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Paediatric visage paralysis: A comprehensive examination and perspectives on treatment strategies

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Abstract
The objective of this article is to present a comprehensive examination of paediatric facial paralysis, encompassing its causes, prevalence, evaluation, diagnostic procedures, and potential treatment modalities. Facial paralysis refers to the condition when an individual has an inability to initiate the contraction of facial muscles, hence resulting in the inability to exhibit facial expressions. In general, the annual incidence rates of this phenomenon are 2.7 per 100,000 children under the age of 10 and 10.1 per 100,000 children aged 10 and above. Facial paralysis can be attributed to several etiological factors, leading to diverse results and necessitating varying treatment approaches. Corticosteroids and face treatment are commonly employed in medical care. However, in cases where facial palsy continues, the consideration of facial deformity surgery arises as a potential intervention to enhance facial symmetry, safeguard vision, and restore dynamic mobility.

Keywords: Facial, paralysis, congenital, treatment, procedure

Introduction
Face paralysis refers to the physiological incapacity to initiate the contraction of face muscles involved in expressing emotions and conveying nonverbal cues. The condition exhibits significant variety. The influence on physiological and psychosocial well-being is a common characteristic observed in all instances of facial paralysis (Fattah *et al*., 2012) [1]. Patients may exhibit a range of challenges in the areas of eating, speaking, pronunciation, and the capacity to safeguard or moisturize the cornea. The capacity to convey emotions through facial expressions plays a crucial role in facilitating social interactions. The absence of the ability to express such emotional states can result in a lack of alignment between the internal experiences of an individual and the external perceptions of an observer. This discrepancy has the potential to negatively affect one's overall well-being and contribute to social and psychological detachment. The aforementioned characteristics may exhibit heightened prominence in instances when facial paralysis manifests during early stages of development. Hence, it is crucial to address facial paralysis throughout childhood. The objective of this study is to provide a comprehensive overview of the epidemiology and genesis of facial nerve palsy, as well as to identify the key priorities in its assessment, potential investigations, and available treatment options.

Aetiology and Epidemiology
The occurrence rate of facial nerve palsy in children below the age of 10 is 2.7 cases per 100,000 individuals, whereas in children aged 10 and above, the rate is 10.1 cases per 100,000 individuals on a yearly basis (Lorch & Teach, 2012) [10]. The aetiology of facial nerve palsy may be categorized into two main groups: congenital and acquired, as outlined in Table 1 (Özkale *et al*., 2015) [3]. The aetiology remains unidentified in 50% of instances (Ciorba *et al*., 2015) [4]. Bell's palsy is the medical term used to describe idiopathic facial nerve palsy. Congenital facial palsy can arise as a consequence of obstetric difficulties that give rise to damage and/or developmental abnormalities. Unilateral facial palsy is the most prevalent manifestation in these cases. The user's text is too short to be rewritten in an academic manner. Congenital facial nerve palsy is mostly attributed to perinatal trauma, which represents the most prevalent cause.

Nevertheless, it is important to note that these cases often exhibit positive prognoses, since the underlying cause is typically neuropraxia, leading to complete restoration of facial nerve function (Pavlou *et al*., 2011) [5]. The cranial nerve known as the face nerve is closely related with the second brachial arch (Sataloff & Selber, 2003) [6]. Consequently, the presence of facial palsy can be observed in congenital anomalies of the first and second cleft, such as hemifacial macrosomia (Bentz *et al*., 2017) [7]. Additional examples of congenital syndromic malformations encompass syringobulbia, which presents clinically as dysphagia, speech impairments, and facial paralysis, as well as Arnold-Chiari syndrome, characterized by the herniation of the brainstem through the foramen magnum due to posterior fossa malformation, resulting in subsequent impairment of cranial nerves (Özkale *et al*., 2015; Pavlou *et al*., 2011) [3, 5]. Bilateral facial nerve paralysis, while uncommon, is predominantly observed in individuals diagnosed with Moebius syndrome. The condition being described is a kind of facial nerve palsy that is present at birth, and it is accompanied by paralysis of the 6th, 9th, 10th, and 12th cranial nerves (Bentz *et al*., 2017) [7]. The incidence rate of this particular illness is estimated to be 1 in every 150,000 live births, and its aetiology remains unidentified (Finsterer, 2008) [8]. The prevalence of acquired facial paralysis is lower compared to congenital causes of facial paralysis among the paediatric population. The clinical manifestation of acquired facial paralysis varies depending on the underlying aetiology. The etiological factors of infection encompass viral pathogens such as adenovirus, varicella zoster (with or without the co-occurrence of Ramsay-Hunt syndrome), Epstein-Barr virus, TB, and Lyme disease (Özkale *et al*., 2015; Sataloff & Selber, 2003; Finsterer, 2008) [3, 6, 8]. Bilateral facial palsy can occur as a consequence of blunt trauma to the cranial base, although isolated damage to a specific branch of the facial nerve is typically observed in cases of penetrating injury (Bentz *et al*., 2017; Ross *et al*., 1996) [7, 10]. Facial nerve paralysis, whether unilateral or bilateral, can be attributed to the presence of intracranial or extracranial tumours. Specifically, the occurrence of unilateral facial palsy is commonly associated with schwannomas affecting the seventh cranial nerve. Conversely, bilateral facial paralysis is often observed in cases where medulloblastomas are present. Iatrogenic causes encompass surgical procedures performed on the parotid gland or middle ear. Facial paralysis can also occur as a result of several medical illnesses, such as Kawasaki disease, leukaemia, and Henoch-Schonlein purpura, among others, as indicated in Table 1 below (Bentz *et al*., 2017; Özkale *et al*., 2015) [7, 3]

Table 1: Know cause of congenital and acquired facial nerve palsy

Table 1 Known causes of congenital and acquired facial nerve palsy

Congenital	Acquired
Syndromic	Idiopathic
• Moebius syndrome	• Bell's palsy
• Goldenhar syndrome	Infectious
• Syringobulbia	• Ramsay–Hunt syndrome
• Arnold–Chiari malformation	• Epstein–Barr virus
Delivery trauma	• *Haemophilus influenzae*
• Primiparity	• Tuberculosis
• Large birthweight	• Lyme disease
• Forceps delivery	• Cytomegalovirus
• Prematurity	• Adenovirus
Genetic	• Rubella
• Hereditary myopathies	• Mumps
• 3q21-22 mutation	• *Mycoplasma pneumoniae*
• 10q21.3-22.1 mutation	• Human immunodeficiency virus
	• Acute otitis media
	• Cholesteatoma
	Inflammatory
	• Henoch–Schonlein purpura
	• Kawasaki disease
	Neoplastic
	• Facial Nerve schwannoma
	• Haemangiomas
	• Rhabdomyosarcoma
	• Parotid gland tumours
	Traumatic
	• Temporal bone fracture
	• Iatrogenic

Assessment

Anatomy

A comprehensive knowledge of the anatomical structure of the facial nerve, originating from the precentral gyrus and converging in the facial nucleus located in the dorsolateral pons, traversing the fallopian canal within the petrous temporal bone through the internal auditory meatus, and exiting by way of the stylomastoid foramen to become the extracranial facial nerve, can facilitate the clinical assessment of facial nerve damage and determine its

severity. It is noteworthy that the frontal branch exhibits symmetrical cortical input, resulting in the preservation of frontalis function in upper motor neuron lesions, whereas its absence is observed in lower motor neuron lesions. Besides the assessment of facial musculature, the intratemporal course presents three primary branches that facilitate identification of the injury site: the greater petrosal nerve, responsible for innervating the lacrimal gland (its absence would result in dry eye); the nerve to stapedius, which if absent would cause hyperacusis; and the chorda tympani, that transmits taste from the anterior two-thirds of the tongue. The onset of such symptoms would suggest that the facial paralysis may be attributed to an intracranial aetiology.

History

Based on the patient's medical background, it is imperative for the healthcare professional to ascertain the aetiology, describe the anatomical aspects, identify the branches that are impacted, assess the functional impairment, and outline the progression of the disease. The selection of intervention is influenced by each constituent of the historical context (Bentz *et al*., 2017; Joseph & Kim, 2008) [7, 9]

Examination

The evaluation of the extracranial facial nerve should be conducted in accordance with the aesthetic components of the face, as depicted in Figure 1, as well as the terminal branches, namely the temporal, zygomatic, buccal, marginal mandibular, and cervical branches. By conducting a comparison between the afflicted and non-affected sides, clinicians are able to discern the spatial orientation of anatomical structures, with particular emphasis on identifying undesired muscular contractions on the side affected (Known as synkinesis) as well as compensatory contractions on the contralateral side. Commencing with the frontal region, meticulous observation is directed towards the manifestation of furrows and the downward displacement of the eyebrow. The evaluation of the middle third of the face primarily focuses on the dynamics of eye closure and the alignment of the upper and lower eyelids. Additionally, this assessment include the assessment of nasal airflow during inhalation. The assessment of the bottom third of the face involves the evaluation of the nasolabial fold's location and depth, as well as the position of the oral commissure both at rest as well as while smiling. The House–Brackmann grading scale and the Sunnybrook facial grading system are often employed assessment methods for evaluating the condition of the facial nerve (Ross *et al*., 1996; Neely *et al*., 2010) [10, 11] The Sunnybrook face grading method encompasses three domains of assessment, using five standardized facial expressions. In addition to its user-friendly interface, empirical research has indicated favorable levels of inter-rater and intra-rater dependability (Neely *et al*., 2010) [11]. The grading system being recommended is that of the writers.

Investigation

The utilization of electrophysiology in the evaluation of paediatric patients is constrained in its scope. Congenital factors might result in a lack of response and frequently preclude the possibility of healing in this particular group. It has the potential to be a valuable supplementary tool in evaluating the effectiveness of rehabilitation after surgical procedures (Bentz *et al*., 2017) [7]. The utilization of cross-sectional imaging techniques, such as computed tomography and magnetic resonance imaging, proves to be advantageous in cases when facial palsy exhibits no indications of improvement. These imaging modalities aid in the investigation of the underlying factors contributing to the occurrence of facial palsy.

Non-surgical management

The management of juvenile facial nerve paralysis is contingent upon the aetiology and degree of the palsy. Facial rehabilitation has been found to be beneficial for children diagnosed with unilateral incomplete facial palsy. This therapeutic approach focuses on improving the afflicted side while simultaneously reducing the activity of the contralateral side using targeted exercises and feedback mechanisms. This approach would be ineffective in paediatric patients presenting with congenital abnormalities affecting the facial nerve. The management process is facilitated by employing a multidisciplinary team (MDT) approach, which includes professionals from several disciplines such as paediatricians, neurologists, plastic surgeons, and physiotherapists, among others.

Approximately half of facial nerve palsies observed in children are attributed to Bell's palsy. It has been shown that over 70% of children affected by this condition have a resolution within a span of three months. Consequently, the primary objective of medication therapy in these cases is to mitigate the potential long-term consequences of the disease rather than directly curing it (Biebl *et al*., 2013; Tiemstra *et al*., 2007) [12, 13].

Oral corticosteroids are frequently employed as a pharmacological intervention during the first stages of a disease. Throughout history, the use of steroids has predominantly been seen in adult populations, with a scarcity of empirical information about their usage in youngsters. The predict.org.au group has conducted the BellPIC Randomized Control Trial with the aim of examining the efficacy of steroids in paediatric patients. The findings of this study published in 2021 (Babl *et al*., 2017) [14].

Acyclovir has a crucial role in the treatment of Ramsay-Hunt syndrome. If administered within 72 hours after the commencement of the condition, 75% of patients achieve complete recovery (Tiemstra *et al*., 2007) [12]. Ensuring the preservation of the cornea is of utmost importance.

In cases when facial nerve dysfunction hinders the ability to fully close the eyelid, using measures such as nocturnal eyelid taping, donning eyeglasses, and administering routine eye drops can effectively safeguard visual health. Additional non-surgical therapy alternatives encompass the administration of botulinum toxin injections. These techniques can be employed to specifically address synkinesis on the afflicted side or to achieve equilibrium on the opposite side in order to enhance overall symmetry. The therapeutic efficacy persists for a duration of 3 to 6 months, within which the implementation of ongoing physiotherapy may provide greater success. Studies have demonstrated favourable outcomes when administering botulinum toxin injections subsequent to cross-facial nerve grafting (Tiemstra *et al*., 2007) [12]. In the event that the underlying cause is a congenital deficiency of the facial nerve, it is unlikely that patients will see spontaneous recovery of function in the absence of intervention.

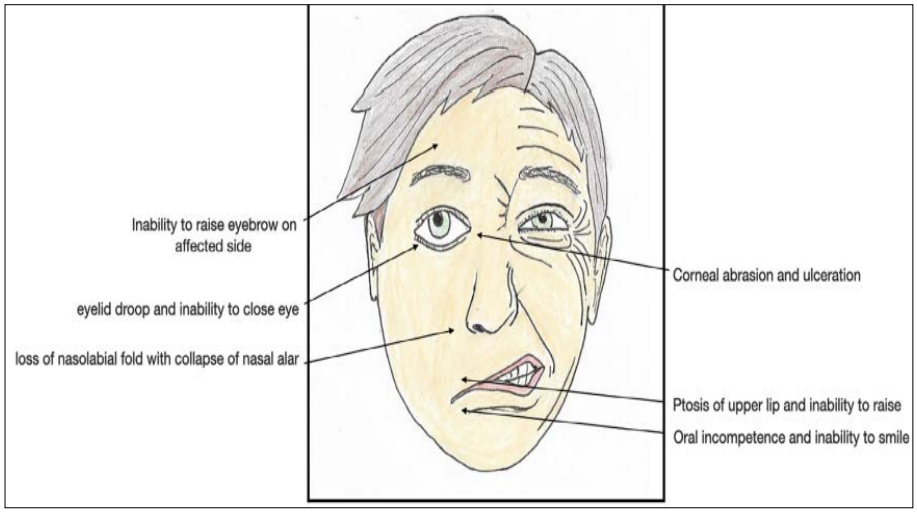

Fig 1: Diagram demonstrating clinical deficits in facial palsy and a right-sided facial nerve palsy

Individuals who experience physical damage to the cranium may undergo early observation and exhibit a progressive restoration of cognitive abilities (Bentz *et al*., 2017) [7].

The occurrence of injury to various branches of the face nerve can lead to distinct functional impairments. Injury to the frontal branch can lead to a deficiency in safeguarding the cornea, hence necessitating the implementation of gold weight insertion in the upper eyelid as a potential remedy. Damage to the buccal branch can result in the inability to exhibit a grin, and individuals with this condition may find relief by the administration of botulinum injections on the unaffected side of the face in order to achieve facial symmetry (Joseph & Kim, 2008) [9]

It is improbable for deficits persisting for a duration over 6 months to spontaneously resolve in the absence of intervention. If the duration exceeds 18 months, it is probable that the motor end plates have undergone degradation, rendering them unsuitable for primary nerve grafting (Joseph & Kim, 2008; Finsterer, 2008) [9, 8].

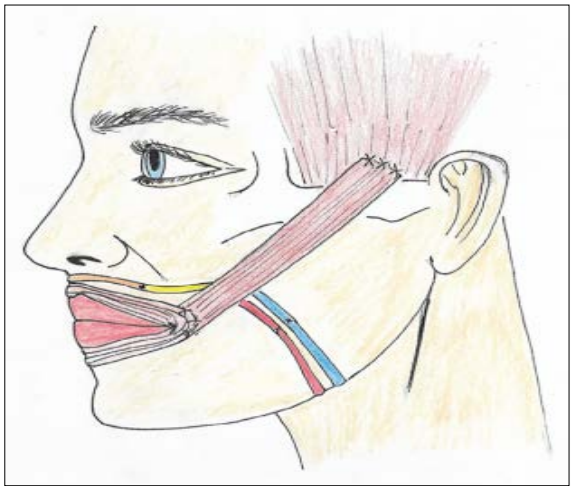

Fig 2: Free tissue transfer using gracilis muscle. Muscle pedicle anasto-mosed to facial artery and vein; innervation from cross-facial nerve graft using sural nerve

Surgical management

The treatment of facial palsy encompasses both static and dynamic procedures within the realm of surgical care. Static methods are designed to mitigate the immobilizing characteristics of illness and reinstate equilibrium in the state of repose, whereas dynamic approaches aim to reinstate both position and motion.

Static Procedures

Preserving visual acuity is of utmost importance, and in cases when traditional methods such as conservative taping and the use of eye drops prove ineffective, it is advisable to consider a surgical intervention. The coexistence of an inability to perform the blinking reflex with an ectropion condition results in compromised corneal protection for the patient (Silver *et al*., 2009) [15]. The aforementioned issues can be effectively resolved with the implementation of a gold or platinum weight in the upper eyelid, facilitating the closing of the eyelid. Additionally, the lower eyelid can be tightened or repositioned by means of a lateral or medial canthopexy procedure (Kinney *et al*., 2000) [16]. Facial paralysis has the potential to result in nasal valving, causing blockage of the nose during inhalation and thus leading to respiratory challenges. The issue at hand can be resolved by the utilization of external nasal splints, relocation of the nasal alar using bone anchors, or the implementation of fascial slings to laterally pull the side wall (Alam, 2014; Banks & Hadlock, 2014) [17, 18]. One of the most incapacitating aspects of facial palsy is the impairment of a child's capacity to produce a smile. While there are established methods available for smile restoration and addressing oral incompetence, it is important to note that these treatments are often prioritized for adult patients. Dynamic techniques are the primary approach for smile and mouth reanimation in juvenile patients (Ueda, 1998; Terzis & Olivares, 2009) [19, 20].

Dynamic procedures

In order to achieve effective dynamic reanimation, it is imperative to have three essential components: a stimulating source, a nerve, and an effector muscle. The potential choices for dynamic reanimation are contingent upon the underlying cause of the paralysis and the specific point in time when functional impairment occurred (Lohmeyer *et al*., 2007; Terzis & Olivares, 2009) [21, 20].

The reinnervation of facial muscles is feasible during a two-year timeframe following deinnervation. However, after this period, the occurrence of muscular atrophy renders such reinnervation unattainable. This may involve the implementation of a direct nerve repair technique in

instances of trauma, or the utilization of a nerve graft (such as the sural or larger auricular nerve) or conduit to establish neural continuity. Achieving complete restoration of function is improbable; but, this intervention should facilitate the return of muscle tone and activity, ultimately leading to the restoration of baseline function (Bentz *et al*., 2017) [7].

In cases where it is not feasible to restore the continuity of the facial nerve, one potential option is to perform a nerve transfer procedure. This procedure involves using either a cross-facial nerve graft (CFNG) from the facial nerve on the opposite side of the face, the hypoglossal nerve (which controls half of the tongue's function), or the masseteric nerve (which controls the muscles involved in chewing). These nerves have been found to exhibit coordinated contraction during the act of smiling (Samii & Matthies, 1994; Jensson *et al*., 2018; Yamamoto *et al*., 2007) [22, 23, 24]. However, these reconstructions entail the weakening of another muscle in order to facilitate facial muscle action. Nerve-based reconstructions possess inherent limits, particularly in instances of chronic facial palsy characterized by the absence or atrophy of the facial muscle. Consequently, other strategies must be employed to generate a smile, necessitating the utilization of novel muscular structures.

In a general sense, there are two approaches to address this issue: using local muscles, such as the lengthening temporalis myoplasty technique, or employing muscles from other regions through a free tissue transfer procedure. The technique of extending temporalis myoplasty has gained popularity due to its modification by Daniel Labbe in the procedure of temporalis myoplasty (Yamamoto *et al*., 2007) [24].

The temporalis muscle is surgically separated from its point of insertion on the mandible and point of origin on the temporal crest, and afterwards repositioned towards the corner of the mouth in order to facilitate the formation of a grin. The neurovascular supply remains unaltered, resulting in a surgical procedure duration of around 4 to 5 hours. The healing process is comparatively rapid, allowing patients to potentially regain the ability to smile within a few weeks (Labbe, 1997; Nguyen *et al*., 2020) [25, 26].

The procedure of free tissue transfer entails the relocation of a muscle from a specific region of the body. This process comprises the detachment of the muscle's blood and nerve supply, followed by its transplantation to a different spot where it is then reattached.

Various muscles have been utilized, such as the pectoralis minor and platysma; but, the gracilis muscle is the muscle most frequently employed (Harii *et al*., 1976) [27]. A free flap reconstruction procedure may be categorized as either one-stage or two-stage. In the one-stage approach, the flap nerve is directly connected to the nerve to masseter. On the other hand, the two-stage approach involves the use of a CFNG as the donor nerve (As shown in Figure 2), which is implanted during the first step of the procedure.

The surgical procedure known as free flap surgery presents considerable challenges and typically requires a full day to complete. Post-operative care should be administered with caution in order to guarantee the continuous blood supply to the flap, hence necessitating regular monitoring of the patients. The duration of reinnervation varies depending on the nerve utilized to reinnervate the free flap. Masseteric coaptation typically requires 8-10 weeks for reinnervation, while CFNG may take several additional months. Following reinnervation, the use of uncomplicated yet efficacious workouts utilizing a mirror can facilitate biofeedback and enhance the symmetry of the smile and muscular excursion.

Prognosis

The overall outlook of facial palsy depends contingent upon the underlying cause and the specific treatment administered. The provision of treatment for these patients within a multidisciplinary team (MDT) context facilitates the delivery of optimal care, offering possibilities for physiotherapy, botulinum toxin injections, or surgical interventions as required. When dynamic surgery is required, the optimization of outcomes is achieved by the implementation of physiotherapy, smile relearning, and patient compliance.

Conclusion

Paediatric facial palsy constitutes a multifaceted illness that needs careful examination and analysis. It frequently results in considerable distress for both the sufferer and the parent. Thorough evaluation of the underlying causes and functional impairment is essential in order to determine the optimal therapy modalities that are now accessible. As previously said, it is advisable to use a multidisciplinary team (MDT) strategy in the management of this intricate condition. This approach should involve the collaboration of many professionals including paediatricians, plastic surgeons, face rehabilitation experts, and social workers. By doing so, it will be possible to address both the physical and psychological ramifications associated with this disease.

Conflict of Interest

Not available

Financial Support

Not available

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