15 minute consultation: a structured approach to the management of facial paralysis in a child

Vikas Malik,¹ Vineeta Joshi,² Kevin M J Green,¹ Iain A Bruce¹

¹Department of ENT, Royal Manchester Children's Hospital, Manchester, UK

²Department of Paediatrics, Royal Manchester Children's Hospital, Manchester, UK

Correspondence to

Mr Vikas Malik, Department of ENT, Royal Manchester Children's Hospital, Oxford Road, Manchester, M13 9WL, UK; malikvik@yahoo.com

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Abstract

Objective To present a structured approach for an outpatient consultation of a child with facial paralysis.

Method Review of literature and description of approach followed in our unit.

Conclusion A focused history and examination is key to establish the cause and draw a management plan for paediatric facial paralysis.

A 12-year-old child is referred by his general practitioner with unilateral facial paralysis.

What should you cover in the history?

Facial nerve paralysis (FNP) in a child can be due to diverse aetiologies (table 1). Paediatric facial nerve palsy can be congenital or acquired, with the acquired being more common. The common causes of FNP in children are Bell's palsy (40–70%),^{1 2} infection (13–36%), trauma (19–21%), congenital (8–14%) and neoplasm (2–3%).^{3 4} A focused history and examination is crucial in order to reach a working differential diagnosis and plan further management. Following points should be covered in the history.

Onset, progression and duration

Rapidity of onset of facial palsy does not point to a specific diagnosis.⁵ Almost all patients demonstrate signs of recovery between 3 weeks and 3 months,^{2 3} and this course indicates a benign cause. Facial palsy that continues to progress beyond 3 weeks or lacks improvement after 3 months is suggestive of neoplastic/neurological cause and should be appropriately investigated.⁴ Recurrent facial nerve paralysis (RFNP) is rare, with an incidence of 6%.⁶ Causes of RFNP are Bell's palsy, Melkersson–Rosenthal syndrome (recurrent alternating facial palsy, fissured tongue, facial oedema, positive family history) and rarely secondary to an underlying neoplasm. Therefore, all children with RFNP should be investigated.

Laterality

Bilateral FNP is likely to be due to neurological causes such as Gullian–Barre syndrome, Moebius syndrome (bilateral facial nerve and bilateral abducens paralysis due to intrauterine brainstem necrosis) or secondary to infection (cytomegalovirus infection, Epstein–Barr virus and Lyme disease).

Associated aural symptoms

Presence of aural symptoms indicates otitis media, cholesteatoma or tumour. Facial paralysis secondary to otitis media is rare and has incidence of 0.005%.⁷ Hearing loss, otalgia and facial pain may be seen in Bell's palsy and Ramsay–Hunt syndrome.

History of trauma

The lateral position of the facial nerve in children makes it more susceptible to damage. Facial nerve injury can occur in head trauma especially with longitudinal fracture of temporal bone (90%).⁸ FNP may also be seen following blunt cheek injury. Rarely, facial palsy occurs following parotid surgery, middle ear/mastoid surgery^{9 10} or following forceps delivery.⁴

History of immunisation

Facial nerve can rarely be affected by immunisation against polio, rabies, tetanus and influenza.¹¹

Underlying illness

Haematological disorders, such as leukaemia,^{9 12} haemophilia,¹³ may rarely present with FNP. Acute onset FNP has also been reported in cases of malignant hypertension.¹⁴ Ear infection (otitis media, Ramsay–Hunt syndrome) and neurological infections such as meningitis, encephalitis, leprosy, botulin and tetanus may also affect the facial nerve.

Eye symptoms

History of grittiness, burning or stinging sensation in ipsilateral eye, excessive watering and blurred vision should be taken.

Focused examination in a child with FNP

Assess severity of facial weakness at presentation

It is important to document the severity of facial weakness at presentation to allow assessment of recovery or progression at future visits. Clinical examination of facial function in older children is similar to an adult, allowing grading of facial function using the House-Brackmann (H.B.) scoring system (table 2).¹⁵ In younger children where examination is difficult and a meaningful grading (H.B.) score is not achievable, observation of spontaneous facial movements is important. It is useful to document which facial movements are witnessed during the consultation and which movements the parents feel that a child can achieve. Forehead movements should be particularly noted as they are affected in infranuclear palsy whereas preserved if the lesion is above the facial nucleus, ie, supranuclear.

Assessment for aetiological factors

Examination of pinna, external auditory canals, tympanic membranes and oral cavity/oropharynx should be undertaken; presence of vesicles is suggestive of Ramsay–Hunt syndrome. Signs of acute otitis media, acute mastoiditis and chronic otitis media with or without cholesteatoma should be looked for. An abnormally sited external ear, ear deformity or dysmorphism may be associated with congenital FNP. Stigmata of a temporal bone fracture should be sought in the presence of history of trauma (Battle's sign, haemotympanum and traumatic perforation).

Examination of eye

Look for degree of eyelid closure, redness of conjunctiva, frequency of blinking and excessive watering or dryness of the involved eye.

Besides focused examination, a complete general and systems examination should be carried out including remaining cranial nerves and the central nervous system. Examination of the neck must be performed to illicit a parotid neoplasm or cervical lymphadenopathy. Joints should be examined in endemic areas of Lyme disease (bilateral FNP with other cranial nerve involvement, due to *borrelia spirochete*). The cerebrospinal fluid (CSF) in patients with Lyme disease often shows an inflammatory response.

What management should you provide?

Investigations

The following investigations may be carried out as indicated from the history and examination.

Hearing test

Audiometry and tympanometry should be performed if middle ear involvement is suspected.¹⁶ These tests are usually abnormal in the presence of middle ear infection/cholesteatoma. Normal tympanogram indicates a normal middle ear space.

Haematological tests

Full blood count to rule out leukaemia,^{9 12} *borrelia* serology in endemic areas for Lyme disease¹⁷ should be done as indicated.

Table 1 Causes of facial nerve palsy

1. Idiopathic
a. Bell's palsy
b. Melkersson–Rosenthal syndrome (recurrent alternating facial palsy, furrowed tongue, faciolabial oedema)
2. Infection
a. Otitis media
b. Mastoiditis
c. Herpes zoster cephalicus (Ramsay–Hunt syndrome)
d. Chickenpox
e. Encephalitis
f. Meningitis
g. Poliomyelitis (type I)
h. Mumps
i. Infectious mononucleosis (glandular fever)
j. Malaria
k. Tuberculosis
l. Lyme disease
m. HIV
3. Trauma
a. Skull base fractures
b. Facial injuries
c. Penetrating trauma to middle ear
d. Barotrauma (altitude paralysis/scuba diving)
4. Metabolic
a. Diabetes mellitus
b. Hypertension
c. Acute porphyria
5. Neoplastic
a. Cholesteatoma
b. Leukaemia
c. Haemophilia
d. Fibrous dysplasia
e. Parotid tumours
f. Facial nerve tumour
g. Cerebello-pontine angle tumours
6. Toxic
a. Tetanus
b. Diphtheria
c. Thalidomide
d. Carbon monoxide
7. Iatrogenic
a. Postimmunisation
b. Antitetanus serum
c. Vaccine for rabies
d. Parotid surgery
e. Mastoid surgery
f. Forceps delivery
8. Autoimmune syndrome
a. Thrombotic thrombocytopenic purpura
b. Kawasaki disease
c. Guillain barre/Miller–Fisher syndrome
9. Neurological
a. Millard-Gubler syndrome (abducens palsy with contralateral haemiplegia due to lesion in the base of pons involving corticospinal tract)
b. Opercular syndrome (cortical lesion in facial motor area)
10. Congenital
a. Dystrophia myotonica
b. Moebius syndrome (facial diplegia associated with other cranial nerve deficits)

Radiological imaging

MRI brain may be recommended in patients with FNP progressing beyond 3 weeks, lack of improvement after 6 months, recurrent facial palsy, single facial

segment involvement or if there is a high suspicion of an underlying neurological disorder. High resolution CT scan of temporal bones is indicated in cases of temporal bone trauma or if there is a suspicion of middle ear cholesteatoma.

Prognostic tests

If the FNP is partial and does not progress to complete paralysis, then the patient can be reassured of excellent eventual recovery without further testing. Suggested time for monitoring recovery is 3 weeks, 3 months, 6 months and 1 year from onset. Electrodiagnostic (neurophysiological) tests can provide prognostic information and evaluate patients who do not show recovery within 3 weeks.¹⁸ However, these tests are difficult to carry out in children, requiring significant cooperation of the child and are generally not performed. The simpler tests are measurement of fibrillation potentials (as part of electromyography) and recording of blink reflex.¹⁸

Treatment

The medical management of a child with FNP is similar to that of an adult with FNP.

Eye care is the most important treatment in FNP. Regular use of artificial tears (eg, hypromellose eye drops) during daytime and moisturising eye ointment (eg, lacrilube) during sleep prevents dryness of eye. Glasses should be worn outdoors to protect the eye from windborne particles. If done improperly, taping of the eye can cause corneal injury and should be discouraged in children. Ophthalmological opinion should be sought if the patient has eye discomfort or redness of the sclera at any point.

If there is an obvious physical cause for the FNP (cholesteatoma or neoplasm), it should be dealt with by the involvement of appropriate teams.

Children who have FNP as a part of a syndrome or have neurological/haematological disorders should be managed in conjunction with the appropriate paediatrician.

In the absence of other causes in the history or examination, the presumptive diagnosis is Bell's palsy. Most authors believe that herpes simplex virus infection is the likely cause of Bell's palsy. Treatment for Bell's palsy with steroid or antiviral or both is controversial. The vast majority of children will recover completely, with or without treatment. Due to a paucity of randomised-control trials in children with Bell's palsy, the efficacy of steroids as well as other therapies such as acyclovir can only be extrapolated from studies done in adults.¹⁹ A double-blinded randomised-control trial conducted by Sullivan *et al* in Scotland showed statistically higher rate of recovery of Bell's palsy following prednisolone alone. There was no additional benefit from combining acyclovir and prednisolone compared with prednisolone alone. Acyclovir alone did not show any significant difference in the rate of facial recovery compared with placebo.²⁰ A Cochrane review of the role of antivirals in Bell's palsy found insufficient

Table 2 House-Brackmann scoring of facial function

Grade	Definition
I	Normal facial functions in all areas
II	Slight weakness noticeable only on close inspection Complete eye closure with minimal effort Slight asymmetry of smile with maximal effort No synkinesis, contracture of haemifacial spasm
III	Obvious weakness, but not disfiguring May not be able to lift eyebrow Complete eye closure and strong but asymmetrical mouth movement with maximal effort Obvious, but not disfiguring synkinesis, mass movement or spasm
IV	Obvious disfiguring weakness Inability to lift brow Incomplete eye closure with asymmetry of mouth with maximal effort Severe synkinesis, mass movement, spasm
V	Motion barely perceptible Incomplete eye closure, slight movement of corner of mouth Synkinesis, contracture and spasm usually absent
VI	No movement, loss of tone, no synkinesis, contracture, or spasm

evidence supporting their role and recommended further randomised-control trial.²¹

Prednisolone is typically prescribed in dose of 1 mg/kg (maximum 60 mg) for 7 days to be tapered over next few days, started within a week of onset (ideally within 72 h) provided there is no contraindication.

The prognosis of a child with Bell's palsy is generally excellent, and the risk of recurrence is only from 7% to 10%.²² Almost all patients demonstrate signs of recovery between 3 weeks and 3 months.²³ The lack of any signs of recovery or progression of FNP should raise a suspicion of an underlying cause which should be thoroughly investigated including radiologically.

Surgical treatment: facial reanimation techniques, static or dynamic or both, may be employed for treatment of FNP that does not recover. The ideal time for the intervention is controversial.

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