

CLASS 6: Motor activity. Methods of examination. Cerebellar syndromes. Syndromes of imbalance.

Cerebellum

Anatomo-physiology of the cerebellum (the main parts of cerebellum and their afferent and efferent pathways).

1. Vestibulocerebellum – corresponds to the lobulus flocculonodularis

Afferent pathways:

- Directly from the neurons of ganglion vestibulare – informative for head position
- From vestibular nuclei- forming the vestibulocerebellar tract
- From proprioceptors in the cervical muscles and reticular formation

Efferent pathways:

- Ipsilateral vestibulospinal lateral tract – it controls axial muscles keeping balance against gravity
- Bilateral paramedian vestibulospinal medial tract – it controls the movements of the head and eyes
- Vestibular nuclei – connected by fasciculus longitudinalis medial control the conjugate eyes movements

Functions:

- Control of the equilibrium – head and body position - by coordinating the muscle tone by axial, postural and vestibular reflexes.
- Control the eye movements and synchronize them to those of the head and the body

2. Spinocerebellum – consists of vermis and the paramedian part of cerebellum's hemispheres (paraflocculus)

Afferent pathways:

1. Direct spinocerebellar pathways
 - Tr. spinocerebellaris dorsalis (Flechsig) – an uncrossed pathway starting from posterior horn, ipsilaterally passes through the peduncle cerebellar inferior and transmits the proprioceptive sensation related to the posture and movements of the body and the limbs.
 - Tr. spinocerebellaris ventralis (Gowers) – a double crossed (spinal cord and mesencephalon) pathway, ipsilaterally passes through the peduncle cerebellar superior and transmits the proprioceptive sensation for the upper limbs
2. Indirect spinocerebellar pathways

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- Tr. cuneocerebellaris – a homolateral pathway that transmits additional afferent information for superficial and deep sensation for the cervical area and the upper limbs
- Tr. reticulocerebellaris – somatosensory information, received by the reticular formation

Efferent pathways

1. From Purkinje cells in vermis through nucl. fastigii and from this nuclei
 - Tr. Cerebellothalamicus – after a crossing in decussatio pedunculorum cerebellarium superiorum (Stilling) ends in nucl. ventralis lateralis and nucl. ventralis anterior of the thalamus. It controls the voluntary movements of the ipsilateral side of the body and regulates the proximal limb muscles via tr. Corticospinalis ventralis.
 - The descending pathways via the nucl. fastigii go bilaterally through the peduncle cerebellar inferior to the brainstem reticular formation and vestibular nuclei. There are formed tr. Reticulospinalis and tr. Vestibulospinalis as a part of medial descending system controlling muscle tone of axial and proximal extensor muscles maintaining the body posture
2. From Purkinje cells in the intermediate part of cerebellar's hemispheres to the homolateral nucl. globosus and nucl. emboliformis and from these nuclei after decussating in the peduncle cerebellar superior originate:
 - An ascending efferent pathway, as a part of tr. cerebellothalamicus - its projections via the ventrolateral nucleus of the thalamus to the motor cortex. By tr. corticospinalis lateralis crossed in decussatio pyramidum its impulses pass to the motoneurons in the anterior horns and control the distal muscles.
 - A descending efferent pathway – forms tr. cerebellorubralis and from nucl. ruber originate tr. rubrospinalis, crossed in decussatio of Forel ends in motoneurons of the spinal cord and control the distal flexor muscles of the limbs.
3. Tr. rubroreticularis and tr. rubroolivaris – via the peduncle cerebellar inferior go back to the cerebellar cortex and form so called a cerebello-olivo-cerebellar circle (a feedback system modulating the afferent information from the motor cortex and the spinal cord

Functions:

- Control and correct the body and limbs muscle movements by comparing the planned motor commands from the motor cortex with the afferent information from proprio- and exteroceptors and spinal cord neurons activity.
- Regulate the muscle tone of agonists and antagonists related to the motor act through the activity of gamma-motoneurons

3. Cerebrocerebellum (pontocerebellum) – the lateral part of the cerebellar hemispheres, corresponding to **neocerebellum**

Afferent pathways – they start from areas in frontal, parietal, temporal and occipital lobes, pass through crus cerebri like tr. frontopontinus and tr.parieto-temporo-occipito pontinus and reach nuclei pontis and by tr. pontocerebellaris decussate and end at Purkinje cells in contralateral cerebellar hemisphere

Efferent pathways – originate from nucl. dentate and like tr. dentatothalamicus decussates and ends at nucl. ventralis lateralis and nucl. ventralis anterior of the thalamus. After that the projections reach the motor and premotor cortex. In this way is formed a feedback loop (cortex-pons-cerebellum-thalamus-cortex), important for performing the fine and brisk limb movements.

Functions:

- coordinate and control the synergistic and antagonistic muscles related to the exact time in performance of the motor act
- process the information related to the motor act planning and also coordination in time and place of the motor activity and transferring it to the motor and premotor cortex for realization
- participate in development of motor cognitive functions.

Syndromes of imbalance

Ataxia is a term used for a loss of coordination.

The Vermis (vestibulo-spinocerebellar) syndrome:

- Static ataxia – a loss of balance of the body in stand up position; Romberg's test is negative (the patient is unstable with opened and closed eyes in undefined position)
- Locomotor ataxia – a wide-based (ataxic) gait
- Disturbance of the eye movements
- Body and head tremor (titubation)
- Muscle hypotonia

Clinical examples for a vermis syndrome: medulloblastoma, varicella encephalitis, etc.

Neocerebellar (cerebrocerebellar) syndrome:

- Dynamic ataxia – ataxia of the limbs, accompanied by dismetria, intention tremor and dysidiadochokinesia
- Static and locomotor ataxia – Romberg's test is negative (the patient is unstable with opened and closed eyes to the side of the lesion)
- Muscle hypotonia

- Dyssynergia
- Saccadic or explosive speech
- Disturbed handwriting (megalography)

Clinical examples for neocerebellar syndrome: brain tumors (astrocytoma, hemangioblastoma), multiple sclerosis, brain stem stroke (Wallenberg's syndrome), acute intoxications by alcohol, barbiturates, phenytoin, lithium etc, chronic intoxications with alcohol, mercury, lead, thallium, some viral encephalitis, prion infections, metabolic disorders like hepatolenticular degeneration, hypothyroidism, paraneoplastic cerebellar degenerations (carcinoma ovarii), Friedreich's disease, hereditary spinocerebellar degeneration, ataxia teleangiectatica, etc.

Differential diagnosis of a syndrome of imbalance

A syndrome of a central vestibular lesion:

- Ataxia – often bilateral with asymmetrically expressed symptoms
- Spontaneous nystagmus, depending on the side of the lesion – horizontal, vertical
- No systemic vertigo, often dizziness
- Lack of autonomic symptoms
- Focal neurological symptoms, caused by a lesion of the brain stem

Clinical examples for a syndrome of central vestibular lesion: TIA in vertebrobasilar system, stroke (Wallenberg's syndrome), hemorrhages, multiple sclerosis, brainstem tumors or trauma, alcohol, intoxications, etc.

A syndrome of a peripheral vestibular lesion:

- Ataxia
- Spontaneous nystagmus – horizontal or horizontal-rotatory
- Systemic vertigo – a subjective feeling for rotation of the surrounded objects
- Autonomic symptoms – nausea, vomiting, sweating

Clinical examples for a syndrome of peripheral vestibular lesion: Meniere's disease, benign paroxysmal vertigo, neurinoma of statoacoustic nerve, tumors at pontocerebellar angle, commotio labyrinthi, use of ototoxic medications, etc.

Ataxia, caused by a lesion in posterior columns (fasciculus gracilis and fasciculus cuneatus)

- Static and locomotor ataxia - Romberg's test is positive (the patient is unstable only with closed eyes)
- A damage or even a loss of proprioceptive sensation
- Intact superficial sensation

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- In some cases – hyperreflexia

Clinical examples for ataxia, caused by a lesion in posterior columns: multiple sclerosis, tabes dorsalis, funicular myelosis in Vit. B 12 deficiency, Friedreich disease, etc.

Ataxia, caused by a lesion of peripheral nerves (pseudotabes polyneuropatica)

- Static and locomotor ataxia (esp. at night) - Romberg's test is positive (the patient is unstable only with closed eyes)
- A damage or even a loss of deep sensation
- Diminished superficial sensation distally
- Hypo- to areflexia
- Accompanied by distal paresis and atrophy of the distal muscles

Clinical examples for ataxia, caused by a lesion of peripheral nerves: acute inflammatory demyelinating polyneuropathy, metabolic polyneuropathy (diabetic, porphyria), toxic polyneuropathy (alcoholic), etc.

Ataxia, caused by a lesion of the frontal lobe

- Ataxia – contralaterally to the side of the lesion or bilaterally
- No symptoms of lesions of the cerebellum or the sensory system
- Other frontal symptoms – motor aphasia, apraxia, positive grasp reflex
- Dementia

Clinical examples for ataxia, caused by a lesion of the frontal lobe: tumors, Alzheimer disease, Binswanger disease, normal pressure hydrocephalus

Coordination tests

A coordinated combination of a series of motor actions is needed to produce a smooth and accurate movement. This requires integration of sensory feedback with motor output. This integration occurs mainly in the cerebellum. In the presence of weakness, tests for coordination must be interpreted with caution and are unlikely to be informative if there is significant weakness. Loss of joint position sense can produce some incoordination (sensory ataxia). This is made substantially worse when the eyes are closed. Joint position sense should be tested before coordination.

WHAT TO DO

- **Test the gait**

In all tests, compare right with left. Expect the right hand to be slightly better (in a right-handed person).

- **Arms**

Ask the patient to hold his arms outstretched and ask him to close his eyes. Tell the patient to keep his arms in this position. Then push

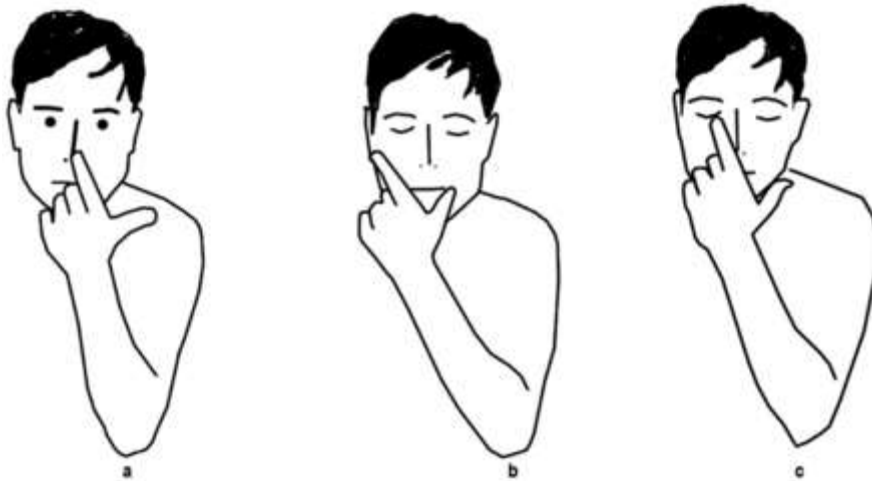
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his arm up or down suddenly.

Finger–nose test

Hold your finger out about an arm's length in front of the patient. Ask the patient to touch your finger with his index finger and then touch his nose. When he has done this correctly, ask him to repeat the movement faster. Watch for accuracy and smoothness of movement.



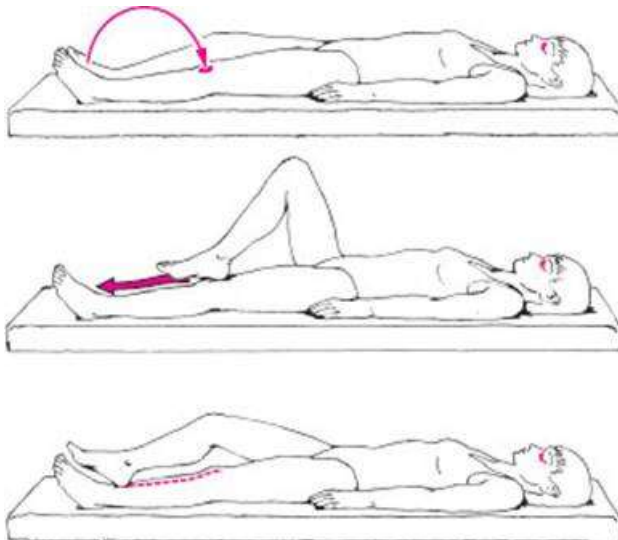
Ask the patient to twist his hand as if opening a door or unscrewing a light bulb.

Ask the patient to tap the back of his right hand alternately with the palm, and then the back of his left hand. Repeat with the right hand.

- Legs

Heel–shin test

The patient is lying down. Ask him to lift his leg and place the point of his heel on his knee, and then run it down the sharp part of his shin. Watch for accuracy and smoothness of movement.



- Trunk

Romberg test



- ### Heel–shin test

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- Disorganisation of movement with the heel falling off the anterior part of the shin, and the knee falling from side to side.

Trunk

- The patient is unable to sit from lying without falling to one side: *truncal ataxia*. This is associated with gait ataxia.