Cultural Article

Prognosis and Chances of Recovery in Bell's Palsy

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Abstract

The Bell's palsy was firstly described about two century ago by the neuroanatomist Charles Bell. This paralysis affects the VII cranial nerve and, up to date, the aetiology of the disease appears to be multifactorial. In the present manuscript, focusing on the anatomical structures related to the VII nerve, such as its the stapedial muscle innervation, we highlight the role of impedance testing as a helpful examination for the facial nerve function, together with its role as effective and safe prognosis in Bell's palsy by the stapedial reflex.

Keyword: Facial Nerve, Impedance Audiometry, Stapedial Reflex, Stapedium Muscle.

Introduction

It is essential to define the difference between paralysis (or plegia), understood as completely abolished clinical nerve function, and paresis, which refers to the deficit of nerve function that will be reduced, limited, but not absent. Paralysis is clinically framed as stupor if the deficient nerve function is temporary and not permanent.

Charles Bell (1774-1842), noted anatomist and brother of John Bell, founder of his school of anatomy in his home town of Edinburgh, was the first to describe the most common form of acute mono-neuropathy, the most common cause of acute unilateral facial nerve palsy.

House and Brackmann, doctors specialising in Otolaryngology in Los Angeles in 1985, were the first to classify VII cranial nerve dysfunction into six degrees of severity from a clinical point of view. Other types of classification (based on nerve conduction and electromyographic studies) followed, but this is still the most widely used classification today (Table 1).

Table 1: The House-Brackmann VII cranial nerve dysfunction classification.

Grade	Nervous Function	Symmetry at Rest	Eyes	Mouth	Forehead
I	Normal	Normal	Normal closure	Normal	Normal
II	Reduced	Normal	Easy and complete closure	Slightly asymmetrical	Slight reduction in movement
III	Moderate	Normal	Complete closure with effort	Slightly asymmetrical with effort	Slight-moderately reduced movement
IV	Moderate-Severe	Normal	Incomplete closure	Asymmetrical with maximum effort	No movement
V	Severe	Facial asymmetry	Incomplete Closure	Slight movement	No movement
VI	Paralysis	Paralysis	Paralysis	Paralysis	Paralysis

To date, the aetiology of the disease appears to be multifactorial, and there is still no clear demonstration of a single causative factor. However, there is a correlation with specific viral agents (Herpes simplex I, Epstein-Barr) that can cause upper airway infections with subsequent trophism towards the geniculate ganglion and subsequent acute ganglion neuritis. The incidence is 25-40 new cases per 100,000 inhabitants per year with no preponderance of sex, age or seasonality[1]. The only clinical factors that seem to correlate with a worse prognosis are advanced age, hypertension and diabetes, which play a fundamental role in the pathophysiology that, through a process of acute inflammation, creates a deficit in nerve function. The course is variable with *restitutio ad integrum* in 70% of cases. Cases of permanence are also due to an abnormal re-innervation leading to synkinesias, *i.e.* involuntary synchronous facial movements in unwanted areas. To date, the most accepted therapy consists of administering 1mg/kg Prednisone daily for 5-10 days combined or not with an antiviral *per os*[2].

Functional anatomy of the stapedial reflex

The stapedius muscle is the tiniest striated muscle in the human body. It consists of 1 mm of muscle that originates from the pyramidal eminence of the middle case of the eardrum and 7 mm of the tendon that inserts at the level of the stirrup (stapes) head[3]. When the muscle contracts, it displaces the stirrup from the oval window, reducing the pressure on the perilymphatic fluids, modulating the sound and favouring the transmission of lower sounds. The appropriate stimulus of activation of the so-called stapedial reflex relates to the hearing threshold of discomfort, which in humans is physiologically reached between 70 and 100 db sound wave intensity[4]. This muscle plays antagonistically with the tympanum tensor muscle, which differs from the stapedius muscle not only anatomically and functionally but also in terms of innervation and embryology (it derives from the first pharyngeal arch, is innervated by the V cranial nerve and modulates sound in favour of high frequencies by producing tension in the tympanum through the hammer handle during its contraction and thus increasing its vibration frequency)[5]. The stapedius muscle we will focus on is instead innervated by the VII cranial nerve and derives from the II pharyngeal arch.

The VIII cranial nerve is of no less importance in this process; in fact, it is fundamental as it is responsible for the transmission of the transduced sound wave at the cochlear level: it originates at the level of the retro-olivary dimple of the brainstem. It passes through the cerebellar flocculus, passes through a connective tissue sheath together with the VII cranial nerve and is accompanied for a short distance by the anterior inferior cerebellar artery (AICA). It thus reaches the level of the internal auditory canal of the petrous rocca of the temporal bone, where it supplies the two cochlear and vestibular sensory components, respectively, for the auditory function and static and dynamic balance concerning the position of the head in space. The spiral or Corti's ganglion within the cochlear bone structure represents the centre of origin of the bipolar or 'T' neurons in the auditory sensory perception of the cochlear component of our interest[6].

The tone-topical distribution of sound is immediately evident as there is an anatomical correspondence between the grave sounds, taken as an example previously in its periphery, the basal gyrus of the cochlea as the transduction centre, and the more lateral portions of the dorsal and ventral cochlear nuclei (located lateral to the

medulla oblongata of the brainstem) of afferent of the central fibres of the bipolar neurons that run along the aforementioned anatomical pathway of the VIII cranial nerve.

At this point, at the level of the medulla oblongata the cochlear nuclei also present a central modulation (remember the peripheral modulation in the middle case previously described through muscular antagonism) of the sound for its afferents with the superior colliculus, while the result of the elaboration of the sound wave proceeds through its efferences given by the ventral acoustic striae. These intermediate and dorsal acoustic striae run in an ascending manner reaching the superior olivary complex and then the nuclei of the lateral lemniscus at the level of the pons and then the inferior colliculus and the medial geniculate nucleus at the level of the midbrain until the conscious cortical sound perception in the temporal auditory area (Brodmann areas 41 and 42).

There is a direct anatomical correlation between peripheral sound modulation, V/VII/VIII cranial nerve and consequently with the function of facial expression.

We can find our anatomical focus of this correlation within the previous anatomical description at the level of the lateral lemniscus. The lateral lemniscus has further homo- and contralateral efferences of nerve fibres that pass from the medial lemniscus and the reticular formation to the motor nuclei of the V and VII cranial nerves at the level of the brainstem bridge, which then directly controls the tensor muscles of the tympani and the stapes of both ears as well as the entire facial mimicry.

The motor nucleus of the facial nerve, which with its fibres makes up the facial colliculus at the level of the floor of the fourth ventricle, has an upper and a lower portion for the corresponding facial mimic motor function of the upper and lower half-liver. While the superior portions of this nucleus are innervated bilaterally, the inferior portions are innervated only by the contralateral so that paralysis of an entire half-vein may in itself conclude clinically as peripheral and not central.

The VII cranial nerve, in its motor component, innervates the entire facial mimicry contained in the superficial aponeurotic muscle system of the head-neck, the stapedius muscle, the posterior belly of the digastric muscle and the stylohyoid muscle.

We, therefore, note that there is also a peripheral embryological correspondence between the motor component of the VII cranial nerve and its innervation, as well as with the stylohyoid process of the temporal bone.

Conclusions

Impedance audiometry test is the examination that makes it possible to study the presence of the stapedial reflex through the sending of an auditory stimulus above the threshold of discomfort that consequently activates the system described above to the point of contraction of the stapedium muscle itself to the detriment of the tensor muscle of the tympanum and varying the rigidity of the tympanum-ossicular system that the machine can record through the measurement of the pressure exerted in the middle tympanic cavity by the contraction of these two antagonist muscles[7,8].

We also know that physiologically, the recruitment of muscle fibres depends on the intensity of the impulse produced and the release of acetylcholine at the level of the neuromuscular plate. It is directly proportional to the diameter of the fibre and the speed of conduction[9]. Consequently, the fibres with lower conduction velocity and smaller axonal diameter will be recruited first.

The stapedius muscle is the smallest in the human body and has the finest innervation of all possible recruitment muscles in the motor component of the facial nerve.

We can therefore conclude, not only as anatomically demonstrated, that impedance testing can be a helpful study examination for the function of the VII cranial nerve as it is known, but also that it is possible to establish an effective and safe prognosis of recovery in Bell's palsy through the presence of the stapedial reflex.

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