Disorders of symbolic functions

Breakdown of symbolic functions

Acalkulia[1]

Number processing disorder in dominant parietal lobe lesions.

Agraphia[1]

It accompanies expressive aphasia. Movement patterns for writing are missing, the own movement of the limbs is not violated. It occurs in the case of a lesion of the dominant parietal lobe.

Agnosia[1]

It is a disorder of cognition through the senses.

Tactile agnosia (stereoagnosia)

Impaired recognition of objects by touch. It happens bilaterally. In the case of unilateral involvement (hemiastereognosia), the posterior part of the corpus callosum is affected (tactile gnosia is stored in the parietal lobes, if a part of the corpus callosum is cut, the parietal lobes lose part of the interhemispheric connections). The disease can imitate stereohypesthesia and stereoanesthesia, which arise from lesions of somatosensory structures.

Auditory agnosia

It is very rare. Auditory gnosis is located bilaterally in Heschl's convolutions with many commissural connections. For auditory agnosia, a bilateral lesion of area 41, 42, 22 and the corresponding white matter is therefore necessary. The patient cannot distinguish typical sounds such as the sound of a passing car or the sound of running water.

Visual agnosia

The patient cannot recognize what is seen, but can identify things by hearing or touch. It is necessary to rule out bilateral elementary lesions of the visual cortex. This is done by means of the blink reflex or an optomotor response involving the area parastriata. The cause of visual agnosia is often heart attacks in the basin of the posterior cerebral artery. A form of visual agnosia is *prosoagnosia* , when the patient does not recognize faces, but identifies a person by voice.

Alexia

Inability to read and understand what is written. Basically a special form of visual agnosia. The cause is thrombosis of the left posterior cerebral artery with malacia of the dominant occipital lobe (including the white matter connecting the hemispheres)

The combination of acalculia, agraphia and agnosia is the so-called *Gerstmann syndrome* . The patient cannot tell the fingers apart and confuses the right and left sides. The patient articulates and understands speech normally. It arises from a lesion of area 39 (gyrus angularis) due to vascular causes or during expansive processes.

Amusia [1]

It is characterized by the loss of the ability to communicate using rhythm, tones and melody. Both hemispheres participate in music. In the non-dominant hemisphere, the pitch (frequency) of the tone is encoded. Therefore, when the non-dominant hemisphere is affected, the patient reproduces music monotonously. The more professional and experienced the musician is, the more engaged the dominant hemisphere is.

Motor amusia

- Oral - inability to make sounds with the mouth and vocal cords.
- Instrumental - inability to play a musical instrument including tapping out a beat.
- Inability to use notation (editor's note: the author probably means writing the melody into notes from an ideational idea).

Sensory amusia

- Inability to distinguish pitch.
- Inability to distinguish tone color, sound of instruments and melody.
- Inability to read sheet music.

Apraxia[2]

With apraxia, the patient is unable to perform learned, coordinated movements with intact mobility. If we want to perform a targeted movement, we must first have an idea to perform the movement, then a plan to perform it, and then execute the movement. A person with apraxia has a problem in one or more of these steps. More severe forms of apraxia occur with a lesion of the dominant hemisphere, lighter forms with a lesion of the non-dominant hemisphere.

Motor apraxia [2]

In this apraxia, the plan of how to perform the movement is preserved, but the execution is impaired. This is, for example, bad fastening of buttons or difficulty inserting the key into the lock.

Ideomotor apraxia [1]

The patient lacks a movement plan. For example, he uses the key in the opposite direction, cannot comb his hair, etc.

Construction apraxia [1]

The patient cannot assemble cubes, pictures, draw geometric figures, etc. When the lesion is in cooperation with both parietal lobes.

Dressing apraxia [1]

With a lesion in the cooperation of both parietal lobes.

Ideational apraxia [2]

The patient does not have a plan or an initial idea for performing the movement. He doesn't understand what we want from him. It is most common in atrophies (Alzheimer's muscle) and deep parieto-occipital white matter lesions. [1]

Disorders from a lesion of the speech non-dominant hemisphere [1]

Hemiasomatognosia

Left-sided hemiplegia within the neglect syndrome . The patient considers the left limbs to be foreign, belonging to someone else. There is often a conjugate deviation of the eyes and head homolateral to the plegia.

Anosognosia

The patient is not aware of his illness, e.g. hemiplegia. Often in extensive encephalomalacia in the non-dominant hemisphere.

Corporis callosi impingement syndrome [1]

The corpus callosum is sometimes transected therapeutically in intractable epilepsies. This artificially creates a disconnection syndrome, or split brain syndrome. Each hemisphere works independently, but only the dominant one (mostly the left one) has the ability to communicate verbally or in writing. In reality, it looks like this. The patient is unable to read from the left half of the visual field. After reading tasks from the right visual field, the patient is unable to perform this task with the left limbs.

In children under the age of ten, the consequences are less, for example, impaired orientation in space, dyspraxia, the feeling that the left hand does not belong to the child (alien hand).

Anterior callosotomy induces gross memory disturbances.

Links

related articles

- Symbolic functions
- Speech disorders

Reference

1. NEVŠÍMALOVÁ, Soňa – RŮŽIČKA, Evžen – TICHÝ, Jiří. *Neurology.* 1. edition. Galén, 0000. 0 pp. ISBN 80-7262-160-2.
2. AMBLER, Zdeněk. *basic neurology.* 6. edition. Galén, 2006. 0 pp. ISBN 80-7262-433-4.