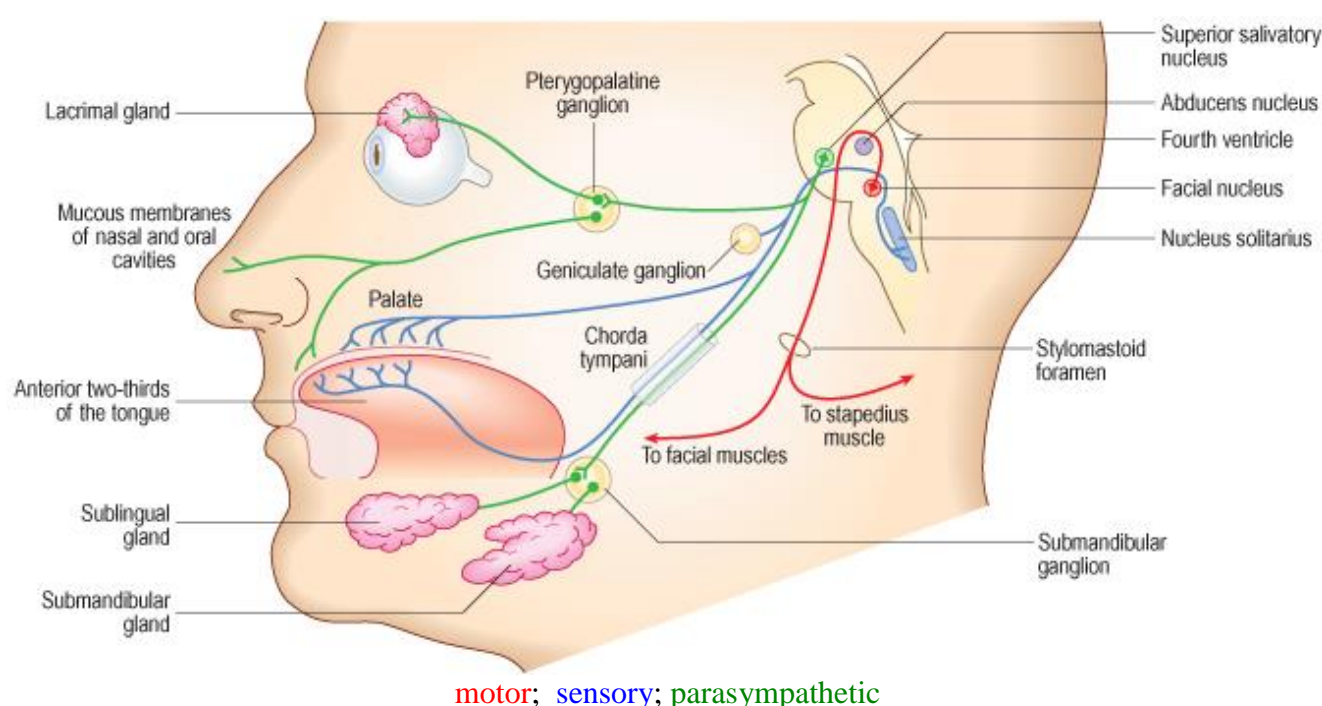


Facial Nerve Disorders

Last updated: January 18, 2020

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motor; sensory; parasympathetic

FACIAL PALSY

facial paralysis in OTITIS MEDIA – see p. Ear38 >>

ETIOLOGY

- Systemic disorders** - autoimmune diseases, systemic malignancy, hypertension, sarcoidosis*, acute porphyria, hyperthyroidism, pregnancy.
***uveoparotid fever (s. Heerfordt syndrome)** see below >>
N.B. sarcoidosis is common cause in blacks!
- Ear pathology** (ear infection, cholesteatoma). *facial paralysis in OTITIS MEDIA* - see p. Ear38 >>
- Infections** (tuberculosis, mononucleosis, Lyme disease, poliomyelitis, syphilis, HIV, herpes simplex*, herpes zoster**, EBV).
*reactivation of latent infection in geniculate ganglion
****herpes zoster oticus (s. geniculate zoster, Ramsay Hunt's syndrome)** see below >>
CN7 palsy is rare in mumps!
- Immunizations** (for polio and rabies).
- Trauma** (fracture of temporal bone, birth trauma)
improvement is rule (when palsy is associated with head trauma), but recovery may not be complete!
- Surgery**:
a) palsy immediately after surgery - **nerve transection** → prompt surgical evaluation
b) delayed onset - **nerve edema** - improves with time.
- Tumors** (of nerve, of temporal bone, of parotid gland)
facial schwannoma (5% of all CN7 palsies) see p. Onc62 >>
palpate parotid bimanually in all patients!
thorough neck examination for adenopathy!
- Toxins** (arsenic, CO, ethylene glycol).
- Part of **polyneuritis** (leprosy, Guillain-Barré syndrome, diphtheritic; only seldom in diabetic or alcoholic neuropathy!)
- Idiopathic - Bell's palsy.**

Etiology of NUCLEUS LESION - vascular lesions, tumors, inflammatory lesions, acute poliomyelitis, MS.

Bilateral facial palsy (any lesion that can cause unilateral palsy) - most often sarcoidosis, Guillain-Barré syndrome, leprosy, leukemia, meningococcal meningitis.

Positive FAMILY HISTORY:

- MELKERSSON-ROSENTHAL syndrome.**
- MÖBIUS syndrome.**
- BELL'S palsy** (occasionally).

Perinatal facial palsy:

- Maternal **infections** (e.g. rubella)
- Drugs** used during pregnancy (e.g. thalidomide)
- Difficult delivery** (esp. forceps) - recovers spontaneously in few days or weeks.

GUIDE TO LESION SITE LOCALIZATION

1. **Other neurological symptoms** (e.g. hemiplegia, loss of sensation, cranial nerve dysfunction).
 - ipsilateral hemiparesis** - cortical / subcortical lesion;
 - contralateral hemiparesis** - pontine lesion near facial motor nucleus.
2. **Otologic signs and symptoms.**
3. What **other functions of CN7** are involved:
 - 1) change in **TEARING** (quantified by **Schirmer test**) see p. D1 >>
 - a) lesion proximal to geniculate ganglion → **tear production** ↓.
 - b) lesion distal to geniculate ganglion → **tear production** ↑ (corneal irritation from lagophthalmos); atonic **epiphora** (ectropion from orbicularis oculi weakness → tears pool in lower conjunctival sac and spill over lower lid margin).
 - 2) reduced **TASTE** (quantified by **electrogustometry** - taste threshold in response to small electric current applied to anterior tongue). about abnormal taste → see p. A79 >>
 - 3) reduced **SALIVATION** (quantified by **cannulating Wharton duct** or by **checking saliva pH** - pH rises with increased salivation).
 - 4) **HYPERACUSIS** (confirmed by **acoustic reflex testing**).

CLINICAL GRADING OF SEVERITY

HOUSE-BRACKMANN GRADING SCALE

Grade	Definition
I	Normal symmetrical function in all areas
II	Slight weakness noticeable only on close inspection Complete eye closure with minimal effort Slight asymmetry of smile with maximal effort Synkinesis barely noticeable, contracture, or spasm absent
III	Obvious weakness, but not disfiguring May not be able to lift eyebrow Complete eye closure and strong but asymmetrical mouth movement with maximal effort Obvious, but not disfiguring synkinesis, mass movement or spasm
IV	Obvious disfiguring weakness Inability to lift brow Incomplete eye closure and asymmetry of mouth with maximal effort Severe synkinesis, mass movement, spasm
V	Motion barely perceptible Incomplete eye closure, slight movement corner mouth Synkinesis, contracture, and spasm usually absent
VI	No movement , loss of tone, no synkinesis, contracture, or spasm

CLINICO-ANATOMICAL SYNDROMES

Lesion Site	CN7 Findings	Possible Other Findings	Common Etiologies	Comments
Motor cortex, internal capsule	Contralateral central* palsy of lower face (upper face spared**); tearing, salivation, taste intact.	Hemiparesis (ipsilateral); tongue weakness (ipsilateral); frontal lobe signs	Vascular	
*weakness, spasticity, hyperreflexia; emotional expressions may be spared. **mild upper face weakness is possible!!! (rarely, upper face may be severely affected, but eye blink reflex is retained, vs. lost in LMN lesion)				
Pontine motor nucleus	Ipsilateral full* palsy ; tearing, salivation, taste intact.	Hemiparesis (contralateral); CN6 palsy (ipsilateral)	Infarction (Millard-Gubler syndrome), glioma, thalidomide, multiple sclerosis	*upper and lower face
Cerebellopontine angle		CN5, CN8 lesion; cerebellar ataxia	Tumors (acoustic / facial neuroma, meningioma, cholesteatoma)	CN8 is usually involved first
Internal auditory meatus (MEATAL SEGMENT)	Ipsilateral full palsy ; tearing ↓, salivation ↓, taste ↓	CN8 lesion	Intracanalicular neuroma (acoustic or facial)	
Facial canal: LABYRINTHINE SEGMENT			Bell's palsy, Ramsay Hunt syndrome, temporal fracture, hemangioma	
Facial canal: TYMPANIC SEGMENT	Ipsilateral full palsy ; salivation ↓, taste ↓;	Inner, middle ear dysfunction	Bell's palsy, otitis media, cholesteatoma, temporal bone fracture	With lesion distal to chorda tympani, salivation & taste are spared.
Facial canal: MASTOID SEGMENT	tearing spared (may be ↑ with atonic epiphora).			
Stylomastoid foramen	Ipsilateral full* palsy ; salivation**, taste, tearing intact.		Facial trauma or surgery; parotid tumors; sarcoidosis	*sparing of peripheral branches possible; **drooling

SUPRANUCLEAR (CENTRAL) PALSY

- only contralateral **lower face** is paralyzed (esp. perioral musculature); mild upper face weakness is possible. *see table above*
 - paralysed *m. buccinator* → chewing difficulty (food pools in vestibulum oris).
 - paralysed *m. orbicularis oris* → flattened nasolabial fold, drooping mouth corner, drooling.
 - when patient attempts to smile, lower face is pulled to opposite side (this distortion may give false appearance of deviation of protruded tongue or open jaw).
- **emotional control** of facial motion is provided by **EXTRAPYRAMIDAL INPUT** (via RF - from frontal cortex, thalamus, globus pallidus) – volitional (pyramidal) and emotional (extrapyramidal) components may be affected separately:

- if supranuclear lesion spares extrapyramidal input → preserved emotional facial expressions (e.g. surprise or pain) despite facial palsy.
- lesions of thalamus or midbrain → contralateral palsy of emotional facial expressions with intact voluntary motion.
- lesions of globus pallidus → facial bradykinesia (masklike facies); some facial motion is detectable (cortical volitional and other extrapyramidal emotional pathways).

NUCLEAR LESION

- motor CN7 nucleus lies:
 - within RF of **caudal pons**;
 - between **trigeminal nucleus** (dorsally) and **superior olivary nucleus*** (ventromedially).
- ipsilateral **full (upper & lower) face** paralysis;
 - paralysed *m. orbicularis oculi* → lagophthalmos, atonic epiphora.
 - when patient attempts to close eye, eyeball diverts upward (**BELL phenomenon**).
- tearing, salivation, taste intact.
- contralateral **hemiplegia** may be seen (corticospinal tract has not crossed yet) - **MILLARD-GUBLER syndrome**.
- ipsilateral **CN6 palsy** may be seen.

*spatial sound localization

CEREBELLOPONTINE ANGLE SYNDROME

- ipsilateral **full (upper & lower) face** paralysis.
- **stapedius** paralysis (HYPERACUSIS - painful sensitivity to loud sounds), **tearing**↓ (XEROPHTHALMIA), **salivation**↓ (XEROSTOMIA), **taste**↓.
- **CN8** is often involved; **cerebellum** and **CN5** can also be involved.

FACIAL CANAL SYNDROME

- LABYRINTHINE SEGMENT – most full facial palsy (SVE, SVA, GVE, GVA, GSA)
- TYMPANIC SEGMENT – lacrimation spared.
- MASTOID SEGMENT distal to chorda tympani – only motor facial palsy.

STYLOMASTOID FORAMEN SYNDROME

- only motor facial palsy.
- some motor branches may be spared.

DIAGNOSIS

Schirmer test (lacrimal secretion) → see p. D1 >>

- Check cornea for **exposure keratitis!**
- AUDIOMETRY** should be performed in all patients.
- NEUROIMAGING** (MRI, CT) indications:
 - unclear etiology
 - recurrent / progressive / atypical paralysis
 - trauma.
- ELECTROPHYSIOLOGY**:
 - ENoG (electroneurography)** - in acute unilateral complete paralysis (if threshold is 3.5 mA greater than normal side or if $\geq 90\%$ axons are lost, there is poor prognosis for spontaneous recovery) during days 4-21.
 - even in complete transection, nerve conducts impulses distal to injury for up to 72 hours (until wallerian degeneration occurs) - ENoG is not performed until more than 3 days after total paralysis!
 - ENoG is also not of benefit in long-standing paralysis (> 3 weeks) because of polyphasic potentials when degeneration and regeneration are occurring.
 - ENoG is not employed in paresis - even minimal voluntary motion after 3 days indicates minor injury, with full recovery to be expected.
 - voluntary EMG** is helpful in long-standing or congenital facial paralysis.
 - trigemino-facial reflex** (electrodes record blink reflex after percutaneous stimulation of supraorbital nerve) - conduction of reflex arc between CN5 (afferent) and CN7 (efferent) – the only direct test able to measure intracranial CN7 pathology!!!
- If facial reanimation procedure is considered (in longstanding or congenital facial paralysis) → facial **MUSCLE BIOPSY** (whether viable muscle fibers are present).

GENERAL MANAGEMENT

All complete TRAUMATIC cases must be considered for surgical repair!

Good guide (also for patients) - <http://www.bellspalsy.ws>

Eye protection from **exposure keratitis** (esp. if ipsilateral CN5 deficit co-exists - patient has no reliable guide as to severity of corneal epithelial disruption):

DAY:

- artificial tears** frequently (q15-30min); best are "for sensitive eyes", "non-allergic", or "preservative free" artificial tears; *THIMEROSAL* is preservative that can be particularly irritating!
- room humidifiers
- soft **contact lenses** are hydrophilic (hold water) - can provide source of moisture directly over cornea + protect eye from debris.
 - eyelids help to hold contact lens in place - if lids become limp, contacts cannot be worn until function begins to return.
 - wearing lens is not complete replacement for your tears - use plenty of saline or eye drops designed for use with contacts.
 - at acute stage, eye can be so dry that it is impossible to keep contact lens moist for more than few minutes - contacts can not be worn!!!
Do not let the lens dry out! (dry contact directly over cornea is dangerous).
If you cannot keep lens moist, do not wear it!
- sunglasses** while outside (avoid contaminated air!)
- moisture chamber**:



- Punctal Plug Insertion** - simple procedure to partially block tear duct; little or no discomfort; removal is simple. **Collagen plugs** - option for short term use (self-dissolving - effective for ≈ 10 days).

NIGHT:

- ophthalmic ointment** (e.g. with dexpanthenol) – thicker than artificial tears due to addition of mineral oil.

2) **eye patch** or **taping** closed affected eyelid*.

*if not done correctly eyelid can easily pop open, exposing eye to worse damage than without tape; make sure to use gentle, non-abrasive and easily removed tape (such as paper surgical tape), and learn correct way to apply it.

Any corneal abrasion / infection should be treated immediately!

REHABILITATION

For **early period** (when muscles are completely flaccid) - limit therapy to:

- 1) **moist heat** (to ease soreness and reduce swelling)
- 2) **massage** (to ease soreness, to provide degree of motion & stimulation to muscles and increase circulation)
- 3) **mental exercises** (to retain "memory" of facial motions).

Focus your exercise energy on maintaining brain-to-nerve-to-muscle connection - this is more important than physical motions your muscles did before, and will do again.

- when laughing / talking support face muscles with hand – to counteract pulling by muscles of nonparalysed side.
- some advocate during recovery period – electrophoresis with PROSERINE.
- **ELECTRICAL STIMULATION** continues to be widely used in treatment of facial paralysis although there is mounting evidence that it may be contraindicated:
 - **facial muscles resist degeneration** post denervation for longer periods of time than other skeletal muscle - **facial muscles may remain viable for 3 or more years** - use of electrical stimulation to maintain viability of facial muscle is unfounded!
 - electrical stimulation may **interfere with neural regeneration!**
 - patients who undergo electrical stimulation acutely may demonstrate **more synkinesis and mass action** than those who do not (it is difficult to produce isolated contraction of facial muscles using electrical stimulation due to their small size and close proximity to each other - contraction produced causes mass action which reinforces abnormal motor patterns and can be painful)!
- avoid **gross maximum effort exercises** ("close your eyes as hard as you can", "smile broadly", "pucker your lips", etc) - recruit excessive motor units (producing patterns that differ from typical facial expressions which are gentle and fluid) + reinforce abnormal movement patterns.

N.B. one of first signs of regenerating nerve may be pain; in case of CN7 – pain in ear canal!

For **permanent facial paralysis**:

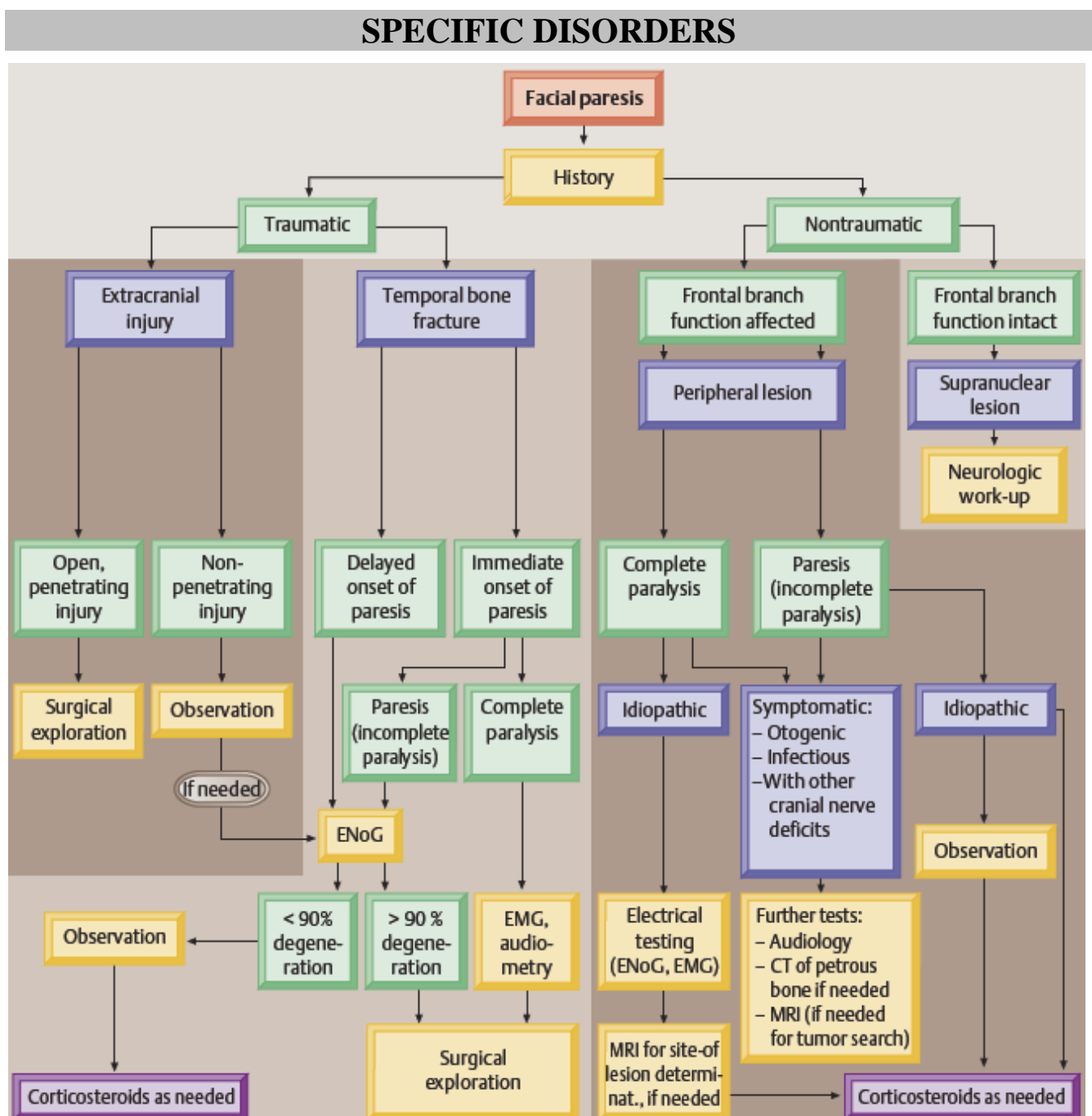
- massage / electric stimulation of paralyzed muscles (no proven benefit!) – to prevent atrophy, to relieve strain on relaxed muscles and to preserve tone.
- **wait minimum 6 months** for spontaneous CN7 function return (exception - **nerve cut during surgery** – restore nerve continuity ASAP!)

A. STATIC PROCEDURES:

- 1) **gold weights**, weight-adjustable magnet, palpebral springs, silicone-encircling bands can be inserted into eyelids.
- 2) **lateral tarsorrhaphy** (reversible), **canthoplasty** (irreversible), **lower lid shortening**.
- 3) **sling suspension** (with fascia lata or alloplastic strips) – for very old / very ill patients.

B. DYNAMIC PROCEDURES:

- 1) extracranial CN12→CN7 or CN11→CN7 **anastomosis**;
sooner or later, new motor pattern develops in cerebral cortex and movements of facial muscles are dissociated from those of tongue / shoulder.
- 2) **cross-facial nerve grafting** (part of proximal nerve on intact side is anastomosed to distal nerve on paralyzed side with use of nerve grafts)
- 3) **muscle transfer procedures** are preferred in patients > 50 years (have relatively small chance of success with nerve grafting):
 - temporalis or masseter transposition;
 - free muscle grafts;
 - microvascular free nerve-muscle grafts.



BELL'S PALSY (s. idiopathic acute facial palsy)

EPIDEMIOLOGY, ETIOLOGY

- 60-75% of all cases of facial paralysis.
- INCIDENCE 20-30 cases per 100,000 population per year (increases with age).
- male ≈ female; right ≈ left.

- 7-10% cases are **recurrent** (ipsilateral* or contralateral).
*consider tumor as etiology
- 4-10% have positive **family history**.
- RISK FACTORS: pregnancy (triples risk!), diabetes mellitus (quadruples risk!), immunosuppression.
- 60-70% had **viral prodrome** 7-10 days before.
- most commonly accepted cause is **HERPES SIMPLEX-1** infection (reactivation of HSV genomes from geniculate ganglia); other suggested causes – **other viruses**, **vascular ischemia**.
- mechanism – inflammatory nerve edema within rigid facial (fallopian) canal.
- alternative cause / mechanism - body arteries elongate and become tortuous with age (may do so even in third decade of life) → **sudden shift of arterial loop** in cerebellopontine angle:
 - a) rapid stretch of CN7-8 bundle between brain stem and internal auditory meatus from anterior aspect (facial nerve side), actively compressing nerve against petrous bone.
 - b) direct compression of intrapontine facial nerve.

CLINICAL CHARACTERISTICS

- *possible preceding symptoms* – facial paralysis may be heralded / accompanied by **pain behind ear** - sense of mastoid fullness (pain at stylomastoid foramen), periauricular paresthesias or even otalgia (without vesicles!).
- **sudden onset**; maximal facial weakness is reached within 48-72 hours (vs. CN7 schwannoma – progresses over > 3 weeks).
- peripheral CN7 dysfunction *may involve all branches* (LMN palsy, dysgeusia, hyperacusis).
- may complain of **numb / heavy feeling in face**, but no sensory loss is demonstrable.
- pain is absent (if present – consider Ramsay-Hunt syndrome).
 - at onset, and for variable time thereafter, ipsilateral **occipital headaches** and frequently **tenderness over ipsilateral occipital bone** is present.
- no other symptoms & signs!!!
 - may develop ipsilateral **CN8 dysfunction** (tinnitus, vertigo, disequilibrium, sensorineural hearing loss).

DIAGNOSIS

- **clinical!**
- diagnosis of exclusion!!!
- **no imaging is required!** (MRI may show contrast enhancement of nerve, but CT and X-rays are typically negative) – imaging results do not change initial management.
N.B. perform MRI if tumor is suspected!
- in endemic areas, **Lyme disease** is consideration.
- **ENoG (electroneurography)** during days 4-21 – for prognostic purposes. *see below*

PROGNOSIS

- spontaneous improvement within 6 months.
- without treatment, improvement is noted within 3-12 weeks in 85%; remainder show improvement by 3-6 months (≈ 70% regain normal function; vs. 92% with treatment).
N.B. *all patients should show some improvement by 6 months!* (vs. CN7 schwannoma – gradually worsening paralysis, steroid responsiveness eventually disappears)
- denervation (after 10 days from onset) indicates axonal degeneration - will be long delay (3 months, as rule) before regeneration occurs (may be incomplete).
- when recovery is partial, “contractures” may develop on paralyzed side (continuous diffuse contraction); H: botulinum toxin.

TREATMENT

see also GENERAL MANAGEMENT (*above*)

- **mainstay of treatment** - **10-day course** (must be started early!) of **PREDNISONE** (60 mg/d for 5 days → tapered down by 10 mg/day for 5 days) – **level A** recommendation!
- **ACYCLOVIR** (400 mg ×5 /d) is not helpful; adding acyclovir is **level C** recommendation;
 - ACYCLOVIR alone is not effective in facial recovery!!! (must be used only in COMBINATION WITH PREDNISONE).
 - alternative – **VALACYCLOVIR** 1 g/d for 5 days.
- in Lithuania, **diuretics** are also administered.
- *those who will not recover conservatively must be identified* early* – within 2 weeks (to prevent permanent paralysis).

*degeneration of ≥ 90% axons on electroneurography → **SURGICAL DECOMPRESSION**

(**NEUROLYSIS**) of entire infratemporal facial nerve;

- because presumed site of entrapment is meatal foramen (beginning of labyrinthine segment), **middle fossa approach** is used to decompress this region; remainder of nerve can then be decompressed via **transmastoid approach**.
- localization of decompression may be guided by MRI.

N.B. neurolysis after 2 weeks (from paralysis onset) does not improve outcome!!!

According to newest studies *surgical decompression of CN7 is not beneficial* in Bell palsy!

Treatment protocol:

Acute PARESIS:

A) patient first seen on **days 0-14** → **ACYCLOVIR + PREDNISONE** → follow-up in 5 days:

- a) **paresis** → follow-up in 1 month
- b) **paralysis** → paralysis protocol

B) patient first seen **after 14 days** → observation, follow-up in 6 months.

Acute PARALYSIS:

A) patient first seen on **days 0-14** → **ACYCLOVIR + PREDNISONE**; ENoG on 3rd paralysis day and repeated every other day until 2 weeks have elapsed from onset of total paralysis:

- a) **< 90% degeneration** → continue **ACYCLOVIR + PREDNISONE** until full course.
- b) **> 90% degeneration** → surgical **NEUROLYSIS**.
- c) **100% degeneration** → voluntary EMG to confirm complete degeneration.

B) patient first seen **after 14 days** → EMG → follow-up in 5 months.

RAMSAY-HUNT syndrome (s. geniculate zoster, herpes zoster oticus)

- **herpes zoster** of geniculate ganglion:

see p. 256 (7-8) >>

- 1) **very painful** vesicular eruption on pinna, external auditory canal, pharynx
- 2) severe CN7 palsy
- 3) often CN8 is affected (vertigo, high-tone deafness)

Treatment – **VALACYCLOVIR**.

HEERFORDT syndrome (s. uveoparotid fever)

- form of **sarcoidosis**:

- 1) chronic parotid enlargement → CN7 palsy
- 2) uveitis

- 3) long-continued low degree fever.

MELKERSSON-ROSENTHAL syndrome

- 1) recurrent CN7 paralysis
- 2) recurrent and eventually permanent facial (particularly labial) edema [cheilitis granulomatosa]
- 3) fissured tongue.

MÖBIUS syndrome

- failure of motoneuron development in bilateral CN6 & 7 nuclei → CONGENITAL facial diplegia with abnormalities of horizontal gaze (convergent squint).

- most cases occur sporadically due to *destructive (frequently vascular) lesions*.
- 50% patients have *limb anomalies* (talipes, hypoplasia of digits, transverse terminal defects, syndactyly).
- Möbius' syndrome is found more frequently among children with *congenital heart malformations* than in isolation.
- association with Poland anomaly may be familial (**POLAND-MÖBIUS syndrome**).
- facial immobility can cause significant social handicap (immobile, dull facies may give incorrect impression of mental retardation).
- disabilities can initially be very significant (incl. *feeding difficulties due to poor suck* and speech impairments), but tend to improve with time.
- no treatment other than possible *cosmetic surgery*.

FACIAL OVERACTIVATION SYNDROMES

HEMIFACIAL SPASM

- involuntary, unilateral, painless, episodic **tonic & clonic contraction** of CN7 muscles.

- most often in *middle-aged women* (esp. Asian).
- begins as *twitches around eye*.
- progresses (during few months) to involve remaining ipsilateral facial muscles.
- even during sleep!
- stapedius contraction may produce tinnitus.
- muscles are normally relaxed between twitches.
- provoked by voluntary facial motion, emotional stress.
- etiology (generally, only cosmetic problem!)
 - a) **compression** of motor nerve root at brain stem by aberrant / ectopic **vascular loop** e.g. AICA loop in internal auditory canal (→ focal demyelination → ectopic excitation / ephaptic transmission → impulses conduct centrifugally to CN7 nucleus → nucleus hyperactivity → bursts of hemifacial spasm).
 - b) **recovery from Bell's palsy**.
 - c) **tumor**
 - d) **bony abnormalities**.
- differentiate from *blepharospasm* (involuntary spasm of both orbicularis oculi muscles).
- diagnosis:
 - MRI (vascular loop).
 - EMG – 5-20 Hz bursts of muscle action potentials.
- treatment:
 - 1) **BOTULINUM TOXIN** – treatment of choice!!!
 - 2) **CARBAMAZEPINE, CLONAZEPAM**
 - 3) nerve (or its branches) can be **injected with alcohol** or **partially sectioned** → relief from spasms until nerve regenerates.
 - 4) **PERMANENT RELIEF**:
 - a) Jannetta procedure - **decompressing vessels** at root entry zone – see p. Op350 >>
 - b) **anastomosing** CN7 with CN11 or CN12.

MYOKYMIA

- progressive, irregular **fibrillation** of individual facial muscle fibers.

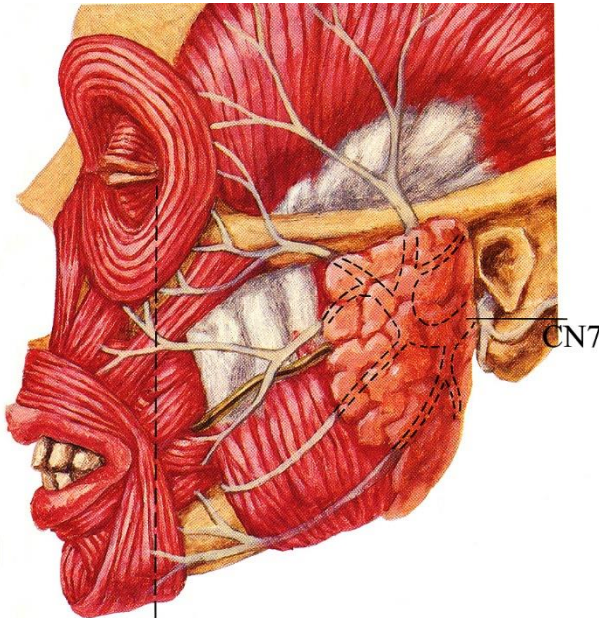
- begins in frontalis → extends to involve all ipsilateral facial muscles.
- EMG – spontaneous regular 30-70 Hz bursts of motor units.
- etiology (grave! vs. hemifacial spasm) - pontine gliomas, multiple sclerosis!

FACIAL NERVE TRAUMA

CN7 injuries should be **repaired surgically** if they occur *posterior to vertical line drawn from LATERAL CANTHUS of eye*.

- injuries *anterior to this line* do not need to be repaired (with microsurgical techniques, some branches can be rejoined).
- if nerve is *cut by sharp object* → repair primarily if wound site is clean.
- *destructive injuries* → nerve repair is usually secondary, following replacement of missing nerve tissue by graft.

REPAIR TECHNIQUE → see p. PN7 >>



RE-INNervation SYNDROMES

- a) incorrect postparalytic re-innervation (axons regrowth to nonoriginal structures).
- b) spread of impulses between fibers within nerve (*ephaptic conduction*).

POSTPARALYTIC SYNKINESIS

Synkinesis - unintentional muscle motion when another muscle is voluntarily contracted.

- example - jaw opening causes closure of eyelids (jaw-winking).
- mechanism - re-innervation of *different muscles* by axons from *same motoneuron*.

CROCODILE TEARS

- unilateral **LACRIMATION** during meals.

- mechanism - axons from *superior salivatory nucleus* (intended for chorda tympani → submandibular ganglion) enter *greater superficial petrosal nerve* (end up in pterygopalatine ganglion).

GUSTATORY SWEATING (FREY syndrome)

- facial **SWEATING** during meals.

- most common cause – **parotidectomy**.
- mechanism - cholinergic fibers originally intended for *parotid gland* end up in *sweat glands*.

BIBLIOGRAPHY for ch. “Cranial Neuropathies” → follow this [LINK >>](#)