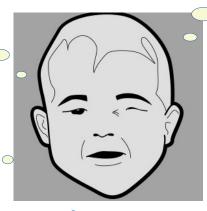
Facial Paralysis in Children

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Not as common As in adults

Has better Prognosis In children Than in adults Bells palsy is the most common etiology of facial palsy in children



May's study on etiology of Facial palsy in children: Bell's palsy – 42% Trauma – 21% Infections – 13% Congenital – 8% Neoplastm – 2%

Embryology & Applied anatomy of Facial nerve

Otic placode forms the Otocyst giving rise To membranous Labyrinth In the 4th Week facial nerve

Becomes distinct In the third week
Of gestation
Facio acoustic
Crest is visible on the
Dorsolateral aspect of the
Hindbrain just cranial to
The otic placode

Geniculate
Ganglion
Forms by 5th week



Malformations of Branchial arches Are associated with Anomalies of chorda

The facial nerve
Nucleus is formed
By neuroblasts
In the pons with the
6th nerve nucles in close
proximity

Pathology In pons will involve both 6th and 7th nerves

As the brain develops and the pons expands, the 6th nerve nucleus ascends so that the facial nerve fibers will have to whirl round the 6th nerve nucleus forming an internal genu

The facial nerve divides into its main Trunk, descending into the second Branchial arch and the chorda tympani, Which is the pretramatic branch. This Nerve curves cranially into the first Branchial arch

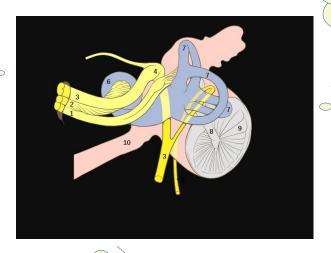
A pretrematic branch of the cranial nerve Is one that supplies the arch preceding The arch to which the cranial nerve Belongs.

The chorda tympani and main trunk of The facial nerve are equal in size during The 5th week of gestation.

In Mobeus syndrome there is agenesis of the facial nucleus along with agenesis of 6th nucleus

Embryology of Geniculate ganglion

Separate Origin from That of 7th nerve



Well defined by 7th week of gestation

Facial nerve
Anamolies should
Be anticipated during
surgery

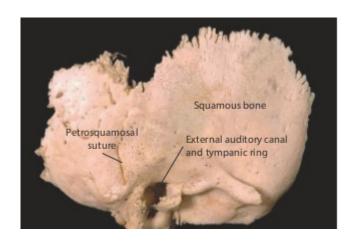
Malformations of the 1st and 2nd arch such As Treacher Collins And Goldenhar syndrome Will have facial nerve Abnormalities also

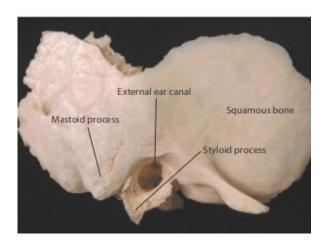
Geniculate ganglion is seen in The 7th week.

It gives rise to sensory roots that Courses through the nervus Intermedius.

As the main facial trunk descends
Down the second arch there is
Caudal movement of the 1st arch
Due to rapid expansion, producing
Horizontal segment of facial nerve,
The first and second genu of the
Vertical nerve with greater superficial
Petrosal nerve acting as an anchor

Differences between adult / infant temporal Bones





Infant temporal bone

Adult temporal bone

At birth infant temporal bone does not have mastoid process. It has just an incomplete Tympanic ring. The U shaped tympanic ring has nodular prominences. On each arm, which seperate the annulus from the future. External canal and the foramen of Huschke.

By the end of 1st post natal year these processes fuse, lengthening the canal. The foramen usually closes sometime later. The chorda tympani and the facial nerve may exit through the stylomastoid foramen in the new born. The mastoid process and external canal are undeveloped so the nerve is very superficial. The mastoid process develops and reaches adult proportions by the age of 12 years. In neonates and small children the second genu of the facial nerve is more acute and courses more laterally. The most common variation in the course of the facial nerve canal involves the tympanic segment. The bony wall may be dehiscent in 50% of population particularly above the oval window.

ASOM inneonates / children may present with facial palsy from neuropraxia or bacterial infiltration of the nerve sheath within the enclosed middle ear cavity. Dehiscence of this segment of facial nerve may be associated with a persistent stapedial artery in its course from the tympanic cavity to the middle cranial fossa where it becomes the middle meningeal artery. Foramen spinosum is absent on the side of the persistent stapedial artery as seen in CT images.

On leaving the stylomastoid foramen the facial nerve enters the parotid gland in a more anterior location than in the adult, as the parotid gland is smaller and more anteriorly placed. The nerve divides into two main divisions and these give rise to branches that supply the face and the upper neck muscles. Parotid surgery in children should take these anatomical factors into consideration.

History

Child with facial Paralysis may Not complain At all

Detailed clinical
Assessment is
Important. The muscle
Tone could be good and
Paralysis could become
Evident only when the
Child cries



Paying attention
To mother's
Story will help

History of Sucking Difficulty Should be sought

Clinical Examination

Complete & Incomplete Paralysis should Be differentiated

Otoscopy should be
Performed and
Inflammation
Of ear
Drum is commonly seen

Careful assessment Of each branch of Facial nerve should Be done

Nasolabial fold Is obliterated. Sagging of cheek



Loss of forehead wrinkles & drooping of eyebrow are features of LMN 7th nerve palsy



Lower eye lid
Falls away from the
Globe causing tears
To collect in the
Eye and spilling
To the face

Craniofacial anomalies and Involvment of other cranial Nerves to be noted

Speech changes in facial palsy: Plosives are distorted with air blowing out on the paralysed side

House Brackmann grading of 7th nerve Paralysis

Grade	Description	Gross function	Resting Appearance	Dynamic appearance		
				Forehead movement	Eye closure	Mouth asymmetry
1	Normal	Normal	Normal	Normal	Normal	Normal
2		Asymmetry; slight	Normal	Slight asymmetry	Complete (minimal effort)	Mild
		Synkinesis; Mild				
		Asymmetry; obvious	Normal	Slight movement	Complete (with effort)	Mild
3		Synkinesis; obvious				
4		Asymmetry; disfiguring	Normal	None	Incomplete	
		Synkinesis; obvious				Obvious
5	Severe	Movement; barely	Asymmetric	None	Incomplete	Obvious (slight nasal movement)
6	Total paralysis	Movement; None	Asymmetric	None	Incomplete	Obvious

Despite its limitiations House Brackmann scale is commonly used to grade facialy palsy in children.

Imaging

CT is useful for identifying bony abnormalities of the intratemporal facial nerve, which can occur with congenital malformations, trauma, and cholesteatoma. MRI is useful for identifying soft tissue abnormalities around facial nerve, as seen in inflammatory disorders, neoplasms and hemifacial spasm.

Imaging plays a Critical role in Evaluation of facial Nerve disorders

MRI can differentiate
Masses around facial
Nerve that require
Excision from those
That should not be
Excised until facial
Function is affected



Hemangiomas
Occur in the
Geniculate
Ganglion and
Show contrast
Enhancement
In MRI

When gadolinum contast is used in MRI a normal facial nerve faintly enhances in the geniculate ganglion, tympanic and mastoid segments. The cisternal, intracanalicular, labyrinthine, and parotid segments of facial nerve dont normally enhance. Enhancement of facial nerve in these regions should raise suspicion of inflammatory or neoplastic processes. Asymmetric enhancement / thickening of tympanic and mastoid segments relative to the contralateral side should be considered normal

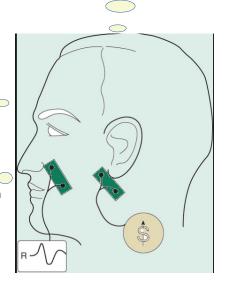
Patients with hemifacial spasm, a loop of the anterior inferior cerebellar artery, PICA, or vertebral Artery compresses the ipsilateral facial nerve at the root exit zone causing involuntary contractions of facial musculature.

Ultrasound – has been used to predict facial nerve outcomes in Bell's palsy. Facial nerve diameter is measured proximally at the stylomastoid foramen, distally just proximal to the pes anserinus, and midway between these two points. The average diameter is calculated using these three measurements and is compared with blink reflex studies and nerve conduction studies.

Electrophysiological tests

Electrophysiological tests Allow objective assessment Of facial nerve function

This test can be Performed in almost All children



This test is not An absolute Predictor for Return of function

Waveform amplitude And morphology Are consistent With that of adult Values except In infants

Shambaugh recommends muscle biopsy in neonates with facial palsy when the electromyography is silent. If muscle is found early, reanimation is advisable.

Congenial Facial Paralysis

Syndromic and nonsyndromic forms of developmental facial paralysis occur. It could be unilateral / bilateral, complete, or incomplete. Prognosis is usually poor in these patients. Craniofacial anomalies associated with the first and second arch derivatives are common in this form of facial paralysis.

Nerve exploration is unrewarding. Reannimation may be considered. The most desired neural tissue source for rejuvenation of paralysed face is direct anastomosis or interpositional grafting.

Other cranial nerves can also be involved. They include abducens nerve, hypoglossal nerve, oculomotor nerve, and trochlear nerve. The nerve least likely to be involved is the accessory nerve. Hence this nerve can be a reliable donor for reannimation procedures.

Congenital facial paralysis is uncommon And when present may cause Problems in newborn. Seen in 8 – 14% Of all pediatric cases of facial paralysis. Developmental causes include those Associated with syndromes and teratogens

Classification: Traumatic Developmental Unilatera / Bilateral Complete / Incomplete

Moebius Syndrome

Absence / under development Of 6th and 7th cranial nerves. It May be unilateral / bilateral. Autism and MR may be seen in A third of patients

Very rare cause of Facial paralysis in neonates

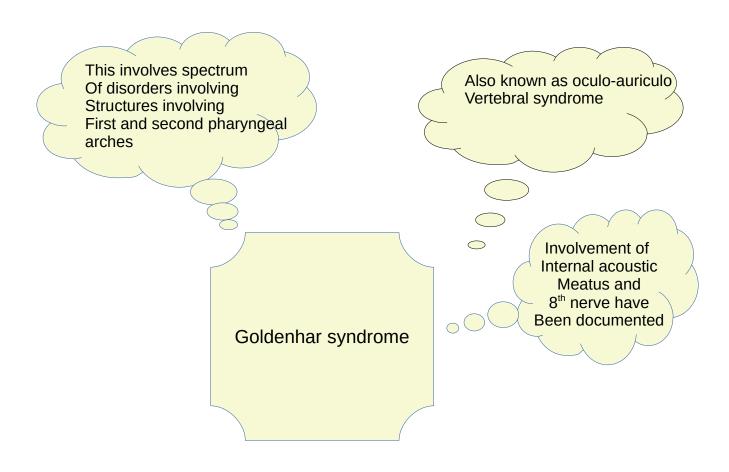
Moebius Syndrome

This disease is not progressive. These children have club foot. Brachial deformities / pectoral muscle hypoplasia are also seen.

Poland sequence – characterised by ipsilateral hand malformations and by partial / complete absence of pectoralis muscles and breast is seen concurrent with Mobius syndrome. This is seen in 15% of these patients.

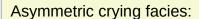
Congenital facial
Paralysis with abnormal ocular abduction

Goldenhar syndrome



- 1. Unilateral craniofacial microsomia
- 2. Lateral facial dysplasia
- 3. Otomandibular dysostosis
 4. 1st & 2nd arch syndrome
- 5. Vertebral anomalies

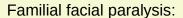
Other Syndromes



Congenital asymmetric
Crying facies is uncommon
And is caused by congenital
Hypoplasia / agenesis of
Depressor anguli oris muscle
On one side of the mouth.
May be associated wtih:
CVS, head and neck,
Musculoskeletal, respiratory,
Gastrointestinal, CNS and
Genitourinary abnormalities

CHARGE Syndrome:

- 1. Colobomata
- 2. Heart defects
- 3. Choanal atresia
- 4. MR
- 5. Genital hypoplasia
- 6. Ear anomalies
- 7. Hearing loss
- 8. Facial nerve dysfunction
- 9. Feeding & swalloing difficulties



This has been reported In three male members From three generations In a family. The paralysis Becomes more pronounced With every successive generation Widening of facial Canal:

Widening of facial
Canal has been
Reported as a cause
Of multiple ipsilateral
Facial palsy in a child.
Childhood recurrent
Fever+ thickened facial
Nerve and widened canal.
This is due to pressure
From inflammation & oedema

Acquired facial paralysis



Acute otitis media

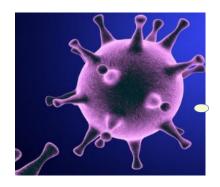


Chronic otitis media



Infection caused by Borrelia burgdorferi. 14 days incubation uni/bilateral facial palsy

Elisa is diagnostic Doxicycline is the choice Most common cause of Facial palsy in US children



Ramsay Hunt syndrome Herpes zoster

Trauma Temporal bone

Birth trauma
Is known to
Case facial
Palsy. Common
In forceps delivery

latrogenic injuries during Mastoid and parotid Surgeries can cause Facial palsy

Bruising on The side of Facial palsy Is an indicator



Fracture can be longitudinal / transverse. Longitudinal fractures cause conductive hearing loss while transverse fractures cause sensorineural hearing loss. A third of fractures are transverse and are associated with facial palsy. Longitudinal fractures rarely cause facial paralysis. In head injury sudden deceleration creates a shearing force on the facial nerve causing paralysis. Hemotympanum is common in these patients. CSF otorrhoea may also be present.