## Neurological examination

- Examination of
  - meningeal signs
  - cranial nerves (I-XII)
  - motor system (muscle bulk, tone, power, involuntary movements)
  - sensory system
  - reflexes (pathological and physiological reflexes)
  - co-ordination, cerebellum
  - speech
  - conscious state
  - + short psychiatric examination

## MENINGEAL SIGNS

- may be caused by:
  - meningitis (bacterial or viral infection of meninges)
  - blood in the subarachnoidal space
  - infiltration of meninges by carcinoma cells
  - increased intracranial pressure
  - dehydration



#### **VISUAL FIELD - LENS - reversed image**



- **1.** The lens produces reversed image
- 2. Fibers, originating from the upper part of the retina keeps their superior/upper position through the whole optic pathway

## Visual field



# Oculomotor (III), trochlear (IV), and abducent (VI) nerves

- Oculomotor nerve (III) innervates: medial rectus, superior rectus, inferior rectus, inferior oblique muscles + superior palpebral levator muscle, ciliary muscle, pupillary constrictor muscle
- Trochlear nerve (IV) innervates: superior oblique muscle
- Abducent nerve (VI) innervates: lateral rectus muscle

## Nuclei of culomotor (III), trochlear (IV), and abducent (VI) nerves

- Oculomotor nerve
  - Main nucleus (mesencephalon) of oculomotor nerve controls medial rectus, superior rectus, inferior rectus, inferior oblique muscles + superior palpebral levator muscle
  - Edinger-Westphal nucleus (mesencephalon) controls ciliary muscle and pupillary constrictor muscle
  - Perlia nucleus (unpaired nucleus in mesencephalon) controls medial rectus muscles and pupillary constrictor muscles at convergence reaction
- Trochlear nerve:
  - Trochlear nucleus (mesencephalon) controls superior oblique muscle
- Abducent nerve:
  - Abducent nucleus (pons) controlslateral rectus muscle

Function of external eye muscles (indicated by arrows) (Muscles innervated by IIIrd, IVth or VIth cranial nerves are indicated by different colours)





#### Eye movement disorder in case of abducent nerve lesion

## Possible causes of double vision

- Damage of
  - Nucleus or nuclei responsible for innervation of eye muscles (III., IV., VI.)
  - Nerves responsible for innervation of eye muscles (III., IV., VI.)
  - Neuromuscular junction (myasthenia gravis)
  - Eye muscles (e.g. endocrine ophthalmopathy)
  - Displacement of one of the eyes e.g. due to orbital tumor.

# Possible causes of ptosis

- Oculomotor nerve innervates: medial rectus, superior rectus, inferior rectus, inferior oblique muscles + <u>superior palpebral</u> <u>levator muscle</u>, ciliary muscle, pupillary constrictor muscles
- Superior palpebral levator muscle has <u>dual innervation</u>: sympathetic (30%) and oculomotor (III.) nerve (70%)

Lesion of III. nerve

Sympathetic lesion (Horner triad)



## WHAT IS GAZE?

Difference between eye movement disturbance and gaze disturbance

- Eye movement disturbance Gaze disturbance
  - Dissociated eye movement
  - Arrows indicate the instructions (Look at forward, left side and then right side!)



**Double vision** 

- Conjugated eye movement
- Arrows indicate the instructions (Look at forward, left side and then right side!)



right sided gaze disturbence: None of the eyes move to the right side. No double vision!

What assures the conjugated movements of the eyes? Control of IIIrd, IVth and VIth cranial nerve functions





## Gaze - eye movement

- Cortical gaze centres
  - Br8 (frontal lobe): saccadic eye movement
  - Br19 (occipital lobe): persuit eye movement
- Subcortical gaze centres
  - Interstitial nucleus, Posterior comissura nucleus
  - Pontin and mesencephalic reticular formation
- Medial longitudinal fascicule
- III., IV., VI. cranial nuclei

In case of damage double vision

In case of

damage -

no double

vision

## **Facial nerve**

#### **Two parts**

- 1. <u>facial nerve</u>: motor innervation of facial muscles
- 2. intermedius nerve (Wriesberg),
  - parasympathic <u>secretomotor</u> function (saliva and tear production),
  - Insteme (special viscerosensory)
    - (taste on the first 2/3 of the tongue),
  - somatosensory fibres
    - (spf. sensation in the external auditory canal).

#### Facial nerve

#### Intermedius nerve

#### **Facial nerve**

#### Motor nucleus of facial nerve

**Corticopontine tracts** from both sides (bilateral supranuclear innervation) to the parts of the nuleus innervating the forehead and orbicularis oculi muscle;

**Corticopontine tracts** only from the contralateral side (contralateral supranuclear innervation) to the part of the nuleus innervating the orbicularis oris muscle.

+ Fibers from the thalamus, hypothalamus, extrapyramidal system (emotional mimic)

#### Superior salivatory nucleus

- lacrimal gland,
- submandibular gland
- sublingual gland

Supranuclear innervation and reflexes to tear production: from hypothalamus (emotions), and from trigeminal sensory nuclei (irritation of conjunctiva), to saliva production: from the olphactory system, and from the solitary tract. <u>Solitary nucleus</u>

Nucleus of taste sensation not only from the facial nerve, but also from the IX. and X. nerves.

Thalamus

Postcentral gyrus (above the insula) Spinal (descend) nucleus of the trigeminal nerve



The lower motor neuron (nucleus) is controlled by the upper motor neuron







Part of the facial nucleus which innervates the muscles around the mouth Part of the hypoglossal nucleus which innervates the genioglossus muscle

#### The <u>nucleus</u> or <u>a part of the nucleus</u> is damaged





Part of the facial nucleus which innervates the muscles around the mouth receives only contralateral but not ipsilateral supranuclear innervation CENTRAL FACIAL PALSY

Wrinkling of forehead Closure of eyes <del>Showing the teeth</del>

Name: right sided central facial palsy





PERIPHERAL FACIAL PALSY

Wrinkling of forehead Closure of eyes Showing the teeth

Name: right sided peripheral facial palsy



Right sided central facial palsy The lesion is somewhere between the motor cortex (UMN) and the facial nucleus Right sided peripheral facial palsy The lesion is in or below the facial nucleus



#### - Facial nerve -

#### **Facial nerve**

#### Motor nucleus of facial nerve

- Pons, tegmentum, ventro-lateral part;
- Genu internum (internal knee) around the abducent nucleus;
- Intermedius fibers join to the facial nerve;
- Leaves the pons (pontocerebellar angle).
- Supranuclear innervation from praecentral gyrus:
- Corticopontin tracts from both sides (bilateral supranuclear innervation) to the parts of the nuleus innervating the forehead and orbicularis oculi muscle;
- Corticopontin tracts only from the contralateral side (contralateral supranuclear innervation) to the part of the nuleus innervating the orbicularis oris muscle.
- + Fibers from the thalamus, hypothalamus, extrapyramidal system (emotional mimic)z

#### Intermedius nerve

#### Superior salivatory nucleus

- sphenopalatine ggl.  $\rightarrow$  lacrimal gland,
- submandibular ggl.  $\rightarrow$  submandibular gland
- sublingual ggl.  $\rightarrow$  sublingual gland

Supranuclear innervation and reflexes - to tear production: from hypothalamus (emotions), and from trigeminal sensory nuclei (irritation of conjunctiva), - to saliva production: from the elaboratory system, and

from the olphactory system, and from the solitary tract.

#### Solitary nucleus

Pons, medulla oblongata. Nucleus of taste sensation not only from the facial nerve, but also from the IX. and X. nerves. After synapse in the nucleus, fibers cross to the contralateral side, reaches the thalamus, after synapse in the thalamus, fibers run to the postcentral gyrus (above the insula). Spinal (descend) nucleus of the trigeminal nerve



Prosoplegia (paralyzed facial muscles) +ageusia (disturbance of taste sensation) +hyperacusis

+xerophthalmia (no tear production)
+tinnitus, deafness, vertigo, vomitus

Prosoplegia (paralyzed facial muscles) +ageusia (disturbance of taste sensation) +hyperacusis

+xerophthalmia (no tear production)

Prosoplegia (paralyzed facial muscles) +ageusia (disturbance of taste sensation) +hyperacusis

Prosoplegia (paralyzed facial muscles) +ageusia (disturbance of taste sensation)

Prosoplegia (paralyzed facial muscles)





Sir Charles Bell 1774 - 1842

Most frequent form of peripheral facial palsy.

**Epidemiology** 

- Praevalance: : 640 2.000 / 100.000
- Incidence (increases with age): General : 50 / year / 100.000
   20 y : 10 / year / 100.000
   80 y : 60 / year / 100.000
- Male to female ratio = 1:1
- Recurrance: 7 %
- Right side:left side = 63 : 37
- Increased risk: diabetes, pregnancy

# Bell's palsy

### ➢ Pathogenesis

- Not known. Draughty place. <u>Herpes</u> <u>simplex type 1 virus activation?</u>
- Nerve damage occurs because of compression due to inflammation, edema, micro-bleeding in the very narrow facial canal (Fallop canal). At first <u>demyelinisation</u> later <u>axonal-damage</u> <u>develop</u>.

# Bell's palsy - Treatment

#### • Prednisolon, methylprednisolon

- Adults: 40-80 mg/die
- Children: 1 mg/kg/die
- After 5 days decrease the dose gradually.
- <u>Acyclovir</u>
  - Adults 5 x 400 mg for 7 days
  - Children 80 mg/kg/die for 5 days
  - Most effective in Ramsay-Hunt syndrome!
- **Protection of the eye!** 
  - Eye drops (antibiotics), if necessary blepharoraphia.
- Prevention of atrophy of facial muscles
  - Electrotherapy
  - Active gymnastics of facial muscles

<u>Ramsay – Hunt syndr.</u>: right sided peripheral facial palsy + eruptions in the external auditory canal. Herpes zoster infection from the geniculate ganglion.

# Similarities of nuclei of the VIIth, IXth and Xth cranial nerves

Nerve	VII.	IX.	Х.
Motor nucleus	Motor nucl. of the facial nerve (facial muscles)	Ambiguus nucl. (muscles of pharynx)	Ambiguus nucl. (muscles of larynx)
Parasympathetic nucleus	Suprior salivatory nucl. (lacrimal and salivatory glands)	Inferior salivatory nucl. (parotid gland)	Dorsal nucleus of the vagus nerve
Special sensory nucleus	Solitary tract nucl. (taste)	Solitary tract nucl. (taste)	Solitary tract nucl.
Somatosensory nucleus	Spinal nucl. of the trigeminal nerve (ear)	Spinal nucl. of the trigeminal nerve (pharynx, middle ear)	Spinal nucl. of the trigeminal nerve (ear)

## Damage of the glossopharyngeal and vagal nerves

- Symptoms
  - Swallowing problem (= dysphagia, aphagia)
  - Difficulty in articulation (=dysarthria, anarthria)
  - Vocal cord palsy (dysphonia, high pitched voice)
  - Disturbance of taste sensation (clinically not relevant
  - Vegetative signs  $\rightarrow$  breathing freq., pulse rate increases

# Hypoglossal nerve (XII.)

- Only motor function
- Innervation of muscles of the tongue
- Most caudal nucleus in the medulla oblongata Leaves the skull: canalis nervi hypoglossi
- Special supranuclear innervation of the nucleus!
   → Genioglossus muscl !








### CENTRAL HYPOGLOSSAL LESION

Deviation to the contralateral side

Name: right sided central hypoglossal lesion



**LESION** Deviation to the ipsilateral side

+atrophy +fasciculation

Name: right sided peripheral hypoglossal

## Damage of hypoglossal nerve functions

- Lesion of the XII. nucleus or the nerve:
  - ( = periferial type hypoglossal palsy)
    - Very early atrophy (10-14 days)
    - Fibrillation, fasciculation
    - Protruded tongue deviates towards side of weakness (ipsilateral to the lesion)
- Lesion of the supranuclear innervation
  - ( = central type hypoglossal palsy)
  - No atrophy
  - No fibrillation
  - Protruded tongue deviates towards side of weakness (contralateral to the lesion)

## Examination of hypoglossal nerve

- Ask patient to open mouth
  - Inspect tongue in the mouth
  - Look for evidence of atrophy, fibrillation
- Ask patient to protrude tongue →
  deviation?



## Bulbar and pseudobulbar palsy

- Site of the damage
  - Bulbar lesion: brain stem (cranial nuclei)
  - Pseudobulbar lesion: bilateral supranuclear fibers

### Damaged functions

- swallowing, phonation, articulation, chewing
- dysphagia, dysarthria, difficulty of tongue movements

### • Bulbar palsy:

- Depressed gag & soft palate reflex
- Atrophy of the tongue
- Depressed jaw reflex

### • Pseudobulbar palsy:

- Gag & soft palate reflex can be elicited
- NO tongue atrophy
- Increased jaw reflex

#### Supranuclear innervation of the ambiguus nuclei



## **Bilateral supranuclear innervation of the ambiguus nuclei** (bilateral supranuclear damage, pseudobulbar palsy)



### Bilateral damage of the ambiguus (and hypoglossal) nucl. →bulbar palsy



## Bulbar and pseudobulbar lesions

- Bulbar lesion
- Medulla obl. or peripheral nerves are damaged
- Dysarthria, dysphagia
- Absent gag- and palatal reflexes
- Protrusion of the tongue is not possible
- Atrophy and fasciculation in the tongue

- <u>Pseudobulbar lesion</u>
- Damage of bilateral supranuclear fibers
- Dysarthria, dysphagia
- Gag- and palatal reflexes are preserved
- Protrusion of the tongue is not possible
- Neither atrophy, nor fasciculation in the tongue
- Forced laughing and crying

## Motor system

- CENTRAL NERVOUS SYSTEM: Upper motor neurone, originating mainly from the frontal lobe (motor cortex)
- PERIPHERAL NERVOUS SYSTEM: Lower motor neuron, originating from the anterior horn of the spinal cord. Axons form the anterior radix, and then peripheral nerve. The impulse reaches the muscle through the neuromuscular junction.



### Pyramidal tract

- Preacentral gyrus
- Internal capsule
- Cerebral pedunculus
- Mesencephalon
- Pons
- Medulla oblongata
- Decussation
- Lateral coloumn

### Extrapyramidal system

 Cortex, basal ganglia (nucl. lentiformis, nucl. caudatus, nucl. ruber, substantia nigra, nuclei tegmenti, oliva inferior, nucl. subthalamicus, cerebellar nuclei ...)

## Motor system

- Atrophy? muscle bulk
- Fasciculation, involuntary movements
- Muscle tone
- Motor power:
  - Paresis: weaker muscle
  - Plegia: no movement
- Monoparesis, Hemiparesis, Tetraparesis, Paraparesis



## Role of the upper motor neurone

- Activation of lower motor neurones (alpha motor neurone) → voluntary movement
- Regulation of gamma motoneurones (inhibitory impulses)
  - Damage of upper motor neurone results in overactivation of the gamma motoneurones (stop of inhibition) → contraction of muscle spindle – contracted muscle spindle will be more sensitive → more sensitive muscles → increased tone, brisk reflexes, pathological reflexes
  - Muscle spindle
    - is activated by stretch of the muscle: Ia, motoneuron, muscle contraction
    - is inactivated by muscle contraction (stop of stretching)
    - is made more sensitive by impulses through the gamma motoneuron contracted muscle spindle

Notes: fibers from the extrapyramidal system (rubrospinalis, reticulospinal tracts...), run together with the pyramidal tract. Damage of these fibers contribute to increased muscle tone.

## Innervation of muscles



### Muscle tone

(resistance during the passive movement of the muscle)

- Decreased tone
- Hypotonicity
  - Usually caused by peripheral damage
  - Damage of the reflex arch

- Increased tone
- Spasticity or rigidity
- Spasticity
  - Increased tone and reflexes
  - Damage of the UMN
  - Clasp-knife sign
- Rigidity
  - Constant resistance
  - Lead-pipe sign
  - Damage of the extrapyramidal system
  - If it is accompanied by tremor: cog-wheel sign

## Motor power

- Monoparesis, Hemiparesis, Tetraparesis, Paraparesis
- Paresis: weaker muscle
- Plegia: no movement
- Localisation: spasticity always refers to central lesion, but central lesion does not cause always spasticity!!!

## Examination

- Atrophy, fasciculation: observation
- Muscle tone: resistance during passive movement
- Motor power:
  - from distal to proximal
  - Comparison of
    - Right and left side
    - Different myotoms

# Comparison of signs uf upper and lower motor neuron lesions

Sign	UMN Lesions	LMN Lesions
Atrophy	Mild global, disuse atrophy in the affected muscle groups	Yes
Fasciculations	No	Yes (mainly in case of LMN lesion)
Weakness	Yes	Yes
Deep reflexes	Increased (+pyramidal signs)	Decreased
Superficial reflexes	Decreased	Decreased
Muscle tone	Usually increased (spasticity), but could also be decreased in the acute phase of the UMN lesion	Decreased

## Central and peripheral lesions - differences

### • Central lesion

- Usually spasticity
- Global, disuse atrophy
- Paresis
- Brisk or increased reflexes
- Pyramidal signs
- Decreased superficial reflexes

- Peripheral lesion
  - Hypotonicity
  - Individual atrophy
  - Paresis
  - Decreased or absent reflexes
  - No pyramidal signs
  - Decreased superficial reflexes



## Sensorium Types of sensory dsiturbances

## Sensory system

- Superficial sensation (vital, protopathic)
  - Light touch
  - Pain
  - Temperature

**SPINOTHALAMIC PATHWAY** 

- Deep sensation (gnostic, proprioceptive, epicritic)
  - Vibration
  - Discriminating touch (two points discrimination)
  - Graphaesthesia (dermolexia)
  - Joint position sense

#### **POSTERIOR COLOUMN PATHWAY**

### + Subconscious proprioception

SPINOCEREBELLAR PATHWAY



### Sensory system ,,conscious" sensation

- Spinothalamic pathway
  - Vital, or
  - Prothopathic, or
  - Superficial sensation

- Posterior coloumn pathway
  - Gnostic, or
  - Proprioceptive, or
  - Epicritic, or
  - Deep sensation

- Crossing to the contralateral side at the level of entering the spinal cord → spinothalamic tract → Thalamus → Cortex
- Through posterior coloumn ascends to the medulla oblongata → Crossing to the contralateral side → Thalamus → Cortex

### 3 neurons, 2 synapses, 1 crossing over

## Examination

### **Superficial sensation**

- Light touch: cotton wool
- Pain: pin prick
- Hot and cold stimuli: tubes with hot (40 C) and cold water (22 C)

### **Deep sensation**

- Vibration sense: vibrating tuning fork on bony prominences
- Graphaesthesia or dermolexia: draw numbers to the patient's skin
- Joint position sense: hold the last phalanx of one of the fingers and toes AT THEIR SIDES. The patient should recognize which finger or toe is held. Move the last phalanx up and down; the patient should recognize the direction of the movement.

## Examination (cont.)

- Compare the sensory stimuli on
  - the right and left sides (hemisensory loss?)!
  - the proximal and distal parts (polyneuropathy?)!
  - the supplying territories of the nerves (lesion of a peripheral nerve?)!
  - the supplying territories of the radices (lesion of a radix?)!

## **Types of sensory disturbances**

### Dissociated sensory disturbances

- Spinothalamic pathway and dorsal coloumn pathway run at different parts of the spinal cord
- They can be damaged separately
- → deep and superficial sensation might be disturbed separately
- E.g. only supericial sensation is disturbed in syringomyelia

## **Types of sensory disturbances**

sensory cortex  $\leftarrow$  thalamus  $\leftarrow$  spinal cord  $\leftarrow$  roots  $\leftarrow$  nerves  $\leftarrow$  receptor

- Peripheral nerve damage (like a map)
- Distal type of sensory disturbance (polyneuropathy gloves and socks) (alcohol or diabetes)
- Radicular type dermatomal
- Spinal cord damage altitudinal level, segmental and funicular features
- Brainstem damage alternating symptoms
- Hemi-sensory disturbance cortical or subcortical damage



Distal type of sensory disturbance (<u>polyneuropathy</u> – glove and socks)

> Symmetric, distal, or ,,glove-socks-like'' distribution of sensory disturbance in polyneuropathy







## **Spinal cord damage**

- Transsectio medullae spinalis ("horisontal laesion)
- Hemisectio medullae spinalis (Brown-Sequard syndrome)
- Damage of central grey matter (syringomyelia)

## Alternating symptoms (ALWAYS REFER TO BRAINSTEM DAMAGE)



## Cerebellar signs

- Note that cerebellar hemisphere is responsible for the coordination of ipsilateral extremities.
- →one sided cerebellar signs indicate ipsilateral cerebellar damage
#### Function of cerebellum

- Maintenance of balance and posture
- Co-ordination of voluntary movements
- Motor learning
- Co-ordination of muscles involving in speech

#### Main connections of the cerebellum



http://humanphysiology.academy/Neurosciences%202015/Chapter%205/A.5p%20Cerebellar%20Pathways.html

### Cerebellar examinations

- Romberg's test
- Blind walking
- Barany test
- Check the nystagmus
- Dysmetria (finger-to-nose test, heel-to-knee test)
- Dysdiadochokinesis (rapid alternating movements) – rapid, alternating pronation and supination, or arm-rolling test
- Rebound phenomenon (Holmes test)



## Rapid, alternating pronation and supination of the forearm.

Fig. 3.17 Testing of diadochokinesis by rapid pronation and supination of the forearms.

a



**Examination of rebound phenomenon** 

normal abnormal

http://www.neurologicaltx.medicalph.com/neu-fun\_3-EUL-1.html



http://www.neurologicaltx.medicalph.com/neu-fun\_3-EUL-1.html

## Aphasias – dominant hemisphere

- Broca aphasia (=non-fluent, motor, expressive, anterior) aphasia
  - gyrus frontalis inferior (Br44,45)
    - Non-fluent speech
    - Good comprehension
    - Anxious because of the symptoms



- temporal or parietal lobe (Br22,39,40)
  - Fluent speech, use of non-existing words, difficult or impossible to understand
  - Bad comprehension
  - Does not realise her/his symptoms



## Aphasia

#### • Motor aphasia

- Gyrus frontalis inferior
- Non-fluent speech
- Good comprehension
- Rhythm is disturbed

- <u>Sensory aphasia</u>
- Temporal or parietal lobe
- Fluent speech
- Bad comprehension
- Normal rhythm
- Neologism, paraphrasia

## Examination of aphasias

#### • In case of suspicion of motor aphasia

- Ask questions from the patient (where do you live, what is your name, where are we now?)
- Ask the patient to name objects (pen, eye-glasses, thumb)
- Ask the patient to count, name the days of a week...

#### • In case of suspicion of sensory aphasia

- Ask the patient to perform simple instructions
  - Close your eyes! Protrude your tonge! Raise your hand!
- If he/she can perform them, ask to perform more complicate instructions
  - Touch your left ear with your right second finger with eyes closed!

#### • Examination of cunductive aphasia

- Ask the patient to repeat a sentence!

#### Agnosia, apraxia

- Agnosia: disturbance of recognition despite good visus/sight, hearing, sensory system...
- Apraxia: disturbance of execution despite good muscle power and co-ordination

### Gerstmann syndrome

- Dominant angular gyrus (parietal lobe)
  - Dyscalculia
  - Agraphia
  - Right-left confusion
  - Finger-agnosia

# Signs of non-dominant parietal lobe dysfunction

- Disturbance of orientation in space (e.g. in the flat)
- Dressing apraxia (cannot dress properly)
- Constructive apraxia (cannot build a cube from 8 identical small cubes)
- Anosognosia (does not recognize his/her disease mostly the left sided hemiparesis)
- Neglect syndrome (subdominant parietal lesion)
  - Does not recognize the stimuli from one direction of space