Lobar syndromes

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Lobar syndromes

Lesions of specific areas of the cerebral cortex produce focal neurologic signs as well as a focal and sometimes a generalized mental syndrome.
Intracranial parenchymal hemorrhage
Occlusion of left internal carotid artery
Frontal lobe I.

a./ precentral region:

- contralateral focal paresis, paralysis (area Br. 4), focal motor epileptic seizure
- disturbances of gaze (area Br. 8) Gaze is directed initially to the affected side. Signs of irritation (focal motor epilepsy often referred to as adverisive attacks, although the eyes turn away from the discharging focus.
Frontal lobe II.

b./ parasagittal surface

- incontinence
  (bladder and bowel function are disturbed)
- contralateral leg paresis, paralysis
Frontal lobe III.

c./ convexity of the frontal lobe:

- reappearance of primitive reflexes (Br. 6)
  - grasping and sucking reflexes
- involuntary resistance to passive limb movement: “gegenhalten reflex”, paratonia
- postural retention
  (once a limb has been placed in a particular position, that position is maintained for an unusually long time)
- echopraxia
  (movements observed may be imitated)
- echolalia (words and sentences heard may be repeated)
Frontal lobe IV.

- **motor aphasia** (Broca’s speech center, Br. 44 *aphasia* is a general term relating to a loss of language ability).
- **ataxia**
  (frontocerebellar tract are interrupted, the area 6 alpha, beta lesion)
- **general loss of interest and drive, spontaneity and activity: apathy**
  (convexity of the frontal lobe is affected)
- **inappropriate or uninhibited social behavior: lesion of the orbital area**
  (leads to a progressive mental blunting and a picture of impulsive behavior, humorlessness and moral decay, and ultimately to affective dementia)
Parietal lobe I.

Postcentral gyrus with sensory cortical representation, supramarginal gyrus, angular gyrus which is important for gnostic function.
Symptoms of parietal lobe I.

- Contralateral sensory disturbances (paraesthesia, hypaesthesia)
- Abnormal tactile discrimination
- Contralateral homonymous lower quadrant visual field deficit, and reduced response of *optokinetic nystagmus* to stimuli
- Epileptic seizures ("jacksonian" sensory seizures)
- Amnestic aphasia
Symptoms of parietal lobe II.

- Disturbance of spatial orientation and right-left discrimination may be present (dominant hemisphere)

- Constructional apraxia
  (difficulty performing purposeful or complex movement without evidence of paralysis or sensory loss; the dominant hemisphere, the supramarginal gyrus is affected).

*Apraxia* is a general term for disorders of practice.
Symptoms of parietal lobe III.

- **Agnosia**
  (dominant hemisphere; *agnosia* is a general term for a loss of ability to recognize objects, people, sounds, shapes, or smells; that is, the inability to attach appropriate meaning to objective sense-data. It usually is used when the primary sense organ involved is not impaired).

- **Dyslexia, alexia**

- **Gerstmann syndrome** (dominant hemisphere, left angular gyrus is affected):
  - finger agnosia, right-left confusion
  - agraphia, acalculia

- **Anosognosia**
  (right supramarginal gyrus is involved)
Symptoms of temporal lobe

- Wernicke’s aphasia
  (Br. 41; if the lesion affects the dominant hemisphere)
- Temporal epilepsy (“déjà vu”, “jamais vu”, dreamy state, complex partial seizures)
- Contralateral homonymous visual field defects
  (upper quadrantanopia); reduced response of \textit{optokinetic nystagmus} to stimuli
- Memory disturbances (lesions of the mediobasal part of the temporal lobe: hippocampus)
- Depression and irritability
Symptoms of occipital lobe

- Contralateral visual field defect, homonymous hemianopia
- Cortical blindness, sensory blindness
- Reduced response or absent of optokinetic nystagmus to stimuli
- Disturbances of conductive eyeball movements
- Alexia
- Disturbance of color recognition “color or visual agnosia”
- Disturbance of visual-spatial orientation
- Signs of irritation: abnormal visual sensations
Occipital lobe infarct
Cortical blindness in pregnant patient
Investigating aphasia

- Spontaneous speech
- Naming
- Repetition
- Reading
- Writing
- Spelling
Summary of different types of aphasias I.

• **Broca's aphasia.**
  Also known as "expressive aphasia". Characterized by nonfluent, effortful speech. Comprehension usually good. No paraphasic errors. Typically caused by frontal lobe lesion. These patients may only have (for example), a single word left in their vocabulary.

  **Aphemia** means mute but writes fluently.
Summary of different types of aphasias II.

• Wernicke's aphasia. Also known as receptive aphasia. Fluent effortless speeches with frequent use of wrong or nonexistent words and improper word usage. Poor comprehension and repetition. Typically from dominant temporal lobe lesion.
Summary of different types of aphasias III.

- Conductive aphasia.
  Unable to repeat. Insular region is affected. About 10% of all aphasia.

- Anomic (amnestic) aphasia.
  Unable to name. Parietal lobe lesion.

- Transcortical motor.
  Normal repetition. Decreased fluency and speech output. Paramedian frontal, anterior.
Summary of different types of aphasias IV.

- **Transcortical sensory.**
  Normal repetition. Poor comprehension of spoken language and paraphasic errors. Basal ganglia, posterior inferior temporal.

- **Global aphasia.**
  Combination of Broca's and Wernicke's. Mild forms are called "mixed". Large prerolandic or large deep lesions.
Language abnormalities

- **Anomia**: impaired naming objects

- **Expressive aphasia**: speech disturbance with preserved comprehension

- **Receptive aphasia**: comprehensive difficulty with preserved speech

- **Conduction aphasia**: inability to repeat

- **Aphemia**: mute but writes fluently.

- **Alexia without agraphia**: inability to read with preserved repetition

- **Transcortical sensory aphasia**: comprehension difficulty with preserved repetition
Cerebellar syndromes, brain stem lesion
Cerebellar function

- Cerebellum coordinates the action of individual muscle groups so that
- agonists, accessory muscles, and antagonists act in a fluent and precise way, and
- carry out purposeful, economic, and appropriate movements.
- Stabilizing control system.
- Controls the muscle tone.
Anatomic and physiologic preconditions I.

• The cerebellum must receive constant “feedback” information about movements going on, and about movements intended.
• It must be able to effect corrections and modifications during each individual movement.
• The cerebellum receives forewarning of each motor impulse,
• evaluates each impulse in the light of a sensory “feedback” mechanism, and
• modifies or corrects this impulse.
Information about the position of the extremities and the activity of muscle groups is transmitted by the afferent fibers:

- spinocerebellar tract (dorsal)
- (x) spinocerebellar tract (ventral)
- (x) olivo-cerebellar tract
- vestibulo-cerebellar tract
- (x) ponto-cerebellar tract (transmits stimuli from the cortex)
- bulbo-cerebellar tract (from the Goll-Burdach nucleuses)
Anatomic and physiologic preconditions III.

**Efferent fibers** (origin from the Purkinje cells)

- Pass along the brachium conjunctivum (superior cerebellar peduncle) from the cerebellar nuclei to the globus pallidus and ventrolateral nuclei of the thalamus on the opposite side → to the cerebral cortex

- Other fibers pass to the red nucleus and then turn to the thalamus and pass via the rubrospinal tract caudally to the brainstem and spinal cord.
Symptomatology of cerebellar disorders I.

- **Intention tremor**: increasing deviation from the ideal pathway as the patient tries to carry out a particular movement. Dentate nucleus or its efferent fibers are involved. (Observed in finger-to-nose, heel-to-shin, finger-to-finger tests).

- **Dysmetria**: inability to gauge distance, power, or speed of a movement (the patient may overshoot or undershoot the movement)
Symptomatology of cerebellar disorders II.

- **Ataxia**: muscle groups no longer contract harmoniously to execute a movement.
- **Truncal, walking ataxia**
- **Dyssynergia, asynergia**: lack of coordination between groups of muscles, causing movements to appear awkward and disjointed, instead of fluid.
- **Dysdidochokinesis**: impairment of rapid alternating movements (rapid pronation and supination of the forearm)
- **Pathologic rebound phenomenon**: the antagonists fail to counter overshoot movements promptly.
Symptomatology of cerebellar disorders III.

- **Hypotonia**: decreased skeletal muscle tone during passive movements. The tendon reflexes are decreased.
- **Diminished deep tendon reflexes**: when the knee-jerk is elicited, a pendulum-like movement of the leg may result (pendular reflex)
- **Speech disturbance**: staccato explosive speech (dentate nucleus lesion)
- **Deviation in Bárány’s pointing test**
- **Unsteady posture** (in Romberg test)
- **Nystagmus**
Disease with predominantly cerebellar symptomatology

- Acute cerebellar hematoma (in any age)
- Ischemic lesion (in adults)
- Cerebellar tumor (especially in children)
- MS (in young adults)
- Hereditary cerebellar ataxia (in any age)
- Olivopontocerebellar atrophy (in 3rd and 5th decades of life)
- Poisoning (DDT, diphenylhydantoin, organic mercury salts, 5-fluorouracil, alcohol)
- Familial episodic ataxia (autosomal dominant)
- Symptomatic progressive cerebellar atrophy (in any age, paraneoplastic disease)
- Psychogenic ataxia (in teenagers and young adults)
Diagnosis

- CT,
- MRI,
- DSA, CTA
- PET