Facial Palsy

ENT·Education·Gambia
"Education through Innovation"

Objectives

1. Describe the anatomy and function of the facial nerve, its branches and be able to describe its intra & extra cranial route in the body.

2. Recall the causes, clinical presentations, the management and prognosis of the various diseases leading to facial palsy.

3. Be able demonstrate a fluid and systematic examine of cranial nerve VII.

4. Differentiate between upper and lower motor neuron facial palsies

Incidence

Annual incidence of 15-30 cases per 100,000 population.
Bells palsy accounts for approximately 60-75% of cases of acute unilateral facial paralysis

Aetiology

There are many methods for formulating the differential diagnosis of a condition. One particularly useful method is using a surgical sieve (mnemonic). There are numerous examples of surgical sieves, the one we will use here is **VITAMIN D**.

V ascular	CVA / Cerebral aneurism
I diopathic / Iatrogenic	Post surgical – middle ear surgery, parotid surgery, surgery to the internal acoustic meatus
T raumatic	Fractured temporal bone / laceration to the nerve
A utoimmune	Sarcoid, Multiple sclerosis, Guillain-Barré syndrome, Melkersson-Rosenthal syndrome
M etabolic	Diabetes mellitus
I nflammatory / Infective	Acute otitis media, chronic otitis media, malignant otitis externa, Bell's palsy, Ramsay-Hunt syndrome
N eoplastic	Malignancy in the parotid, acoustic or facial neuroma
D evelopmental	Moebius syndrome, hemifacial microsomia

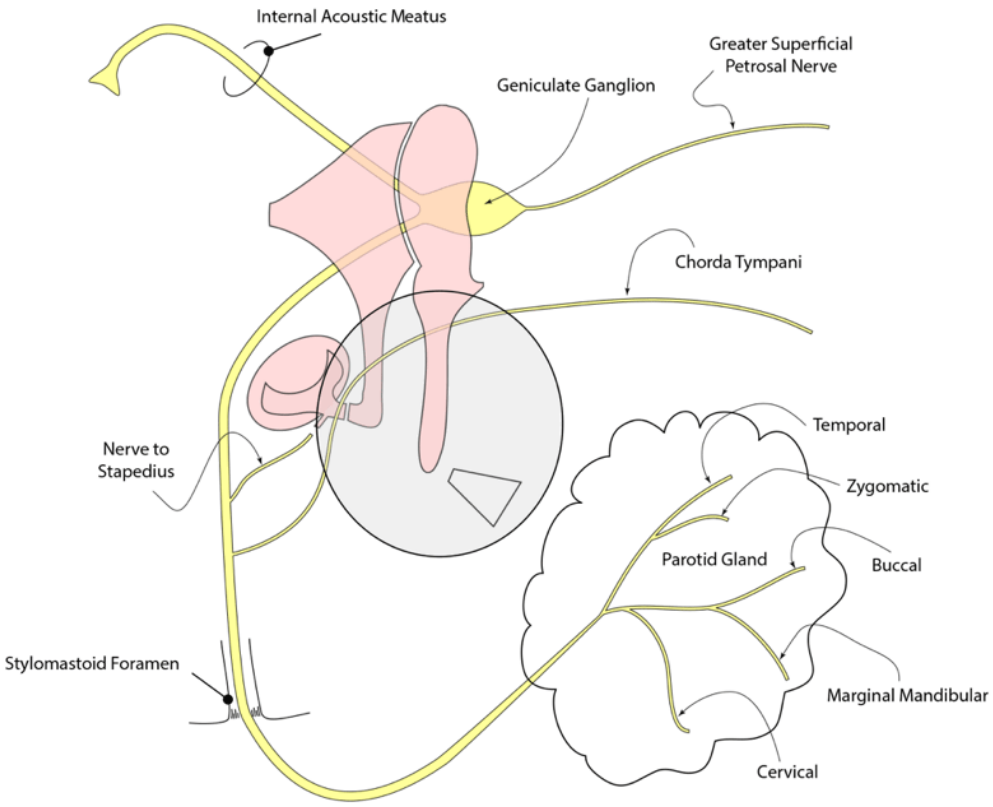
Anatomy

The facial nerve originates in the pons area of the brain. It forms from two separate nerves; a large motor nerve and smaller sensory root (the intermediate nerve). These nerves fuse to form the facial nerve after passing through the (IAM) internal auditory meatus and then pass into the facial canal.

The Geniculate ganglion is a collection of nerve cell fibres and bodies. ("Genu" - "Knee" shaped) The facial canal is a Z shaped structure which enclosed the nerve as it passes through the remainder of its intracranial path.

Before leaving the skull via the stylomastoid foramen, the facial nerve gives rise to a number of branches:

Greater superficial petrosal nerve	This synapses with the sphenopalatine ganglion which provides parasympathetic nerve innervation to the lacrimal gland and the mucosal glands of the nose, palate, and pharynx.
Nerve to the stapedius	Innervates the stapedius which helps dampen excessive movement of the stapes.
Chorda Tympani	Special sense of taste from the anterior two-thirds of the tongue.



The Facial Nerve

Once out through the stylomastoid foramen nerve passes anteriorly through the parotid gland where it branches to form 5 motor branches.

A great mnemonic to remember these branches is

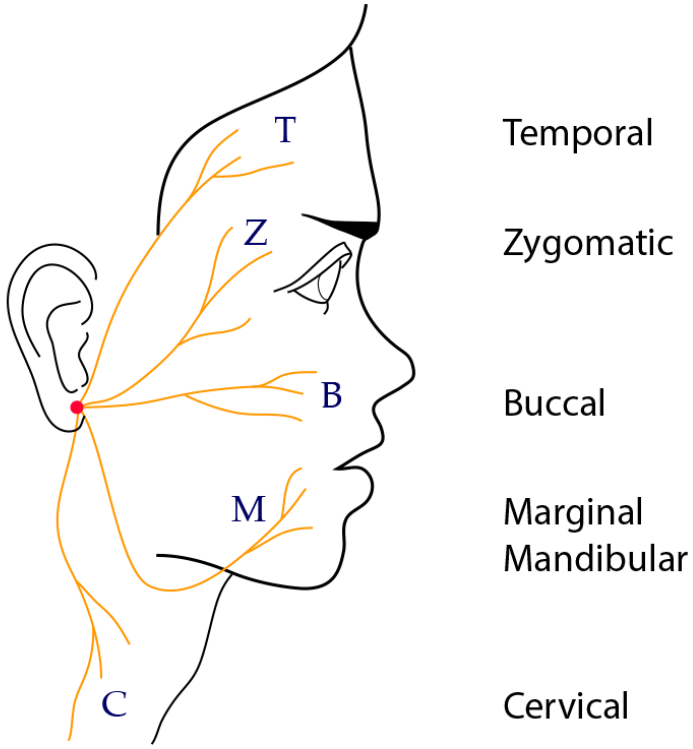
"**T**o **Z**anzibar **B**y **M**otor **C**ar"

These nerves innervate the muscles of facial expression as listed above.

It is only through a thorough knowledge of the underlying anatomy of the facial nerve that a good understanding of the importance of clinical findings and examination can be achieved.

Clinical examination

The first part of the examination is to establish if a facial nerve palsy exists! The level of weakness varies from dense weakness to very subtle. We do this by testing the 5 distributions and corresponding facial expression.



Temporal	Get the patient to raise their eyebrows.
Maxillary	Ask the patient to close their eyes tightly and keep them closed against resistance.
Buccal	Request the patient puff out their cheeks.
Marginal mandibular	Ask the patient to smile showing their teeth.
Cervical	Harder to explain without demonstrating yourself. Get the patient to grimace, showing the platysma muscles of the neck.

Upper Vs Lower motor neurone

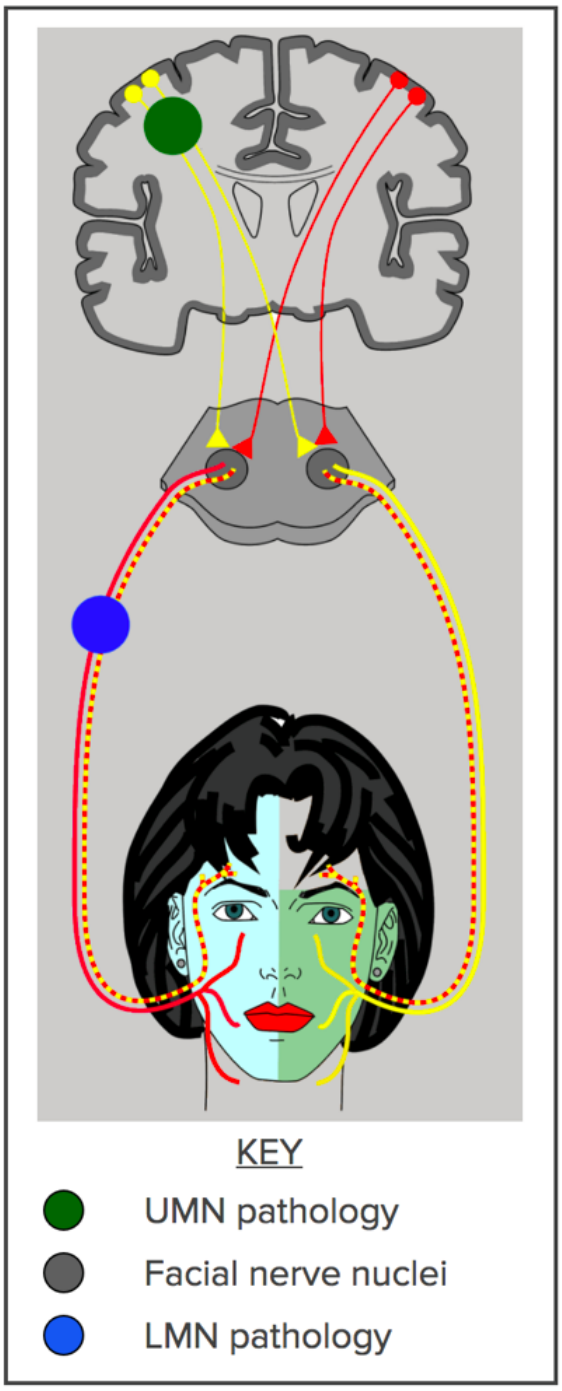
It is important to be able to distinguish an upper from a lower motor neurone weakness. Remember that muscles of the forehead have bilateral cortical representation.

Let's study the diagram opposite. Upper motor neurones from the two hemispheres of the cerebral cortex are represented with differing colours. The patient's left cortex is coloured red, the right cortex is represented by yellow.

We see that each facial nerve nuclei (grey circle) receives upper motor neurone input from both sides of the cerebral cortex (via left and right corticobulbar tract in the posterior limb of the internal capsule). As seen by the mixed red and yellow coloured lines, the temporal branch of the face receives innervation from both hemispheres. The remaining branches of the facial nerve however only have single hemisphere innervation (Single coloured lines).

The Green circle represents an UMN pathology in the right side of the patient's brain e.g. a stroke. We would expect from our neurology anatomy the left side of the patient's face to be weakened (dark green side). The stroke disrupts the yellow nervous innervation to the face (right corticobulbar tract), however the red or left side of the patient's brain still gives innervation to the upper branches of the facial nerve, through the pathway described above. This means the forehead or temporal branch of the facial nerve is spared. So we can deduce an UMN lesion clinically in a facial palsy by the sparing of the forehead muscles from weakness.

If we now take a LMN lesion (blue circle), such as a Bell's palsy. The pathology interrupts the nervous supply of the facial nerve lower down the tract taking out both the yellow (right) and red (yellow) nervous innervation to the facial muscles. As such there is ipsilateral weakness in all of the facial divisions, shown by the right side of the face. (Cyan coloured side). Therefore, in lower motor neurone pathology we expect all branches of the facial nerve to weakened.



The House-Brackmann classification

It is important to be aware of the method used to classifying the severity of a facial weakness. The table below outlines this classification. There is no need to memorize this, just be aware it exists.

Grade		Definition
I	Normal	Symmetrical function in all areas
II	Mild	Slight weakness noticeable. Able to close their eye with minimal effort
III	Moderate	Obvious weakness and may not be able to lift eyebrow
IV	Moderately severe	Obvious disfiguring weakness with inability to lift brow. Incomplete eye closure.
V	Severe	Barely perceptible motion: asymmetry at rest
VI	Total paralysis	No movement

Other areas to examine

A full and thorough examination must be conducted to ensure no pathology is missed and the correct diagnosis is made. Areas to specifically focus on are the parotid, otological examination, and a thorough neurological examination including cranial nerves.
It is also important to get a baseline audiogram to help rule out an acoustic neuroma.

Investigations

For the most part these are unnecessary as a thorough clinical examination and history will give the diagnosis. MRI is excellent for looking for tumours in the internal acoustic meatus. CT is used for temporal bone fractures and for imaging facial nerve schwannomas. Electromyography is used by some to predict the prognosis. (rare)

Management

1. Diagnose and treat the cause

2. Treat the eye

In Bell's palsy oral steroids are found to help and prevent synkinesis & neuropathic pain during recovery. Whether they improve outcome in any other way is uncertain but they are widely used. Protection of the eye is essential as corneal ulceration may occur due to the lack of protective tear film on the surface of the eye. The tear film is spread onto the cornea by blinking and blinking is affected by the palsy.

Herpes Zoster Oticus is treated with acyclovir, oral steroid and pain relief. Eye protection is also required.

Trauma to the nerve is classified below;

Neuroporaxia	Caused by local compression and is recoverable when the pressure is relieved
Axonotmesis	Axonal integrity is disrupted but endoneural sheaths preserved. Distal degeneration of the nerve occurs. Return to normal function is possible.
Neurotmesis	Destruction of axon and support cells. Significant recovery is unlikely.

In sharp trauma, such as surgical accident in the parotid gland, end-to-end suturing of the nerve is possible. Alternately, if a segment of nerve is damaged it may be replaced by a section of the greater auricular or sural nerves.

In cases of head trauma treatment will depend on the timing of the nerve palsy. If the onset is delayed this implies a neuropraxia and can be treated conservatively. If it is immediate this suggests neurotmesis and exploration of the nerve is required.

When the palsy is caused by middle ear disease then the disease must be treated. acute suppurative otitis media is treated with antibiotics and drainage (grommet insertion). Cholesteatoma is treated with mastoid exploration. Malignant otitis externa is treated with oral and topical ciproxin for several months while debridement of the nerve may be attempted.

In all of the above treatments the eye must be protected. This usually comprises of an eye patch at night and protective glasses during the day. A temporary tarsorrhaphy may be required and this is done locally with botox injection of the upper lid to induce ptosis. Other methods include a gold weight in the upper lid.

Facial reanimation may be attempted. There are a number of techniques for this including nerve grafting, neuromuscular transfer and fascial slings.