Facial palsy in childhood

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Abstract
The facial nerve is the most commonly morbid cranial nerve. It contains motor, sensory and parasympathetic fibres, and has a complex course which is a source of vulnerability. Although facial palsy is less common in children than in adults, idiopathic palsy is the most common diagnosis in both groups and is regarded as having a better prognosis in children than in adults, irrespective of treatment. There is a paucity of high-quality evidence to inform the treatment of children with facial palsy; most recommendations are based on evidence from adult studies. The literature generally supports medical over surgical therapies; oral steroid and anti-infective agents are the cornerstones of management.

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Key words
Facial palsy, paediatric, Bell's palsy, paralysis, neuropraxia

Pathophysiology and Epidemiology
There are many potential causes of facial palsy, the more common being infectious, traumatic, congenital, neoplastic and idiopathic. Approximately half of acute cases are idiopathic and are given the label "Bell's Palsy".

Bell's Palsy
In one study of 170 patients aged 18 years or under, Bell's palsy was the final diagnosis in 42%, trauma in 21%, infection in 13%, congenital palsy in 8% and neoplasia in 2%[1]. The estimated prevalence of Bell's Palsy is 20 per 100 000 people per year[2].

One widely accepted theory is that Bell's palsy occurs secondary to axonal spread and multiplication of a latent neurotropic virus, causing neuropraxia through inflammation and demyelination. Since there is no effective method to test for the presence of such a virus within the nerve clinically, however, most patients with idiopathic palsy are given "Bell's Palsy" as a diagnosis[3].

There is no geographical, racial or gender predilection in Bell's palsy, but there is a threefold increased risk in the third trimester of pregnancy and first post-partum week[4], and a fourfold increased risk in persons with diabetes[2].

Infection
Herpes Simplex Virus is widely thought to be the neurotropic virus behind Bell's palsy in both children[3,5] and adults[6,7]. There is some direct evidence for this. One case-control study examined samples of endoneurial fluid and posterior auricular muscle in patients undergoing decompression surgery for idiopathic palsy. HSV-1 genomes were identified in 11 of 14 cases, and in none of the controls[8]. In a prospective case-control study of children with acute unilateral palsy, positive PCR and ELISA for HSV-1 genomes were significantly more likely in cases than controls[3].

Herpes zoster infection has also been linked to facial palsy. In societies with low immunisation rates, acute zoster viraemia has been identified with PCR or serology in up to 37% of cases, including both zoster sine herpete (without skin signs) and herpes zoster oticus (Ramsay-Hunt syndrome).

Acute otitis media (AOM) was once considered a major cause of facial palsy in childhood, although the incidence has fallen from approximately 2% in the pre-antibiotic era to 0.16% more recently[9].

Studies in endemic areas have found Lyme disease to be a common cause. In one paediatric study in an endemic area of the United States, Lyme disease was the causative factor in 50% of cases, followed by idiopathic palsy in 26%, AOM in 12%, varicella (6%), zoster (4%) and coxsackie virus (2%)[10]. Similar findings were reported from a paediatric population in a Lyme-endemic area of Scandinavia[11].

Lyme disease is infection with bacteria of the genus Borrelia – typically Borrelia burgdorferi. The vector is a tick (genus Ixodes, usually in nymphal form). In Europe, Ixodes ricinus (the castor bean tick) is the usual culprit, and transmits the infection more rapidly than Ixodes scapularis, the typical North American vector. The disease is not known to be transmissible between people, via other animal vectors, or through faeco-oral routes.

Facial palsy may be the only symptom of Lyme disease[12], can be unilateral or bilateral, and usually resolves within eight weeks if the underlying infection is treated[13]. It is the most common cranial neuropathy in Lyme meningitis[14,15], but can occur secondary to Lyme disease without meningeal involvement. One theory is that this relates to direct invasion of the nerve trunk by Borrelia; however, evidence is weak, comprising retrospective studies showing that a high percentage of children with confirmed neuroborreliosis and unilateral palsy had an ipsilateral source such as a tick bite in the head & neck region[11,16].

HIV infection rarely causes facial palsy in children directly, although where it does, it typically occurs during the seroconversion illness[17]. Where HIV infection leads to reduced cellular immunity, other opportunistic infections may result in palsy[18].

In areas without access to vaccination programmes, compression from mumps parotitis may be a common cause[19].

Other infections which have been linked to onset of facial palsy in children include CMV, EBV, adenovirus, rubella, mumps, influenza B, coxsackie virus and Rickettsia (Mediterranean spotted fever)[19,20,21]. Palsy in the presence of purulent otorrhoea unresponsive to typical antibiotic therapy raises the possibility of tuberculosis[22,23].

Congenital

Congenital facial palsy may in reality be traumatic and perinatally acquired. The facial nerve position relative to the infant mastoid, the mandibular angle and the maternal sacrum increase its vulnerability during birth; instrumental delivery compounds the problem. The incidence is 1.8 per 1000 live births, and risk factors include large head size (birth weight greater than 3500g), forceps-assisted delivery and prematurity[24,25].

True congenital palsy can occur in isolation, but is commonly associated with multiple cranial nerve palsies, and multisystem dysmorphia. It can occur secondary to branchial arch development sequence disorders[26]. Two genetic loci for developmentally-derived congenital palsy have been identified, designated hereditary congenital facial paresis 1 (chromosome 3q21–22) and 2 (chromosome 10q21.3–22.1)[27,28,29].

Möbius (Moebius) syndrome typically comprises unilateral or bilateral congenital facial and abducens palsy, although cranial nerves III, IV, V and VIII can also be affected[30,31]. Anatomical analysis has shown hypoplasia of associated brainstem nuclei and nerves[32]. Up to one third of affected individuals have associated intellectual problems and/or autism[33]. Genetic linkage analysis points to a locus at 13q12.2–13, although distinct causative genes have yet to be identified[34,35]. It is possible that Möbius and the other facial paresis syndromes overlap and share common aetiology.

Goldenhar syndrome (oculo-auriculo-vertebral dysplasia) is a group of congenital anomalies affecting structures arising from the first and second branchial arches. Congenital facial paralysis is notable in more severe forms[36].

Congenital Asymmetric Crying Facies is caused by unilateral agenesis or hypoplasia of the depressor anguli oris. 45% of cases are associated with developmental anomalies in other systems[37].

Facial palsy has been noted in 38% of patients with CHARGE syndrome[38], and some reports detail patients with an aberrant facial nerve course, complicating cochlear implantation[39].

Trauma

The greater superficial petrosal nerve tethers the geniculate ganglion, leaving the tympanic and mastoid segments of the facial nerve susceptible to shearing forces during sudden head movements, particularly deceleration injures. Despite this, the rate of palsy in paediatric temporal bone fractures is low. One study of children up to 14 years of age found facial weakness in 3% of 72 fractures[40].

Neoplasia

Neoplasia in childhood is thankfully rare. The two most common causes of malignancy-associated facial palsy are infiltration of the temporal bone by leukaemia[41,42], and rhabdomyosarcoma[43,44,45]. Neoplasia of the facial nerve is rare in childhood.

Other aetiologies

Cholesteatoma should be considered if the onset of palsy is gradual. Tympanic membrane examination will often reveal the diagnosis, but congenital cholesteatoma can present with neurological or hearing impairment before the tympanic membrane is involved[2].

Melkersson-Rosenthal syndrome classically comprises facial paralysis, episodic facial swelling and a fissured tongue; although the majority of cases are "atypical" forms, without one component of the triad. Onset is typically in adolescence, with recurrent episodes of facial paralysis thereafter[46]. Although one report noted presence of perivascular granulomas in oedematous tissues[47], the cause is unknown and treatment therefore controversial.

Neurosarcoidosis and Guillain-Barre syndrome have been linked with subacute bilateral facial palsy in children[48]. Palsy has been reported as part of an otological presentation of granulomatosis with polyangiitis (GPA)[49]. It has also been linked with severe systemic hypertension of childhood and adolescence, particularly in combination with headache, altered conscious level, vomiting, convulsions or other focal neurology[50,51,52].

Diagnosis

The history should cover onset, rate of deterioration, associated symptoms such as taste sensation, hyperacusis and headache, ear discharge, recent infections or illnesses, tick bites and recreation in woodlands, and HIV risk factors.

Relevant past medical history includes diabetes mellitus or other immune-modifying diseases, history of chronic ear disease, and immunisation history.

Physical examination should include the facial nerve, parotid, ear, neck and other cranial nerves as appropriate. In a young child who will not comply with examination, the palsy may not be apparent until the child cries. Forehead sparing can be detected by the presence of skin creases and paralysis of the lower eyelid, which may cause ectropion and tear spillage; loss of midfacial muscle tone may cause droop of the cheek and loss of the nasolabial fold. Oral continence may be lost, and speech may become distorted as plosive sounds are undermined by air escape.

The typical history of idiopathic palsy includes involvement of all peripheral branches, with acute onset over 24 – 48 hours. Classically there is rapid progression, reaching maximal clinical weakness within three weeks from symptom onset. There may be a prodrome, including ear pain and altered hearing level, but this does not occur commonly enough to be considered typical.

Painless, nontender swelling and erythema of the face progressing to facial palsy suggests Lyme disease[53], as do presence in a Lyme-endemic area, recent recreation in woodlands, and onset in spring or early summer. The typical course of Lyme disease includes onset of a non-pruritic non-tender rash approximately one week after infection. It can take the form of erythema migrans, and a bullseye rash pattern is considered highly suggestive of Borrelia infection.

Congenital palsy will usually come to attention shortly after birth. Prolonged traumatic labour, instrument delivery, head & facial injury, periauricular ecchymoses and haemotympanum may point towards a traumatic cause, but do not in themselves exclude the presence of developmental anomalies.

Topographical testing to determine the site of a facial nerve lesion has little clinical relevance and carries no prognostic value. It is therefore principally of interest in the study of the history of medicine.

Serology

Lyme serology is negative for four to six weeks after initial infection and is highly sensitive and specific thereafter. It is rare for palsy to develop before the patient is seropositive. Lyme serology is recommended for all children with acute-onset facial palsy in the spring, summer and early autumn.

Lumbar puncture

Clinical suspicion of meningitis should provoke lumbar puncture. The CSF concentration of anti-Borrelia antibody is diagnostic of neuroborreliosis. CSF analysis can also aid the diagnosis of Lyme disease without meningeal involvement, as the majority of children with isolated Lyme disease-associated palsy have abnormal findings, including elevated WBC and/or protein levels. These findings are clearly not pathognomonic, however, therefore the decision to obtain CSF should be made in consultation with paediatric infectious disease specialists, microbiologists and the child's carers[11,54,55].

Imaging

Exact imaging techniques should be discussed with an experienced radiologist; a rule of thumb is that MRI is useful in the evaluation of the intraparotid and intracranial facial nerve, and CT is useful in the evaluation of the temporal bone. Contrast enhancement of the geniculate ganglion on MRI may be seen in idiopathic palsy; however caution is advised, as contrast enhancement of the first genu and proximal tympanic segment may be normal[2,56]. Contrast enhancement of the labyrinth in the context of facial palsy suggests infection with herpes zoster, even in the absence of vesicular eruptions[57].

Coexisting facial and abducens palsy mandates imaging of the brainstem, as the nuclei are co-located in the pons. Imaging is recommended in the slowly-evolving palsy,

and also with those individuals showing a typical onset of idiopathic palsy but failing to show any improvement at six months[58].

Presence of forehead sparing (facial palsy with preserved temporal branch function) mandates imaging. Facial movement may be voluntary or emotional in origin, and the lower motor neurone pathway is common to both. Dissociation of voluntary facial movement from emotional movement therefore suggests a supratentorial problem and mandates imaging.

Imaging is also indicated if clinical findings suggest cholesteatoma, chronic otitis media, mastoiditis, temporal bone trauma or neoplasia.

Electrophysiology

Electrophysiological tests can determine the extent of facial nerve dysfunction, and therefore do have prognostic value. They may also assist in determining when surgical decompression may be recommended and when facial reanimation should be considered. If performed in series, they may allow monitoring of recovery, and so may guide management. In the case of palsy noticeable shortly after birth, electrophysiology can help distinguish between traumatic and congenital lesions.

In the first few days after onset of palsy, assessment of the blink reflex (via stimulation of the supraorbital nerve and the trigeminofacial reflex pathway) can confirm the peripheral location of the lesion, and assess the degree of axonal conduction block, thereby holding prognostic value[59].

Electroneuronography (ENoG, also known as a motor nerve conduction study) compares nerve conduction between the two sides, and therefore requires a functioning contralateral nerve. The main trunks are stimulated at the stylomastoid foramen, and compound muscle action potentials (CMAPs) are detected at the nasolabial fold. Their amplitude in comparison to the unaffected side estimates the degree of axonal loss[59]. Wallerian degeneration normally begins within 3 days, CMAPs generally reach their minimum level between 7 and 14 days post-onset – hence ENoG is most useful between 3 and 21 days post-onset. An amplitude reduction of 95% or more within this window is associated with a poor prognosis[59,60,61].

Electromyography (EMG) is more useful than ENoG in cases of delayed paralysis and can be used in bilateral palsy. The patient is asked to attempt facial movement, and motor unit potentials are measured in the orbicularis oris and orbicularis oculi muscles. Fibrillation potentials are seen in the context of Wallerian degeneration, and polyphasic potentials indicate early reinnervation although, in this situation, clinically obvious muscle movements may not occur for another three months. Electrical silence is a poor prognostic indicator.

Classification

Several classifications of nerve injury exist; the most commonly used are the Seddon and Sunderland classifications[62].

Treatment

Treatment should be guided by the pathophysiology, severity and timescale of the palsy. In all cases, the patient and their carers should understand the importance of eye care. If the patient is unable fully to close their eye, they are at risk of corneal abrasion, and should be treated with artificial lubricants during the day, and ointment and patches at night. Taping the eyelid is no longer advised, owing to the possibility of the tape slipping and abrading the cornea. Further measures are not usually required in the acute setting; tarsorrhaphy and gold weight implantation may be required if the palsy is prolonged.

Idiopathic (Bell's) palsy

Direct evidence is poor in the paediatric population, as there are no high-quality trials, however the prognosis is generally favourable[63,64]. The mainstay of treatment in adults is early administration of oral steroid – a recommendation supported by several high-quality trials[65,66,67,68,69,70,71] and meta-analyses[72,73,74,75,76]. Trials have reached differing conclusions on the merits of antiviral agents administered with steroid therapy; antiviral monotherapy was found to be no better than placebo.

In the absence of high-quality evidence in children, national bodies and learned societies recommend early intervention with oral steroid. A typical regime might be Prednisolone 2mg/kg od (maximum 60mg) starting within 3 days of symptom onset, continuing for 5 days, followed by a short taper[77].

Although large well-regarded clinical trials have not found additional benefit with added antiviral therapy, some trials have found benefit, particularly for (adult) patients with severe palsy[68]. Some bodies therefore also suggest combining steroid therapy with an oral antiviral such as valaciclovir 20mg/kg (maximum 1g) tds for 7 days. Expert opinion has not reached consensus on this point[77].

Ramsay-Hunt syndrome

Patients with herpes zoster oticus should be treated with antiviral therapy and oral steroid.

Other infections

AOM and neuroborreliosis should be managed according to local protocols, in consultation with expert microbiologists. Borrelia is often treated with doxycycline, or with amoxicillin plus cefuroxime.

Surgical intervention

There are no high-quality studies concerning early surgical management of facial palsy in children. One non-controlled study in adults evaluated surgical decompression of the labyrinthine segment via a middle cranial fossa approach, showing benefit in patients with severe palsy (defined as ≥90% reduction in CMAPs on electrophysiology)[78]. Another study reported that early surgical decompression was not associated with improved recovery but was associated with reduction in future episodes of palsy[79]. Guidance from the American Academy of Neurology in 2001[80] and AAO-HNS in 2013[81] do not recommend surgical decompression, owing to the paucity of high-quality evidence and the potential for surgical complications.

Children with congenital or permanent palsy may be offered surgical intervention, either for static appearance or reanimation[82,83]. Static procedures aim to achieve oral and labial symmetry at rest; reanimation techniques aim to allow some facial expression. These techniques cannot restore normal function, although even a modest improvement may be of great psychological benefit[83,84,85,86,87]. The timing of such procedures is best decided in a multidisciplinary team.

Prognosis

True congenital facial palsy has a relatively poor prognosis, owing to underdevelopment of crucial structures. Conversely, perinatally-acquired traumatic palsy has a good prognosis, with nearly all studied patients showing some improvement over time[88].

Most studies have investigated the prognosis of idiopathic palsy, and observational studies of children have been conducted. Overall, the current literature finds that the majority of children recover with minimal or no dysfunction, and that likelihood of complete recovery correlates with severity, with partial/incomplete palsies more likely to recover to normal function than dense/complete palsies. Typically, improvement is seen within six months[88,89,90,91]. The largest cohort of children studied to date was 463, within an overall cohort of 2570 in the 25-year Copenhagen study; 90% of children recovered full facial function[88].

Prognosis is generally more favourable if some recovery is seen within 21 days of onset[92]. If no improvement has been seen within 4 months, a diagnosis of idiopathic palsy should be questioned, and confirmatory investigations arranged[93]. Severe lesions, requiring extensive axonal regrowth, are more likely to lead to axonal misdirection, and thereby synkinesis or "crocodile tears".

The House-Brackmann grading system is intended to be a shorthand for severity, and thereby provide prognostic information. Although the literature bears this out, one study (in adults) has suggested that muscle electrophysiology provides superior prognostication, particularly in more severe cases[94].

Work is underway to determine reliable prognostic thresholds based on electrophysiology. One study reports that, of those (adult) patients with CMAPs >30% of normal, 84% recovered normal facial function[95]. CMAPs <10% of normal are generally agreed to represent poor prognosis, although even in this group up to one-third of patients may show near-complete recovery[91]. There are no recent studies evaluating surgical decompression; those conducted 30–40 years ago agreed that surgical intervention was unlikely to alter prognosis, and carried significant risks[91,95,96].

Recurrence of idiopathic palsy is unusual in all age groups, and should provoke further investigation, including imaging and serial blood pressure measurement[51]. One series reported a 6% recurrence rate (11 of 182 children), of which two cases were associated with Melkersson-Rosenthal syndrome[97].

Key points

- Idiopathic (Bell's) palsy is the most common cause of acquired facial palsy in childhood.
- Other important causes of acquired palsy include hypertension and Lyme disease.
- The anatomy of the childhood facial nerve accounts for its vulnerability during birth trauma.
- Imaging and electrophysiology are the most valuable investigations.
- The prognosis of idiopathic palsy in childhood is generally good.
- Treatment involves supportive measures, particularly eye care, oral steroid therapy and possibly antiviral therapy. Most evidence for this comes from studies conducted in adults.

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