Facial Nerve Disorders

Facial Palsy

facial paralysis in OTITH MEDIA – see p. Eur38 >>

ETIOLOGY

   - uveoparotid fever (s. Heerfordt syndrome) see below >>
   N.B. sarcoidosis is common cause in blacks.

2. Ear pathology (ear infection, cholesteatoma).
   - facial paralysis in OTITH MEDIA - see p. Eur38 >>

   - *reactivation of latent infection in geniculate ganglion
   - **herpes zoster oticus (s. geniculate zoster, Ramsay Hunt’s syndrome) see below >>
   CNT palsy is rare in mumps!

4. Immunizations (for polio and rabies).

5. Trauma (fracture of temporal bone, birth trauma) improvement is rule (when palsy is associated with head trauma), but recovery may not be complete!

6. Surgery:
   a) palsy immediately after surgery - nerve transaction – prompt surgical evaluation
   b) delayed onset - nerve edema - improves with time:

7. Tumors (of nerve, of temporal bone, of parotid gland)
   - facial schwannoma (3% of all CN7 palsies) see p. Onc62 >>
   - palpate parotid bimanually in all patients! thorough neck examination for adenopathy!

8. Toxins (arsenic, CO, ethylene glycol).

9. Part of polyneuritis (leprosy, Guillain-Barré syndrome, diphtheritic; only seldom in diabetic or alcoholic neuropathy?)

10. Idiopathic - Bell’s palsy.
    - Etiology of NUCLEUS LEMNA - vascular lesions, tumors, inflammatory lesions, acute polynielytis, MS.

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

Positive FAMILY HISTORY

1. MEHLERSON-Rosenthal syndrome.
3. Bell’s palsy (occasionally).

Perinatal facial palsy:

1. Maternal infections (e.g. rubella)
2. Drugs used during pregnancy (e.g. thalidomide)
3. 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. Otolologic signs and symptoms

3. What other functions of CN7 are involved?
   (a) change in TEARING (quantified by Schirmer test) to see if D1 >>
   (b) lesion distal to geniculate ganglion → tear production;
   (c) emotional expressions may be spared.

CLINICAL GRADING OF SEVERITY

HOUSE-BRACKMANN GRADING SCALE

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

CLINICO-ANATOMICAL SYNDROMES

<table>
<thead>
<tr>
<th>Lesion Site</th>
<th>CN7 Findings</th>
<th>Possible Other Findings</th>
<th>Common Etiologies</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor cortex, internal capsule</td>
<td>Ipsilateral full* palsy; tearing, salivation, taste intact</td>
<td>Hemiparesis (ipsilateral); tongue weakness (ipsilateral); frontal lobe signs</td>
<td>Vascular</td>
<td></td>
</tr>
<tr>
<td>Posterior motor nucleus</td>
<td>Ipsilateral full* palsy; tearing, salivation, taste intact</td>
<td>Hemiataxia (Millard-Gubler syndrome); glioma, thalidomide, multiple sclerosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cerebellopontine angle</td>
<td>CN5, CN8 lesion; cerebellar ataxia</td>
<td>Tumescent (acoustic / facial neuralgia, meningioma, cholesteatoma)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Internal auditory meatus (MESIAL SEGMENT)</td>
<td>Ipsilateral full* palsy; tearing, salivation, taste intact</td>
<td>CN8 lesion</td>
<td>Intracanalicular neurona (acoustic or facial)</td>
<td>CN8 is usually involved first</td>
</tr>
<tr>
<td>Facial canal: LABYRINTHINE SEGMENT</td>
<td>Ipsilateral full* palsy; tearing, salivation, taste intact, slight asymmetry</td>
<td>Inner, middle ear dysfunction</td>
<td>Bell’s palsy; Ramsay Hunt syndrome, temporal fracture, hemihypoglossia</td>
<td>Cochlea / vestibular organs may be involved.</td>
</tr>
<tr>
<td>Facial canal: TYPANIC SEGMENT</td>
<td>Ipsilateral full* palsy; tearing, salivation, taste intact</td>
<td>Bell’s palsy; otitis media, cholesteatoma, temporal bone fracture</td>
<td>With lesion distal to chorda tympani, salivation &amp; taste are spared.</td>
<td></td>
</tr>
<tr>
<td>Facial canal: MASTOID SEGMENT</td>
<td>Ipsilateral full* palsy; tearing, salivation, taste intact</td>
<td>Facial trauma or surgery; parotid tumors; sarcoidosis</td>
<td>*sparing of peripheral branches possible; **drowning</td>
<td></td>
</tr>
<tr>
<td>Stylomastoid foramen</td>
<td>Ipsilateral full* palsy; salivation*, taste intact</td>
<td>Facial palsy</td>
<td>With lesion distal to chorda tympani, salivation &amp; taste are spared.</td>
<td></td>
</tr>
</tbody>
</table>

NURSE’S RULE (CENTRAL PALSY)
- only contralateral lower face is paralyzed (esp. perioral muscle); mild upper face weakness is possible.
- see table above
- paralyzed m. buccinator → chewing difficulty (food pools in vestibulum oris)
- paralyzed m. orbicularis oris → flattened nasobulbal 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 palidus) – volitional (pyramidal) and emotional (extrapyramidal) components may be affected separately.

FACIAL NERVE DISORDERS

CN7 (2)
NIGHT

DAY

Eye protection

All complete

5.

4.

3.

2.

1.

c)

b)

a)

• motor CN7 nucleus lies:
  - within RP of caudalpons;
    - between trigeminal nucleus (dorsally) and superior olivary nucleus
      (ventromedially).
  *temporal sound localization

• ipsilateral full (upper & lower) face paralysis;
  - paralysed m. orbicularis oculi → lagophthalmos, atonic epiphora.
  - when patient attempts to close eye, eyeball diverts upward (Bell phenotype).

• tearing, salivation, taste intact.

• contralateral hemplegia may be seen (corticospinal tract has not crossed yet) - MILLARD-GUBLER syndrome.

• ipsilateral CN6 palsy may be seen.

SUPERFICIAL TRIGEMINAL ANGEL SYNDROME

• ipsilateral full (upper & lower) face paralysis.

• stapedius paralysis (HYPERacusis - painful sensitivity to loud sounds), tearing[EXOPHTHALMIA], salivation[XEROSTOMIA], taste.

• CNS is often involved; cerebellum and CN5 can also be involved.

FACIAL NERVE SYNDROME

a) LAVERGNE SEGMENT - must full facial palsy (SVE, SVA, GVE, GVA, GSA)

b) TYPANIC SEGMENT - lacrimation spared.

c) 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

1. Check cornea for exposure keratitis!

2. AUDIOMETRY should be performed in all patients.

3. NEUROIMAGING (MRI, CT) indications:
   1) unclear etiology
   2) recurrent / progressive / atypical paralysis
   3) trauma.

4. ELECTROPHYSIOLOGY:
   1) ENoG (electroneurography) - in acute unilateral complete paralysis (if threshold is 3.5 mA greater than normal side or if ≤90% arelets are lost, there is poor prognosis for spontaneous recovery (during days 4-21).
   - even in complete transaction, 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!

   2) voluntary EMG is helpful in long-standing or congenital facial paralysis.

   3) trigeminofacial 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!!!

   4) If facial reanimation procedure is considered (in long-standing or congenital facial paralysis) → facial MUSCLE BIOPS (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 to severity of corneal epithelial disruption):

DAY:

1) artificial tears frequently (q5-15min); best are "for sensitive eyes", "non-allergic", or "preservative free" artificial tears; rabbit tears is preservative that can be particularly irritating!

2) room humidifiers

3) 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 kids 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!

4) sunglasses while outside (avoid contaminated air!)

5) moisture chamber:

6) 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

1) ophthalmic ointment (e.g. with dexamethasone) - thicker than artificial tears due to addition of mineral oil.

If corneal exposure occurs, ensure cornea is covered immediately

http://www.bellspalsy.ws
2) eye patch or taping closed affected eyelid*: if not done correctly eye 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 muscle resistance to 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 undeserved!
  – 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) 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.

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) 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.
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.

SPECIFIC DISORDERS

<table>
<thead>
<tr>
<th>Facial paralysis</th>
<th>Trumatic</th>
<th>Nontrumatic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Derrmanal injury</td>
<td>Temporal/ facial fracture</td>
<td>Facial Branch function affected</td>
</tr>
<tr>
<td>Periheral lesion</td>
<td>Facial Branch function/intact</td>
<td>Neurologic work-up</td>
</tr>
<tr>
<td>Observation</td>
<td>Observation</td>
<td>Observation</td>
</tr>
<tr>
<td>(needed)</td>
<td>(needed)</td>
<td>(needed)</td>
</tr>
<tr>
<td>EABG</td>
<td>EMC</td>
<td>XAEC</td>
</tr>
<tr>
<td>Observation</td>
<td>Observation</td>
<td>Observation</td>
</tr>
<tr>
<td>(needed)</td>
<td>(needed)</td>
<td>(needed)</td>
</tr>
<tr>
<td>Surgical exploration</td>
<td>Surgical exploration</td>
<td>Surgical exploration</td>
</tr>
<tr>
<td>EABG</td>
<td>EMC, audio, meter</td>
<td>XABG, EMG</td>
</tr>
<tr>
<td>Further tests:</td>
<td>Further tests:</td>
<td>Further tests:</td>
</tr>
<tr>
<td>Audiology:</td>
<td>CT of patiens bone &amp;needed</td>
<td>MRI for unilateral lesion detected not... , if needed</td>
</tr>
<tr>
<td>CT of patiens &amp;needed</td>
<td>MRI</td>
<td>MRI for unilateral lesion detected not... , if needed</td>
</tr>
</tbody>
</table>

BELL’S PALSY (s. idiopathic acute facial paralysis)

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: 3-10 days before.
most commonly accepted cause is herpes simplex-1 infection (reactivation of HSV from geniculate ganglia); other suggested causes – other viruses, vascular ischemia.
Mechanism – inflammatory nerve edema within rigid (falloppian) canal.
Alternative cause / mechanism – body alters elongate and become tortedous (may do so even in third decade of life) – sudden shift of arterial loop in cerebellopontine angle:
A) rapid stretch of CN7-B 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 musty fullness (pain at stylonodidastum foramen), periauricular paresthesia 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, dysguesia, 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!!!
- 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 considered.
- 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%; remain 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 (40 mg/d for 5 days + taper down by 10 mg/day for 5 days) – level A recommendation!
-ACYCLOVIR (400 mg 5x/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 (NEURULYSIS).
- Entire infratemporal facial nerve; – because presumed site of entrapment is meatal foramen (beginning of labyrinth segment).
- Facial 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 paralyzes:
A) Patient first seen on days 0-14 – ACYCLOVIR + PREDNISONE – follow-up in 5 days:
- a) paralyzes – 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 has elapsed from onset of total paralysis:
- a) < 90% degeneration – continue ACYCLOVIR + PREDNISONE until full course.
- b) > 90% degeneration – surgical NEURULYSIS.
- 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
FACTORS IN STUDY
FACIAL NERVE DISORDERS
CN7 (5)
3) long-continued low degree fever.

**MELKERSSON-ROSENTHAL syndrome**
- recurrent CN7 paralysis
- recurrent and eventually permanent facial (particularly labial) edema [cheilitis granulomatosa]
- fissured tongue

**MOBIUS syndrome**
- failure of motoneuron development in bilateral CN6 & 7 nuclei → congenital facial diplegia with abnormalities of horizontal (or 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. even during sleep!
- muscles are normally relaxed between twitches.
- provoked by voluntary facial motion, emotional stress.
- a) compression of motor nerve root at brain stem by aberrant / ectopic vascular loop (→ focal demyelination → ectopic excitation / epileptic 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:
  - EMG – 5-20 Hz bursts of muscle action potentials.
  - treatment:
    1) BOTOX® 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.
- permanent relief:
  - Janetta procedure: decompressing vessels at root entry zone – see p. Op350 ->
  - anastomosing CN7 with CN11 or CN12.

**MYOKYMIA**
- progressive, irregular twitches 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.
- etiologies: (grave! vs. hemifacial spasm).
- recurrent and eventually permanent facial (particularly labial) edema [cheilitis granulomatosa].
- fissured tongue.
- diagnosis:
  - EMG – 5-20 Hz bursts of muscle action potentials.
  - treatment:
    1) BOTOX® 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.
- permanent relief:
  - Janetta procedure: decompressing vessels at root entry zone – see p. Op350 ->
  - anastomosing CN7 with CN11 or CN12.

**FACIAL NERVE TRAUMA**

**RE-INNERVATION SYNDROMES**

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

**SYNKRINESIS** - unintentional muscle motion when another muscle is voluntarily contracted.
• example - jaw opening causes closure of eyelids (jaw-winking).
• mechanism - re-inervation 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 pterygopatine 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 >>