TO DAYS TOPIC

FACIAL NERVE AND ITS DISORDER.

PRESENTED BY

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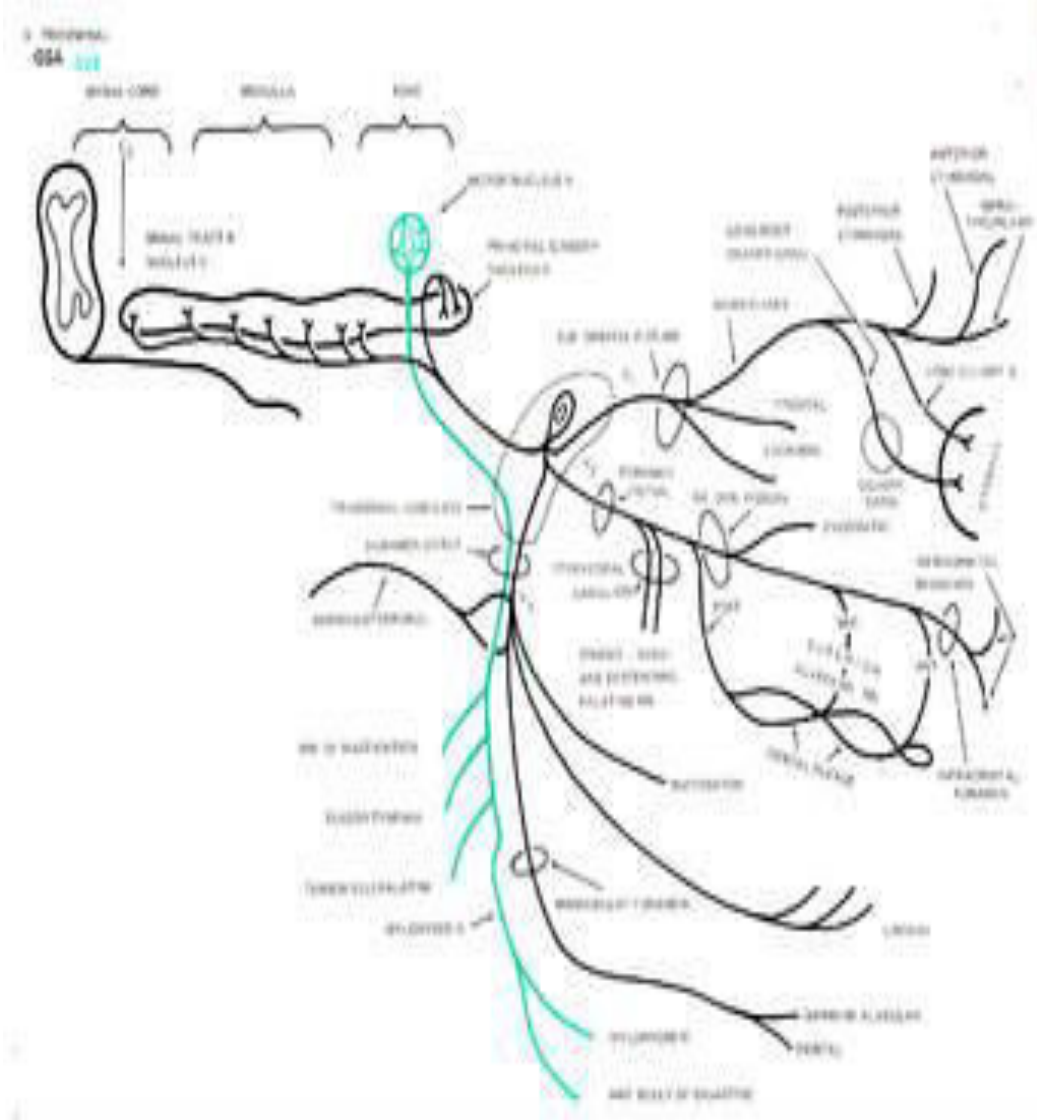
Facial nerve and its disorder

Facial nerve is a mixed nerve having motor and a sensory root.

Course of facial nerve:

- ✓ Motor fibres take origin from the nucleus of VII th nerve.
- ✓ Facial nerve leaves the brainstems at pontomedullary junction, travels through posterior cranial fossa and enters the internal acoustic meatus.



Course of the facial nerve

Course of the facial nerve can be divided into:

1. Intracranial part- from pons to internal acustic meatus.
2. Intratemporal part- from internal acoustic meatus to stylomastoid foramen. It is further divided into;

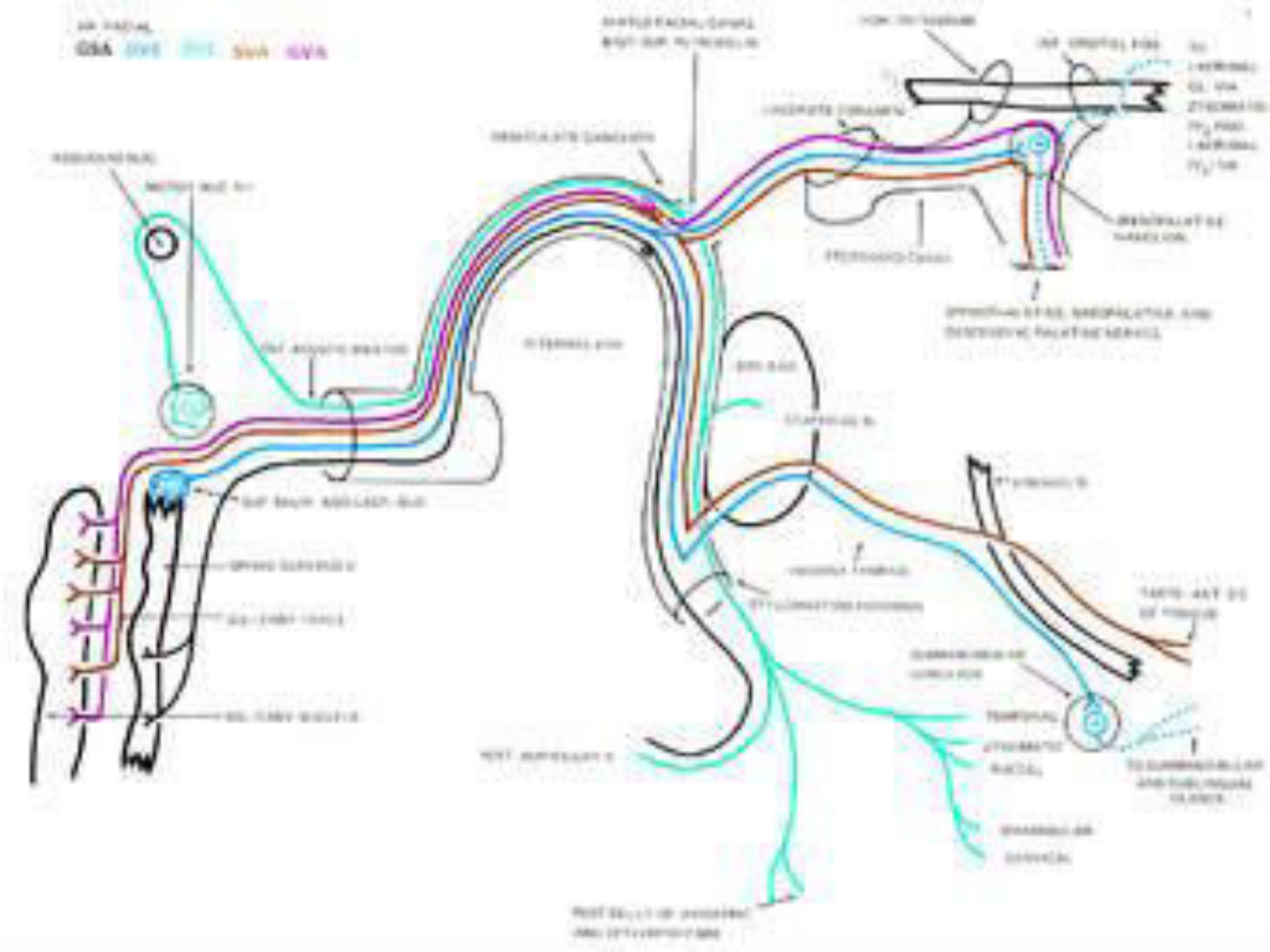
a. Meatal segment- within internal acoustic meatus 8-10 mm.

b. Labyrinthine segment-from fundus of meatus to the geniculate ganglion where nerve takes a turn posteriorly forming a genu. Only 4.0 mm..

c. Tympanic or horizontal segment- from geniculate ganglion to just above the pyramidal eminence. It lies above the oval window and below the lateral semicircular canal . 11.0 mm

d. Mastoid or vertical segment- from the pyramid to stylomastoid foramen. Between the tympanic and mastoid segment is the second genu of the nerve. 13.0 mm.

3. Extracranial part: from stylomastoid foramen to the termination of its peripheral branches.



Severity of nerve injury:

- Neuropraxia – a conduction block. Where flow of axoplasm through the axons was partially obstructed.
- Axonotmesis- injury to axons.
- Neurotmesis- injury to nerve.

Sunderland classified nerve injuries into five degrees of severity based on anatomical structure of the nerve.

- 1^0 = partial block to flow of axoplasm, no morphological changes are seen, recovery of function is comp lete (neurapraxia).
- 2^0= loss of axons but endoneural tubes remain intact.(axonotmesis).
- 3^0= injury to endoneurium. During recovery, axons of one tube can grow into another.(neurotmesis).
- 4^0= injury to perineurium in addition to above.(partial transaction).
- 5^0= injury to epineurium in addition to above (complete nerve transaction).

- The first three degrees are seen in viral and inflammatory disorders.
- Fourth and fifth degrees are seen in surgical or accidental trauma to the nerve or in neoplasm.

Electrodiagnostic tests:

- These tests are useful to differentiate between neurapraxia and degeneration of the nerve.
- Also help to predict prognosis and indicate time for surgical decompression of the nerve.

1. Minimal nerve excitability test-
2. Maximal stimulation test (MST)-
3. Electroneuronography (ENoG)-
4. Electromyography (EMG)-

Causes of facial paralysis:

- may be central or peripheral.
- Peripheral lesions may involve the nerve in its intracranial, intratemporal or extratemporal parts.
- Peripheral lesions are more common and about two-thirds of them are of the idiopathic variety

Causes of facial paralysis are(Cont.)

1. Central cause:

- Brain abscess, pontine glioma, poliomyelitis, multiple sclerosis.

2. Intracranial part (cerebellopontine angle):

- Acoustic neuroma, meningioma, congenital cholesteatoma, metastatic carcinoma, meningitis.

3. Intratemporal part:

a. Idiopathic –

- Bell's palsy
- Melkersson's syndrome.

b. Infection –

- Acute suppurative otitis media.
- Chronic suppurative otitis media.
- Herpes zoster oticus.
- Malignant otitis externa.

c. Trauma –

- Surgical : mastoidectomy , stapedectomy.
- Accidental : fractures of temporal bone

d. Neoplasms –

- Malignancies of external and middle ear.
- Glomus jugulare tumour.
- Facial nerve neuroma.
- Metastasis to temporal bone (from cancer of breast, bronchus, prostate).

4. Extracranial part:

- Malignancy of parotid.
- Surgery of parotid
- Accidental injury in parotid region.
- Neonatal facial injury (obstetrivcal forceps)

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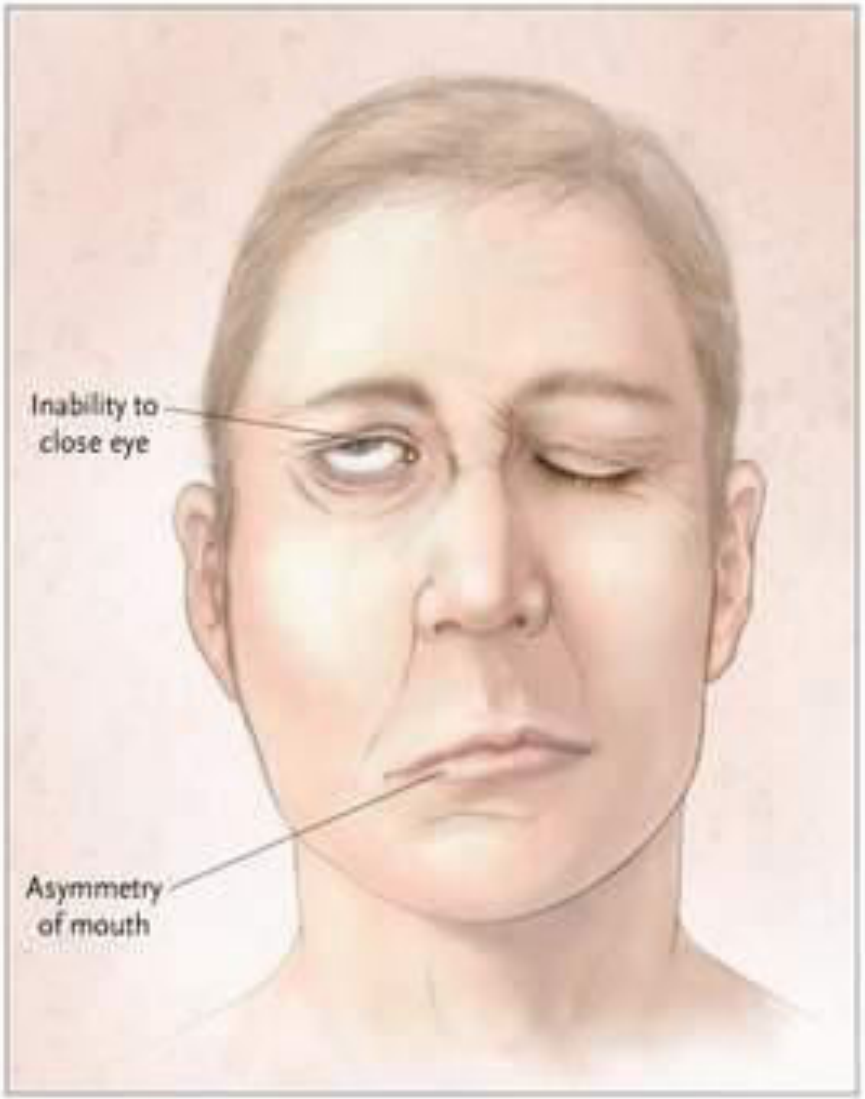
5. Systemic diseases:

- Diabetes mellitus.
- Hyperthyroidism
- Uraemia
- Polyarteritis nodosa.
- Wegener's granulomatosis.
- Sarcoidosis.
- Leprosy.
- Leukaemia
- Demyelinating disease.

Bell's palsy.

- ✓ 60-75% of facial paralysis is due to bell's palsy.
- ✓ It is defined as peripheral facial paralysis or paresis os acute onset.
- ✓ Both sexes are affected with equal frequency.
- ✓ Any age group may be affected through incidence rises with increasing age.
- ✓ A positive family history is present in 6-8% of patients.
- ✓ Risk of bell's palsy is more in diabetics and pregnant women.


Inability to close eye
Asymmetry of mouth


wiseGEEK

Aetiology :

1. Viral infection- due to herpes simplex, herpes zoster or the Epstein-Barr virus.
2. Vascular ischaemia- may be primary or secondary.

- Primary ischaemia is induced by cold or emotional stress.
- Secondary ischaemia is the result of primary ischaemia which causes capillary permeability leading to exudation of fluid,oedema and compression of microcirculation of the nerve.

3. Hereditary – the fallopian canal is narrow because of hereditary predisposition and this makes the nerve susceptible to early compression with the slightest oedema.
4. Autoimmune disorder- T-lymphocyte changes have been observed.

Clinical features:

- Onset is sudden.
- Patient is unable to close his eye.
- On attempting to close the eye, eyeball turns up and out (bell's phenomenon).
- Saliva dribles from the angle of mouth.
- Face becomes asymmetrical .
- Tears flow down from the eye.(epiphora)
- Pain in the ear may precede or accompany the nerve paralysis.
- Some complain of noise intolerance (stapedial paralysis) or loss of taste (involvement of chorda tympani).
- Paralysis may be complete or incomplete.
- Bell's palsy is recurrent in 3-10% of patients.

Diagnosis :

- Diagnosis is always by exclusion.
- All other known causes of peripheral facial paralysis should be excluded.
- This requires careful history, complete otological and head and neck examinations.

❑ X –ray studies, blood tests such as total count, peripheral smear, ESR, blood sugar and serology.

❑ Nerve excitability tests are done daily or on alternate days and compared with the normal side to monitor nerve degeneration.

❑ Localizing the site of lesion (topodiagnosis) helps in establishing the aetiology and also the site of surgical decompression of nerve, if that becomes necessary

Treatment :

A. General treatment.

B. Medical management.

C. Surgical treatment.

A. General treatment:

- Reassurance
- Relief of ear pain by analgesics.
- Care of the eye, eye must be protected against exposure keratitis.
- Physiotherapy or massage of the facial muscles gives psychological support to the patient.

Treatment (Cont.)

B. Medical management:

❖ Steroids –

- utility has not been proved beyond doubt.
- Prednisolone is the drug of choice.
- If pt. reports within 1 week, the adult dose of prednosolone is 1mg/kg/day divided into morning and evening doses for 5 days.
- Patient is seen on the 5^{th} day.
- If paralysis is incomplete or is recovering, dose is tapered during the next 5 days.
- If paralysis remains complete, the same dose is continued for another 10 days and thereafter tapered in next 5 days. (total of 20 days).
- Steroids can be combined with acyclovir for herpes zoster oticus or bell's palsy.

Treatment (Cont.)

C. Surgical treatment:

- Nerve decompression relieves pressure on the nerve fibres and thus improves the microcirculation of the nerve.
- Vertical and tympanic segments of nerve are decompressed.
- Some suggested total decompression including labyrinthine segment by post aural and middle fossa approach.

Prognosis :

- 85-90% of the pts. Recover fully.
- 10-15% recover incompletely and may be left with some stigmata of regeneration .
- Recurrent facial palsy may not recover fully.
- Prognosis is good in incomplete bells palsy (95% complete recovery)
- In those where clinical recovery starts within 3 weeks of onset (75% complete recovery).

Topodiagnostic tests for lesions in intratemporal parts.

The following tests are useful in finding the site of lesions in paralysis of lower motor neuron.

1. Schirmer's test-it compares lacrimation of the two sides. A strip of filter paper is hooked in the lower fornix of ach eye and the amount of wetting of strip measured. Decreased lacrimation indicates lesion proximal to the geniculate ganglion .
2. Stapedial reflex- stapedial reflex is lost in lesions above the nerve to stapedius. It is tested by tympanometry.

Topodiagnostic tests (Cont.)

3. Taste test- can be measured by a drop of salt or sugar solution placed on one side of the protruded tongue or by electrogustometry. Impairment of taste indicates lesion above the chorda tympani.
4. Submandibular salivary flow test- it also measures function of chorda tympani. Polythene tubes are passed into the Wharton's ducts and drops of saliva counted during one minute period.decreased salivation shows injury above the chorda.

Complications following facial paralysis:

Peripheral facial paralysis due to any cause may result in any of the following complications.

1. Incomplete recovery- facial asymmetry persists. Eye cannot be closed resulting in epiphora. A weak oral sphincter causes drooling and difficulty in taking food.
2. Exposure keratitis- eye cannot be closed, tear film from the cornea evaporates causing dryness, exposure keratitis and corneal ulcer.

- It can be prevented by use of artificial tears every 1-2 hours, eye ointment and proper cover for the eye at night. Temporary tarsorrhaphy may also be indicated.

Complications following facial paralysis:

3. Synkinesis (mass movement)- when the pts. Wishes to close the eye, corner of mouth also twitches or vice versa. it is due to cross innervations of fibres, there is no treatment.

4 . Tics and spasm- the result of faulty regeneration of fibres. Involuntary movement are seen on the affected side of the face.

5. Contractures – they result from fibrosis of atrophied muscles or fixed contraction of a group of muscles. They affect movements of face but facial symmetry at rest is good.

Complications following facial paralysis:

6. Crocodile tears (gustatory lacrimation)- there is unilateral lacrimation with mastication. This is due to faulty regeneration of parasympathetic fibres which now supply lacrimal gland instead of the salivary glands. It can be treated by section of greater superficial petrosal nerve or tympanic neurectomy.
7. Frey's syndrome (gustatory sweating)-there is sweating and flushing of skin over the parotid area during mastication. It results from parotid surgery.
8. Psychological and social problems- drooling during eating and drinking and impairment of speech cause social problems.

Surgery of facial nerve:

1. Decompression – nerve may be compressed by oedema, haematoma or a fractured bone in its intratemporal part. The bony canal is exposed and uncapped. The sheath of nerve is also slit to relive pressure due to oedema or intraneural haematoma.
2. End to end anastomosis- done when the gap between severed ends of the nerves is only a few millimeters. It is a suitable procedure for extratemporal part of the nerve.

Surgery of facial nerve (Cont.)

3. Nerve graft-(cable graft)- when the gap between severed ends cannot be closed by end to end anastomosis, a nerve graft is more suitable than extensive re-routing or mobilization of nerve. Nerve graft is taken from greater auricular, lateral cutaneous nerve of thigh or the sural nerve.
4. Hypoglossal – facial anastomosis- hypoglossal nerve is anastomosed to the severed peripheral end of the facial nerve. It improves the muscle tone and permits some movement of facial muscles.
5. Plastic procedures- they are used to improve cosmetic appearance when nerve grafting is not feasible or has failed. The procedures include facial slings, face lift operation or slings of masseter and temporalis muscle.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