Neocerebellar Syndrome

It is characterized by a disorder in the coordination of complex and fine movements caused by a disruption of the function of the neocerebellum, which contains programs for free motor skills.

Symptoms

Damage to the function of the neocerebellum is manifested by hypermetria. Movements are late started and late stopped. Coordination between agonist and antagonist muscle groups is disturbed - dysmetria. The so-called small neocerebellar asynergy affects precise coordinated movements and adiadochokinesia makes it impossible to correctly link rapidly alternating movements. During targeted movements, we observe an intentional tremor (3–5 Hz), which increases as we approach the target. It appears mainly when the nucleus dentatus is destroyed. Others include titubation – swaying of the trunk while standing or sitting (2–3Hz). A person with this syndrome is unable to reduce the muscle force they are working against after a sudden reduction in resistance. This is called the rebound phenomenon. The movements of these patients are jerky and disintegrated into individual components – a movement decomposition that affects both speech and writing. Patients suffer from an articulation disorder with unnatural separation of syllables and slow speech - saccadic speech and macrography, which manifests itself by changing the font or even overshooting when connecting two points on a drawn template. When estimating weight, objects on the side of the lesion appear lighter. Nystagmus in the direction of damage or visual impairment caused by dysmetria of the oculomotor muscles is common. Similar symptoms are also found in paleocerebellar syndrome.

Examination

- Adiadochokinesia – the patient, without visual control, raises his forearms and alternately pronates and supines. The hand on the side of the cerebellar lesion falls out of rhythm. An alternative is flexion and extension of the limbs.
- Small neocerebellar asynergy - the patient is unable to sit up without the help of hands when instructed to change position from lying to sitting and raises the lower limb on the side of the cerebellar lesion.
- Intentional tremor - we ask the patient to touch the nose with the index finger, while observing a visible tremor.
- Stewart-Holmes test – tests the so-called rebound phenomenon. The patient flexes the upper limbs against passive resistance. After the sudden disappearance of resistance, the limb on the side of the cerebellar lesion shoots up.

Etiopathogenesis

The most common reason for functional impairment is ethanol intoxication causing balance disorders, titubation and gait ataxia. The disorder can occur during a heart attack or hemorrhage with the onset of rapid non-specific symptoms such as headache, dizziness, balance disorders or ataxia on the side affected by the thrombus or embolism. Tumors of the posterior cranial fossa can destroy the tissue of the cerebellum or suppress it through growth and disrupt the circulation of cerebrospinal fluid, which can lead to hydrocephalus. Multiple sclerosis or diffuse cerebellar demyelination after infection with the Varicella Zoster virus can also cause ataxia of the limbs and impaired balance. In the pharmacological anamnesis, it is necessary to note the treatment with hydantoin - anticonvulsants, e.g. in the treatment of epilepsy, as they can cause cerebellar disorders.

Sources

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