a child's self-esteem and social interactions.

In summary, the **clinical presentation** often points to a likely cause: typical idiopathic palsy has a classic course with excellent prognosis, while any atypical features or accompanying symptoms should prompt consideration of alternate diagnoses. Prognosis is usually favorable, especially in idiopathic cases, but depends on initial severity and underlying etiology.

Discussion

A critical review of the literature highlights both the strengths and limitations of current diagnostic strategies for pediatric facial nerve neuropathy. The consensus is that **history and physical exam remain the cornerstone** of diagnosis. A careful exam can usually distinguish peripheral from central lesions and can identify "red flags" (e.g. bilateral palsy, gradual onset, systemic symptoms) that warrant further investigation.

Electrophysiology has prognostic value but limited immediate impact on acute management. While ENoG/EMG can identify axonal degeneration and guide counseling on recovery, their utility is constrained by timing (must be done days to weeks after onset) and practicality. As one review notes, these tests "cannot successfully predict recovery" in the acute setting and are not routinely recommended for initial management. Indeed, in uncomplicated Bell's palsy, guidelines generally advise against routine electrodiagnostic testing. Our cited StatPearls source concludes that these tests are "suitable for prognosis" but emphasizes they are expensive and time-sensitive.

Neuroimaging has improved greatly in sensitivity, but its role is still targeted. MRI may reveal nerve enhancement in idiopathic palsy, but such findings do not change management unless a structural cause is identified. Routine MRI of all facial palsy cases yields few actionable findings: one JAMA study in adults found that only 6.7% of routine facial nerve MRIs discovered an alternative cause. In children, sedation risks and cost must be weighed. Thus, most experts recommend imaging only for atypical cases: bilateral palsy, incomplete/no recovery by 3–6 months, recurrent episodes, or suspicious exam findings. In ear-related palsy (e.g. with otitis media), a CT scan is justified to evaluate for bony disease. In summary, imaging is a powerful tool but must be used judiciously, as emphasized in pediatric reviews.

Laboratory testing likewise must be selective. Routine blood tests add little in typical Bell's palsy. AAFP guidelines note that tests and imaging are not needed for straightforward cases, but serologic tests can uncover systemic causes (e.g. Lyme serology for suspected neuroborreliosis). The pediatric literature suggests checking Lyme IgG/IgM in endemic areas (especially if the palsy is bilateral or recurrent). Viral titers (HSV, VZV) or PCR are generally of limited availability and do not routinely alter acute treatment. A lumbar puncture is only indicated if meningitis or Guillain–Barré syndrome is suspected (e.g. if there are headache, fever, or limb weakness). Thus, the limitation is that a definitive diagnosis is often not reached: many cases remain labeled "idiopathic" even after investigations.

Overall, the current diagnostic strategy emphasizes excluding serious causes without over-testing benign cases. This approach has strengths (avoiding unnecessary sedation/tests in kids) but risks under-diagnosis. For example, a subtle temporal bone fracture or early tumor might be missed without imaging. On the other hand, excessive workup in every case could lead to undue anxiety and expense.

Future directions should aim at refining these strategies. Larger studies could better define which clinical predictors most strongly warrant further testing. Development of clinical algorithms (e.g. the pediatric studies on "red flag" symptoms) would help standardize care. Improved noninvasive biomarkers (e.g. novel imaging contrasts, nerve ultrasound) might one day identify nerve inflammation or injury. Finally, research into therapies (such as randomized trials of steroids or antivirals in children) could clarify the optimal management once the diagnosis is made.

Conclusion

Facial nerve neuropathy in children is a multifaceted condition with generally favorable outcomes. The majority of cases are idiopathic (Bell's palsy) with a benign course and high rate of complete recovery. Nevertheless, a careful diagnostic approach is essential to identify the minority of cases with treatable causes. A thorough neurological examination distinguishes central from peripheral lesions and identifies associated findings that suggest infection, trauma or malignancy. Electrophysiological studies can quantify nerve injury and predict prognosis, but are mainly used in severe or unclear cases due to their timing and cost. Neuroimaging (MRI/CT) should be performed when red flags are present, whereas routine imaging in classic Bell's palsy is not routinely indicated. Laboratory tests such as Lyme serology or viral titers should be guided by epidemiology and clinical suspicion.

In practice, most children with isolated, acute-onset facial palsy require no extensive workup. Those with atypical features – bilateral involvement, persistent palsy beyond 3–6 months, recurrence, or other neurological signs – undergo targeted testing. Across causes, the prognosis in children is generally excellent, better than in adults. Future research should focus on prospective studies to validate diagnostic algorithms, as well as on trials of therapies and studies of long-term outcomes. In the meantime, clinicians should maintain a high index of suspicion for underlying diseases while recognizing that idiopathic cases are common and often self-limited.

References

1. El-Deeb ME, Elzayat S, Salamah A, Gamal A, Elgamal S, El-Sobki A. *Is There an Association between Bell Palsy in Pediatric Patients and COVID-19?* Int Arch Otorhinolaryngol. 2025;29(suppl 1):eX–eX. DOI:10.1055/s-0044-1789197.
2. Fancello V, Ciorba A, Monzani D, Genovese E, Bussu F, Palma S. *Acute Onset of Peripheral Facial Nerve Palsy in Children: An Overview*. Pediatr Rep. 2024;16(4):844–853. DOI:10.3390/pediatric16040072.
3. StatPearls. *Facial Nerve Palsy*. Treasure Island (FL): StatPearls Publishing; 2025. Available from: https://www.ncbi.nlm.nih.gov/books/NBK549815/ (accessed 2025).
4. StatPearls. *Diagnosis of Facial Paralysis*. Treasure Island (FL): StatPearls Publishing; 2025. (Section on investigations).
5. Teixeira DAN, de Figueiredo MV, Fernandes RG, et al. *Peripheral facial paralysis as a consequence of acute otitis media: Physiopathology, associated factors and treatment*. Otorhinolaryngology. 2017;7(4):1–7. DOI:10.15761/OHNS.1000225.
6. Psillas G, Antoniades E, Ieridou F, Constantinidis J. *Facial nerve palsy in children: A retrospective study of 124 cases*. J Paediatr Child Health. 2019;55(3):299–304. DOI:10.1111/jpc.14190.
7. Lorch M, Teach SJ. *Facial nerve palsy: etiology and approach to diagnosis and treatment*. Pediatr Emerg Care. 2010;26(10):763–769. DOI:10.1097/PEC.0b013e3181f7e5c7.
8. Wohrer D, Moulding T, Titomanlio L, Lenglart L. *Acute Facial Nerve Palsy in Children: Gold Standard Management*. Children (Basel). 2022;9(2):273. DOI:10.3390/children9020273.
9. AAFP. *Bell's Palsy: Evidence-Based Clinical Guidelines*. American Academy of Family Physicians. 2019. Available: https://www.aafp.org/ (Rapid Evidence Review).
10. Brito LP, Teles AR, Mion O Jr. *Peripheral facial nerve paralysis in children: Clinical manifestations and diagnostic evaluation*. J Pediatr (Rio J). 2015;91(4):342–347.
11. Rocha AC, Pereira NJ, Santos GC. *Facial Paralysis Due to Otitis Media*. Otolaryngol Clin North Am. 2019;52(4):685–701.
12. Lima MA, Silva MTT, Soares CN, et al. *Bell's palsy and COVID-19: A case series and literature review*. Int Arch Otorhinolaryngol. 2021;25(03):e334–e338.

13. Rodriguez JG, Almeida A, Rose A. *Facial nerve paralysis: a primer*. Am Fam Physician. 2018;98(5):284–291.

14. Ash SR, Michels L, Sinha U, Gildenberg P. *Bilateral facial palsy in Lyme disease*. Ear Nose Throat J. 2016;95(4-5):E39–E41.

15. Morris PS, Vassar MJ, Song L, et al. *EMG and ENoG in facial palsy evaluation*. Laryngoscope. 2022;132(8):1597–1602. DOI:10.1002/lary.30457.

16. Psillas G, Gavriel H, Bance M. *Auditory evaluation in pediatric facial palsy*. Int J Pediatr Otorhinolaryngol. 2017;101:134–139.