The Facial Nerve: Anatomy, Approach to Palsies & Management

The facial nerve, also known as the **seventh cranial nerve (CN VII)**, plays a critical role in both motor and sensory functions of the face. It is responsible for the movement of facial muscles, enabling expressions such as smiling, frowning, and blinking. Beyond its motor functions, the nerve also provides sensory input for taste from the anterior two-thirds of the tongue and contributes to the secretion of tears and saliva through its parasympathetic fibres.

Facial nerve palsy refers to a condition in which there is weakness or paralysis of the facial muscles due to damage or dysfunction of the nerve. This condition can arise from a variety of causes, ranging from viral infections (e.g. Bell's palsy) to trauma, tumours or neurological diseases. It can have significant physical and emotional impacts on patients, affecting both facial function and appearance.

This chapter aims to provide an in-depth review of:

1. The anatomy and functions of the facial nerve.
2. The clinical presentation and classification of facial nerve palsies.
3. Management strategies

By the end of this chapter, readers will gain an understanding of the facial nerve's importance, the implications of its dysfunction, and the principles of managing facial nerve palsies.

Anatomy:

To understand facial nerve palsies, it is important to first understand the anatomy of the nerve in order to localize the site of injury. The facial nerve has a complex course – it begins at the brainstem and travels through the temporal bone via the internal acoustic meatus (IAM) to reach the facial canal. The facial canal has 3 parts, this includes the labyrinthine, tympanic and mastoid segments. Once the facial nerve exits the temporal bone via the stylomastoid foramen, it gives rise to its external branches.

Brainstem – 4 Components:

Efferents (Located in pons):	Afferents (Located in medulla)
Branchial Motor (Motor nucleus of CN 7)	General Sensory (Spinal nucleus of trigeminal)
Visceral Motor (Superior salivatory nucleus)	Special Sensory (Gustatory nucleus)

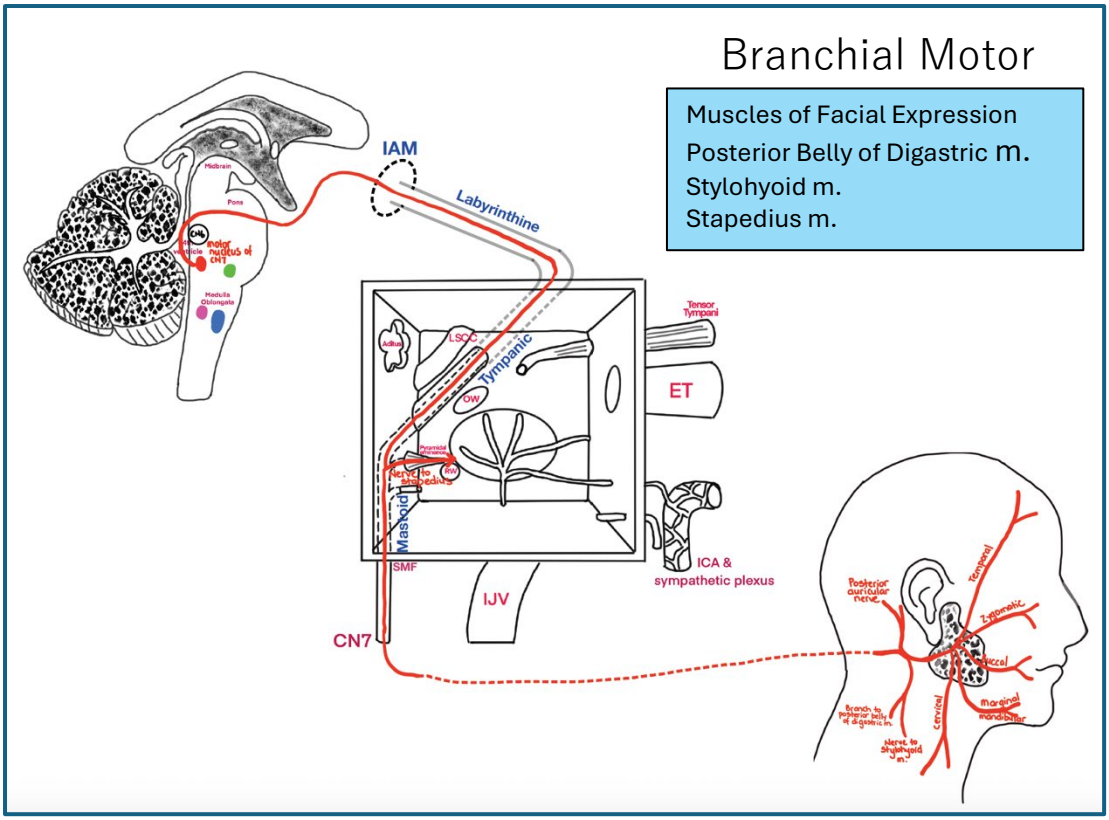
Branchial motor:

Begins at the Motor nucleus of CN 7 where it courses backwards towards the floor of the 4th ventricle and loops around the CN 6 nucleus. The significant branches that this component gives off include:

Within the temporal bone: Nerve to the stapedius muscle

External branches:

- Immediately after exiting the stylomastoid foramen: Posterior auricular nerve, Branch to posterior belly of digastric muscle & Branch to stylohyoid muscle
- Branches given off within the parotid gland: Temporal, Zygomatic, Buccal, Marginal mandibular and Cervical nerve.



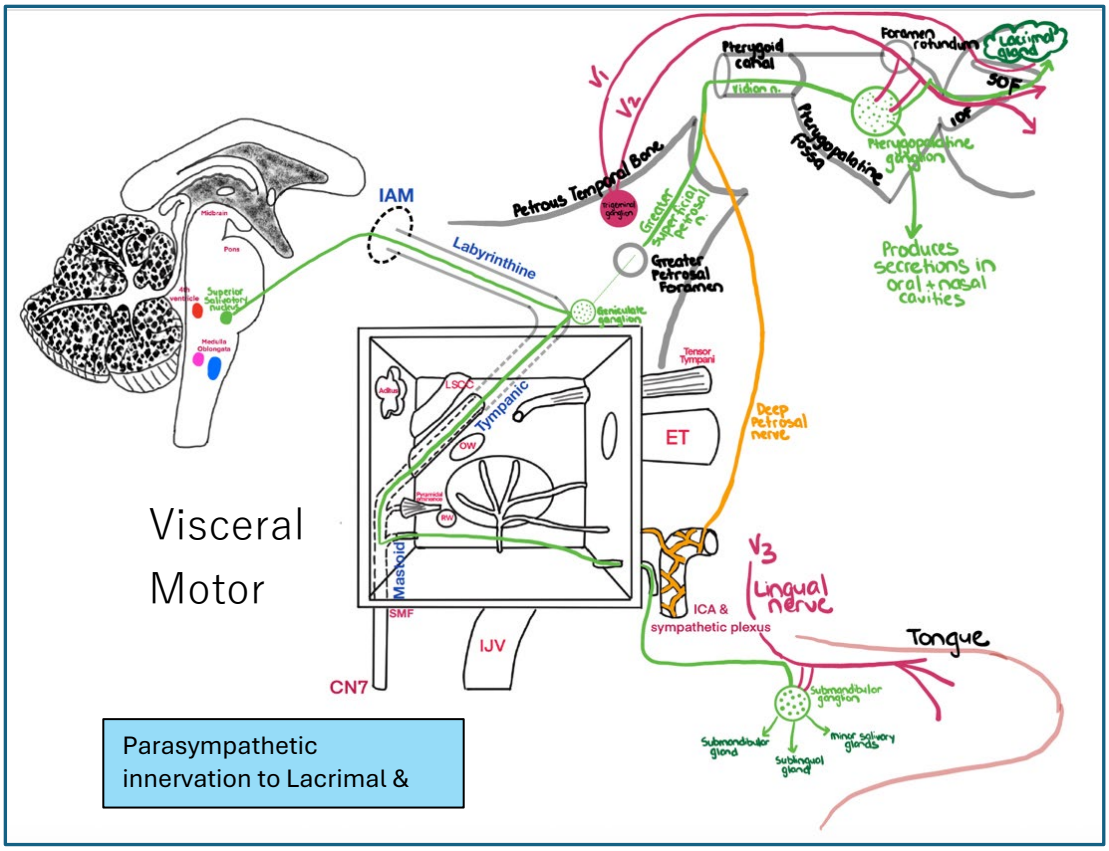
Visceral motor:

Pre-ganglionic fibres arise from the superior salivatory nucleus and travels via the IAM to reach:

1. Geniculate ganglion
2. Submandibular ganglion (via the chordae tympani nerve)

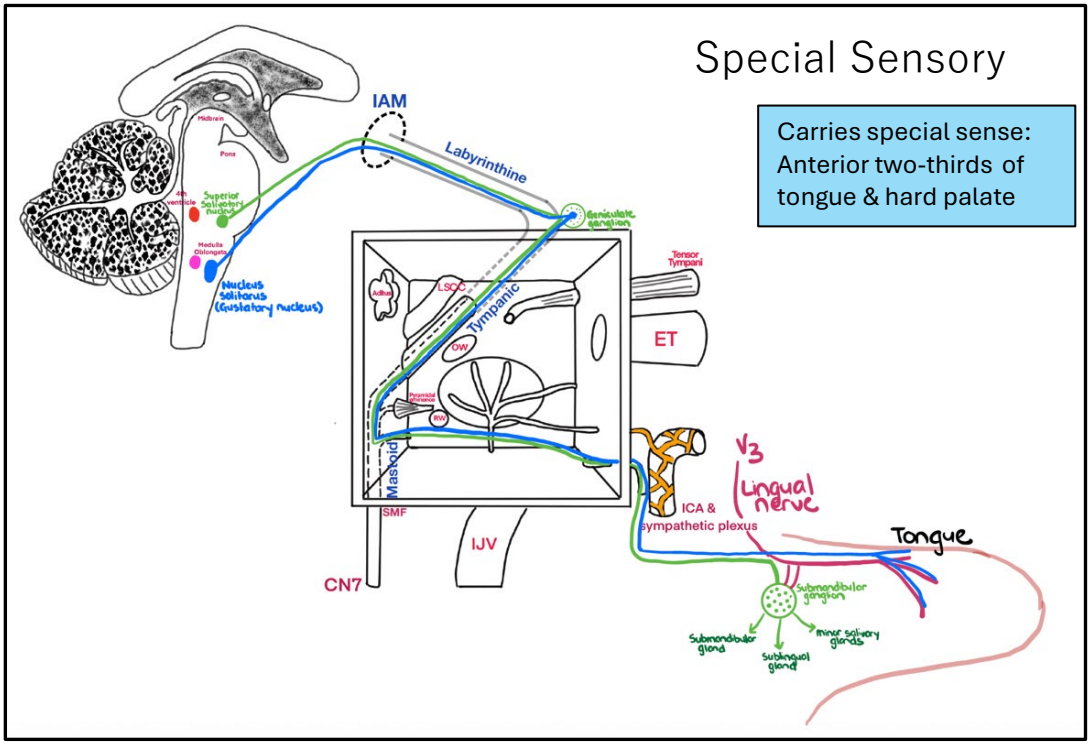
They provide parasympathetic innervation to:

1. Lacrimal gland
2. Salivary glands – this includes, the minor salivary glands of nasal and oral cavity and the submandibular and sublingual glands



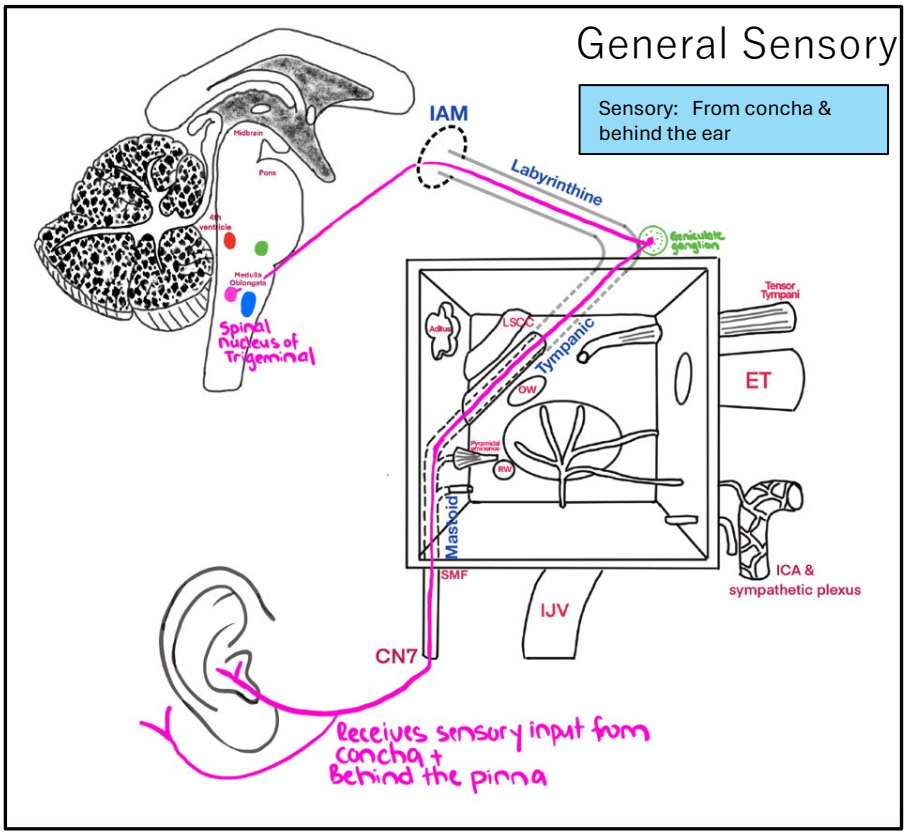
Special sensory:

Information from the taste buds in the anterior two-thirds of tongue is carried via lingual nerve, then chordae tympani towards its cell body in the geniculate ganglion. This information then travels via the IAM towards the gustatory nucleus in the medulla oblongata.



General sensory:

Sensory information from the skin of concha and behind the ear travels through the stylomastoid foramen and the facial canal to reach the geniculate ganglion, where the nerve cell body is located. From there, the information continues through the internal acoustic meatus (IAM) to the spinal nucleus of the trigeminal nerve.



Facial nerve palsies:

Facial nerve palsies can be classified as:

- **Peripheral (lower motor neuron):** Affects the entire side of the face, including the forehead.
- **Central (upper motor neuron):** Spares the forehead due to bilateral cortical innervation.

Nerve injury classification:

Nerve injuries can be classified according to Seddon or Sunderland Classification

Seddon	Sunderland	Pathology	Prognosis
Neuropraxia	Grade 1	Area of blockage of nerve conduction. May have segmental demyelination, but no axonal damage.	Complete recovery
Axonotmesis	Grade 2	There is loss of axons, however, endoneurium, perineurium and epineurium remain intact.	
	Grade 3	As above but endoneurium is also disrupted	Partial recovery
	Grade 4	As above but perineurium is also disrupted	Spontaneous nerve recovery is unlikely and will likely require surgery.
Neurotmesis	Grade 5	As above but epineurium is also disrupted – i.e. Nerve transection.	

Common causes of facial nerve palsies:

Birth	Infective	Traumatic	Neoplastic
• Forceps delivery • Dystrophia myotonica • Moebius syndrome • Facial nerve agenesis	• Otitis media – Acute or Chronic • Necrotizing otitis externa • Encephalitis • Herpes zoster oticus (Ramsay hunt syndrome) • Chicken pox • Mumps • Infectious mononucleosis/ Epstein-Barr virus • HIV • Tuberculosis • Lyme's disease	• Base of skull fractures • Facial nerve trauma – Penetrating injury to face or middle ear	• Parotid tumours • Facial nerve tumour • Glomus jugulare • Glomus tympanicum • Meningioma • Carcinoma • Haemangioblastoma • Leukaemia • Cerebral Lymphoma
Metabolic	**Idiopathic**	**Iatrogenic**	**Toxins**
• Diabetes Mellitus • Hyperthyroidism • Pregnancy • Hypertension • Acute porphyria • Vitamin A deficiency	• Bell's Palsy • Guillain-Barré syndrome • Melkerson-Rosenthal syndrome • Sarcoidosis • Granulomatosis with polyangiitis	• Middle ear & Mastoid surgery • Parotid surgery • CPA angle tumour surgery • Use of local anaesthesia for blocks • Embolization	• Tetanus • Diphtheria • Carbon monoxide • Thalidomide

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History:

1. Onset & Duration

- When did the weakness start?
- Was it sudden or gradual and is it progressing?
- Have you had similar episodes before? If yes, was it the same side that was affected last time?

2. Distribution & Symptoms

- Is the weakness affecting the entire side of the face or just part of the face?
- Have you noticed changes in taste, hearing or tearing?
- Any drooling or difficulty eating?

3. Associated Symptoms

- Any pain around the ear or face?
- Any rash or vesicles? (Suggestive of Ramsay Hunt Syndrome)
- Any associated dizziness, hearing loss or tinnitus?
- Any recent infections, fevers, or upper respiratory tract infections?
- Any trauma or recent surgery – especially around the ear or parotid gland?
- Any recent travel or tick bites? (Concern for Lyme disease in endemic areas)
- Any history of headaches or visual disturbances? (Possible neurological cause)

4. Medical History

- History of Diabetes mellitus or Hypertension? (Risk factors for ischemic cranial nerve palsies)
- Any history of strokes?
- Any autoimmune conditions? (e.g. **sarcoidosis, Guillain-Barré syndrome, myasthenia gravis**)
- History of cancer? (concern for metastatic or parotid malignancies)

Focused Examination of a patient with Facial nerve palsy:

1. Differentiate between upper motor neuron (UMN) and lower motor neuron (LMN) facial nerve palsy. Forehead sparing is generally related to an UMN facial nerve palsy.
2. Record the degree of facial weakness – Most commonly used grading system is the House-Brackmann Staging System.

House-Brackmann Staging System						
Grade	**Degree of injury**	**Function**	**Eyes**	**Mouth**	**Forehead**	**Re-innervation**
I	Normal	Normal	Normal	Normal	Normal	Normal
II	Mild	Mild weakness	Complete closure with minimal effort	Slight asymmetry with maximal effort	Reasonable function	Synkinesis barely noticeable Contracture/Spasm absent
III	Moderate	Obvious weakness, not disfiguring	Complete closure with maximal effort	Asymmetric movement with maximal effort	Slight to moderate movement	Non-disfiguring synkinesis, mass movement or spasm
IV	Moderately severe	Obvious disfiguring weakness	Incomplete closure	Asymmetric movement with maximal effort	None	Severe synkinesis, mass movement & spasm
V	Severe	Motion barely perceptible	Incomplete closure	Slight movement of corner of mouth	None	Absent
VI	Total paralysis	No movement	Total paralysis			Absent

3. Examine the head & neck and the ears
    - If patient has a partial facial nerve palsy – check for a parotid gland lesion
4. Examine for any other cranial nerve fallout
5. Check for any associated symptoms:
    - Impaired vision or iritis - ?Sarcoidosis
6. Look for secondary effects complicating facial nerve injuries: Synkinesis, Contractures, Crocodile tears, Epiphora, Dysgeusia, Pain and Hyperacusis

Investigations:

Electrophysiological tests

These tests are not done in cases of incomplete paralysis. It is done to assist with making decisions regarding facial reanimation surgery.

Options available include:

Electroneurography (ENoG)	Electromyography (EMG)
Measured between >3 days to 3 weeks	**Complementary to ENoG after 2 weeks**

Wallerian Degeneration is the process of denervation of the neural fibres, this takes about 72 hours to be completed. This is why we wait 3 days before the study is done. Two electrodes are used for this test: The **stimulation electrode** is placed near the stylomastoid foramen where the facial nerve exits the skull and the **recording electrode** is placed near the nasolabial fold to record the electrical responses of the facial nerve muscles. The response is recorded as a compound muscle action potential (CMAP) at the nasolabial fold. The test compares the amplitude of the CMAP from the affected side to the healthy side. The percentage response is calculated by dividing the amplitude of the paralysed side by the amplitude of the normal side. A low percentage degeneration (<25%) suggests a good prognosis with a high likelihood of recovery and a high percentage degeneration (>90%) suggests a poor prognosis with a reduced chance of recovery. The ENoG may be repeated at intervals of 3 to 5 days to monitor nerve recovery or deterioration. After 21 days, the predictive value of an ENoG is inaccurate as nerve regeneration and collateral nerve development will begin.	It is often used after several months of paralysis to assess muscle re-innervation. Small needles are inserted into the facial muscles – usually on the orbicularis oculi and orbicularis oris. Electric activity is measured during rest and voluntary contraction. Interpretation of results: **Reinnervation:** Muscles show polyphasic innervation potentials. **No Reinnervation:** Fibrillation potentials are seen which suggest muscles are alive but not receiving nerve signals. Can consider surgical exploration in such cases. **Chronic denervation:** Silence on EMG, thus suggesting irreversible muscle denervation

Bloods

Are not usually necessary in truly idiopathic cases. However, if one wanted to exclude less common causes can consider: ACE/ANCA/HIV/Lyme disease serology/Syphilis serology

Imaging

1. **CT Scan:**
   - HRCT Temporal bone can follow the facial nerve from the internal auditory meatus (IAM) to the stylomastoid foramen (SMF).
   - Contrasted CT Neck is useful if the pathology is intra-parotid

2. **MRI:**

An MRI can provide better soft tissue detail than a contrasted CT scan and can show enhancement of the facial nerve to suggest underlying pathology

Management:

1. Proper eye care is essential:
   - Lubrication: Use artificial tears during the day and ocular lubricants and eye tape at night
   - Eye patch
   - Other option: Upper eyelid weighting
2. Physiotherapy: Facial exercises help to relax the facial muscles and help to improve the asymmetry
3. Botox: Helps to release over-tightened facial muscle and can be used to weaken more active "normal" facial muscles in order to achieve a more balanced facial appearance

4. Facial reanimation surgery is only considered if there is a lack of recovery after 1 year. This procedure is done by Plastic surgery.
5. Psychological counselling and support groups may be useful

References:

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