ATAXIA

Ataxia

= disturbance of movement coordination

- Voluntary movements are in principle possible.
- Muscle power is not reduced (contrary to palsy).
- Deterioration of cooperation between individual muscle groups, timing and contraction intensity adjustment.

  → Disorganized complex movements
- Defective movements with reduced functionality.
- In severe cases loss of mobility, complete disability.

Types of ataxias

- cerebellar
- sensory (spinal)
- vestibular

Cerebellar ataxia

- One of the manifestations of the **extinction** cerebellar sy.
- Consists of following partial disorders:

  1) asynergy

  2) dysmetria

  3) adiadochokinesia

Basal disorders lead to particular symptoms:

- Posture disturbances (posture ataxia)
- Gait disorders (gait ataxia)
- Ataxia of targeted limb movements
- Speech disorders
- Oculomotor disorders
- Macrographia

Cerebellar ataxia does not significantly worsen after closing the eyes (disturbance of information processing in the damaged cerebellum, the problem is due to impaired central control of movement coordination, not due to lack of sensory information)!

Sensory, spinal ataxia

- loss of proprioceptive sensitivity

Mechanisms:

- dorsal column syndrome - preserved thermic sensation and pain perception = tabic dissociation of sensation
- peripheral sensitive nerve damage

Causes

- tabes dorsalis – component of the 3rd stage of syphilis
- severe vit. B12 deficiency
- other demyelinating processes
- HIV infection
- mechanical damage of the posterior parts of the spinal cord (trauma, compression by a tumor, changes of the vertebral column...)
- neuropathy (e.g diabetic)

Manifestation of ssensory ataxia

- Dysmetria: inappropriate mobilization of the muscles, does not maintain movement direction, misses the target
- Rapid, irregular deviations, splashes, titubations
- Prussian soldier gait: excessive and fast elevation of the legs and strong stamping on the ground
- Titubations and falls to any directions when standing

Sensory ataxia worsens after closing the eyes!
Lacking proprioceptive information can be in part substituted by visual control of body and limb position.

Vestibular ataxia

Arises from to lesion of the peripheral part of the vestibular regulatory circuitry, i.e. the labyrinth and n. VIII.

Some authors do not consider it to be really ataxia.

Causes

- Complication of the otitis media
- Skull fracture
- Meniere‘s syndrome
- Tumors in the pontocerebellar angle, n. VIII neurinoma
- Herpes zoster oticus
- Ischemia of the vestibular apparatus

Manifestations of vestibular ataxia

- Dizziness (rotation), nausea, vomiting, perspiration
- Spontaneous nystagmus directed toward the healthy side (oscillate eye ball movements, slow component toward the relatively weaker vestibule, the fast component = compensatory return of the eye ball, the direction of the nystagmus = the fast component)
- Titubations and falls to the side of the affection (depend on position of the head)
- Tonic deviations of the extremities to the affected side
- Often also perceptive hearing disorder with tinnitus

Vestibular ataxia worsens after closing the eyes!
Lack of the vestibular information can be in part compensated by vision.