Ministry of Education and Science of Ukraine
V. N. Karazin Kharkiv National University

LOCALIZATION OF FUNCTION IN THE BRAIN CORTEX. SYNDROMES OF THE DEFECT

Methodical recommendations
for applicants of higher education of the 4th year of study
the discipline "Neurology"

Electronic resourse

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L 73

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L 73 **Localization of function in the brain cortex.** Syndromes of the defect : methodical recommendations for applicants of higher education of the 4th year of study the discipline "Neurology" [Electronic resourse] / compiler T. S. Mischenko, V. M. Mischenko, I. K. Voloshyn-Haponov, I. B. Savytska, I. V. Kabachna, I. A. Nazarchuk. – Kharkiv : V. N. Karazin KhNU, 2025. – (PDF 56 p.)

The methodical recommendations describe the main anatomical and physiological features of neurological disorders in the lesion of the cerebral cortex. Particular attention is paid to the ontogenesis of cerebrum, classification of symptoms of cortical and differential diagnosis of symptoms in different parts of the brain. The publication contains a large number of tasks, tests with standards of answers, a list of basic and additional literature for self-preparation for a practical lesson in psychiatry. Intended for applicants for higher education in the 4th year of study the discipline "Neurology".

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CONTENTS

INTRODUCTION

Human cortical functions have been elucidated by studies of deficits in traumatic and vascular brain damage, outcomes after elective neurosurgical procedures, studies in primates and in more recent years by imaging techniques. Cortical functions are well-defined for primary cortical areas like motor, sensory and visual functions. More complex cortical functions like language and to some degree memory are also well clarified. The associative cortical areas are more difficult to study as functions are integrated to and modulate primary cortical functions. Nevertheless, the structural basis for symptoms like neglect, apraxia and agnosia has been well established.

The doctor of any profile should be guided in matters of anatomy, physiology and syndromes of disorders of the cortical functions. It is very important for students to know the clinical features of normal conditions of the brain functioning while to distinguish it from pathology of the different parts of the cortex. It can occur in the clinical work and timely hospitalization in neurological clinic patients with the provision of the necessary specialized assistance will contribute to the recovery of the patient. Research methods, the ability to assign the necessary method in this clinical case need doctors different profiles.

Particular goal: to teach the students to research methodology cortical functions and the ability to evaluate their destruction syndromes in diagnosis. To be able to investigate patients with various neurological disorders of the nervous system with positive meningeal syndrome, cortex, to be able to analyze, explain symptoms found in patients and predict diseases in which the symptoms can be. Offer the necessary methods of diagnosis of this disease, analyze diseases in which possible changes certain functions.

Basic knowledge, skills, habits necessary for studying the subject (interdisciplinary integration). The need for knowledge of anatomy and physiology of the cortex, physiology of the liquor formation, the components of the liquor in normal, its modifications in meningitis, tumors, hemorrhagic stroke, tuberculosis for

a correct diagnosis of diseases that are manifested in various disorders and cortical lesions.

Tasks for independent work during preparation for the classes.

List of basic terms, parameters, characteristics which the student is to assimilate while preparing for the class:

- The structure of the cerebral hemispheres of the brain.
- Cyto- and myeloarchitectonics of the cortex. Localization of functions in the brain cortex. Dynamic localization of functions. Motor and sensory representations in the cortex. The concept about the functional asymmetry of the hemispheres.
- The gnostic functions. Types of violations of the gnostic functions: visual, olfactory, gustatory, auditory agnosia, astereognosia, autotopagnosia, anozognosia.
- Praxis. Types of apraxia: constructive, ideatory, motor.
- The speech. Disorders of speech: motor, sensory, amnestic aphasia.
- Syndromes of defeat of some shares of large hemispheres: the frontal, temporal, parietal, occipital lobes, the limbic cortex.
- Syndromes of irritation of the cortex of the large hemispheres.
- Syndromes of the defeat of the left and right hemispheres.
- The concept about the between-hemispheres asymmetry.
- The syndrome of chronic vegetative state.
- The syndrome of the «locked» patient.
- The syndrome of death of the brain.

Theoretical questions for the class.

1) Ontogenesis of cerebrum.
2) Anatomy and morphology of the cerebral hemispheres
    a. Structure in general
    b. Sulci and gyrus of the hemispheres of the final brain
        1. Dorso-lateral surface
        2. Medial surface

3. Lower surface

c. White matter of cerebrum

I. Associative fibers

1. Upper longitudinal bundle (fasciculus)
2. Lower longitudinal bundle (fasciculus)
3. Unciform fasciculus (uncinate fasciculus)
4. Other important associative fibers

II. Commissural fibers

1. Corpus callosum
2. Anterior commissure (commissura anterior, precommissure)

III. Projection fibers' system

1. Psalterium (Lyra)
2. Fornix
3. Transparent membrane
4. Internal capsule (capsula interna)
5. Corona radiata

IV. Ventricles of the brain

Lateral ventricles

1. Front horn
2. Posterior horn
3. Inferior horn
4. Ammon's Horn (Cornu Ammonis, hippocampus proper)

d. Cytoarchitectonic and myeloarchithectonic of the cortex of hemispheres (layered structure)

I. Layers of the cerebral cortex

II. Isocortex and alokortex

III. Type of cortex structure

1. Homotypic cortex

a. Molecular layer

b. External granular layer

c. External pyramidal layer

d. Internal granular layer

e. Internal pyramidal layer

f. Multiform layer (layer of polymorphic cells)

2. Heterotypic cortex

IV. Myeloarchithectonic

V. Areas and fields of the cerebral cortex

1. The Cytoarchitectonic Map of Korbinian Brodmann

2. Plasticity of cortical architectonic

3) Physiology of the cerebral cortex

a. General information on the physiology of the cortex of the large hemispheres

b. Localization of functions in the cortex

I. Primary cortical fields

1. Primary somatosensory cortex

a. Primary visual cortex

b. Primary auditory cortex

c. Primary bark of taste

d. Primary vestibular cortex

2. Primary motor cortex

II. Associative zones of the cortex

1. Secondary cortical fields

2. Tertiary cortical fields

4) Syndromes of damage to certain areas of the cerebral cortex

a. Frontal lobe

I. Premotor cortex

II. Violation of speech. Aphasia and its types

1. Non-fluent aphasia (Broca's aphasia)

2. Wernicke’s aphasia

3. Conduction aphasia

4. Total aphasia

5. Amnesic aphasia

6. Transcortical motor aphasia

III. Disengagement Syndromes

1. Disengagement Syndromesin smell analysis system

2. Disengagement Syndromes in the visual system

IV. Apraxia

1. Motor apraxia: ideomotor and ideatory

2. Constructive apraxia

3. Amnestic apraxia

4. Apraxia of gait

5. Sensory apraxia

6. Apraxia of speech

V. Agnosia

1. Visual agnosia

2. Somatosensory agnosia

a. Astereognosis

b. Asomatagnosia

c. Gerstmann syndrome

d. Balint syndrome

VI. Ignoring. The phenomenon of oppression (феномен подавления)

VII. Behavioral disorders

1. Lesions of the prefrontal convexital cortex

2. Damage to the orbitofrontal cortex

5) Methods for studying the functional state of the cortex of hemispheres.

a. Magnetoencephalography

b. Positronemission tomography (PET)

c. Functional magnetic resonance imaging (fMRI)

d. Electroencephalogram (EEG)

6) Methods of visualization of the cerebral cortex

a. Magnetic resonance imaging (MRI)

b. Computed tomography (CT)

7) Localization functions in the cerebral cortex. Syndromes lesions. The structure of the cerebral hemispheres.

8) Cyto- and myeloarhitectonic cortex. Dynamic localization functions. Motor and sensory representation in the cortex. The concept of functional asymmetry hemispheres.

9) Gnostic functions. Types of abuse gnostic functions: visual, olfactory, gustatory, auditory agnosia, astereognosis, autotopagnosia, anosognosia.

10) Praxis. Types apraxia: constructive, ideator, motor.

11) Language. Language disorders: motor, sensory, amnestic aphasia.

12) Syndromes of individual particles cortex: frontal, temporal, parietal, occipital fractions, limbic cortex.

13) Syndromes irritation cerebral cortex. Syndromes of right and left hemispheres.

14) The concept of hemispheric asymmetry.

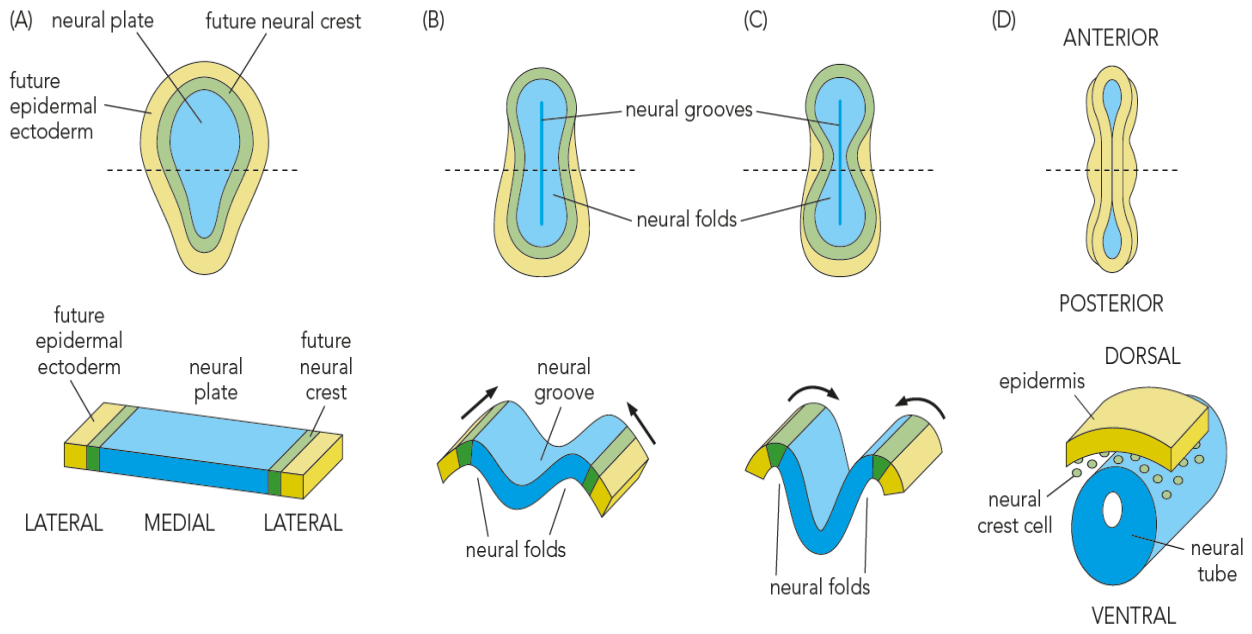
15) The syndrome of chronic vegetative state. Syndrome “locked” patient.

16) Brain death syndrome.

1. Ontogenesis of cerebrum

The nervous system goes from the beginning of its formation in the third week of embryonic development from the outer germ leaf (ectoderma) to the stages of coagulation into the neural tube, followed by the formation of first thickening, and then three (primary: prosencephaphalon, mesencephalon, rhombencelonlon) and then five brain vesicles (secondary: telencephalon, diencephalon, mesencephalon, metencephalon, myelencaphalon).

In the early period of the embironic development of the ectoderma cell on the dorsal half of the embryo, they form a longitudinal plate - the muddular (nerve) plate. Gradually, the medular plate begins to bend ventralally towards the mesodermal leaf. Gradually, the edges of the resulting gutter are sealed due to the reproduction of ectodermal cells, forming a neural tube, which after some time separates from the rest of the ectoderma and deepens into the body of the embryo. The tube cavity becomes the central canal of the spinal cord and the ventricular system of the brain.



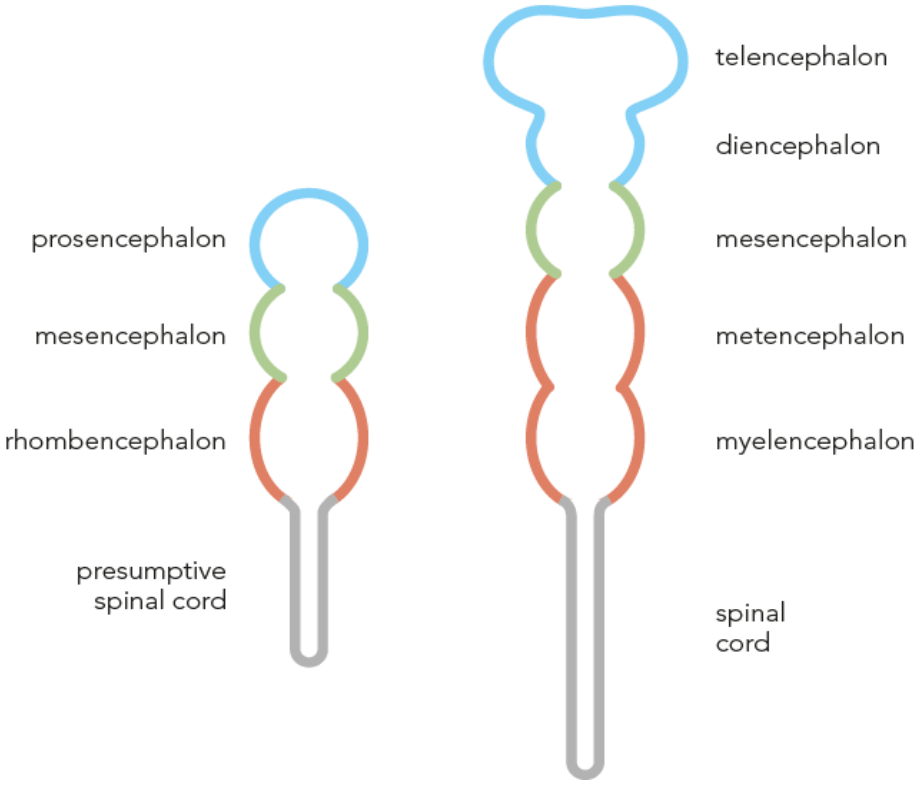
The neural tube consists of two parts – ventral (anterior) and dorsal (posterior) . that form a striped body (neostriatum), an amygdala (amigdala). The above anatomical structures belong to the subcortical ones. The cortex is formed from the

dorsal part of the neural tube. During phylogenesis, older areas of the cortex (paleocortex) are lateral newer areasok (archicortex). At the end of the formation of the nervous system paleocortex shifts closer to the base of the brain, where phylogenetically ancient areas of the sensory system of smell are formed – the olfactory bulb (bulbus olfactorius), the olfactory tract (tractus olfactorius), olfactory triangle (trigonum olfactorium), front punched substance (anterior piercing substance, substantia perforata anterior) and lateral olfactory gyrus (gyrus olfactorius lateralis). Simultaneously with the formation of the sensory sense of smell, there is a gradual shift of the archicortex to the area of the lower hornsand lateral ventricles (ventriculi laterales) as a result of the rapid growth of telencephal blisters. Archicortex forms the Ammon horn, or hippocampus (hippocampus), as well as in its mediadorsal direction, the gray matter (indusium griseum, also called gyrus supracallosal, gyrus epicallosus).

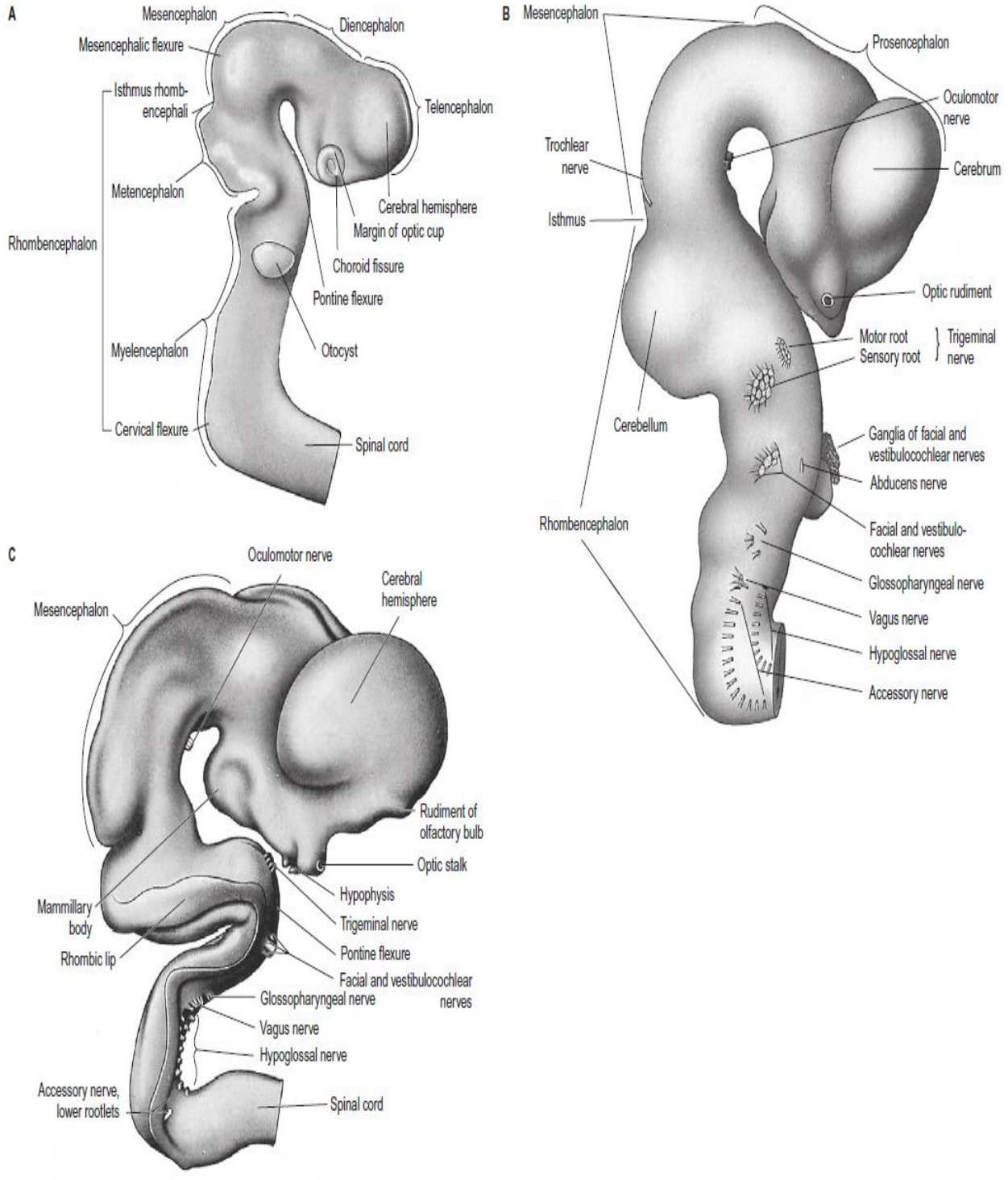
From the intermediate brain (diencephalon) occur: globus pallidus, thalamus opticus, metathalamus, epithalamus, pars mamillaris hypothalami; posterior part of the third ventricle.

Middle brain(mesencephalon): Varolium bridge (pons Varolii), cerebellum,front of the fourth ventricle.

Myelencephalon: medulla oblongata.



Isthmus rhombencephali: cerebellar legs to a four-humped body (corpora quadrigemina) and an anterior brain sail (velum medullare anterior).



Cerebrum is a derivative of the telencephalon, originating from paired teleencephalic brain vesicle, which originate in the anterior parts of the neural tube (prosencephalon). Cavities under the thickness of the nervous tissue, which arise as a

result of wrapping the brain stem, are filled with fluid from the lumen of the neural tube. Thus, the formation of the lateral ventricles occurs, which, together with the subsequent growth and development of the hemispheres of the brain, acquire a semicircular shape. The pathways of nerve impulses (tracts) are formed in the same way in a semicircle, bypassing the walls of the ventricles. According to the same principle, brain vaults (fornix),corpuscallosumand comisural fibers are formed, which will be described in more detail in the following sections.

The final brain at the beginning of its development has a rounded shape, later divided by the sagital furrow in half (the formation of the cerebral hemispheres). Connects the hemispheres of the brain a large cerebral adhesion, or corpus callosum.

Myelination also begins in the fourth intrauterinemonth. All reflex reactions that appear in the fetus in the third intrauterine month are not related to the cortical region of the nervous system and are locked at the level of the spinal cord and oblong brain. On the 6th – 7th months, reflexes appear, in which stem and subcortical formations are involved.

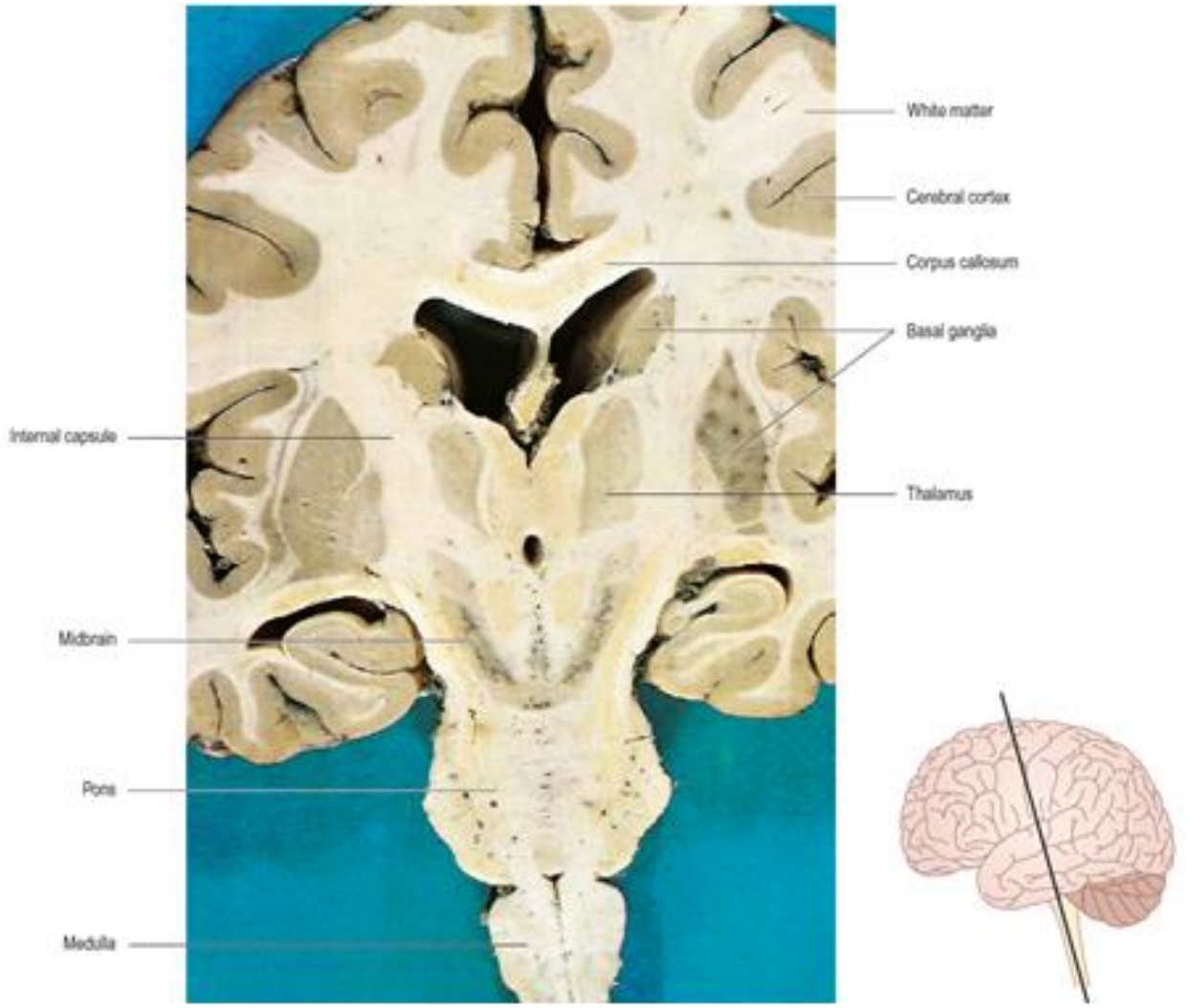
The maximum rate of cortex growth in the postnatal period occurs in the first two years of life (maturation of analyzers, cellular differentiation, myelination,increase in mass and surface area with deepening grooves and the formation of new small ones). Improvement of the nervous system continues until 22 – 25 years.

The cortex acquires a dominant effect already in a five-thousandth infant due to the active participation of all sensory analyzers, and higher nervousfunctions actively progress during the development of speech in the 2nd year of life.

2. Anatomy and morphology of the cerebral hemispheres (pallium)

In this section we will consider the division of the cerebral cortex into particles,detail the features of the structure of gray and white matter of the brain for a better understanding of the morphological and functional difference of these units,

pay special attention to the cellular structure of the gray matter of the brain and its division into layers, taking into account the functional characteristics of each of them according to the mapsand Brodmann and the nuances regarding division into the fields of the latter. Anatomical features of the position of the ventricles of the brain will be considered, taking into account their significant impact on the formation of key tracts of nerve impulses.

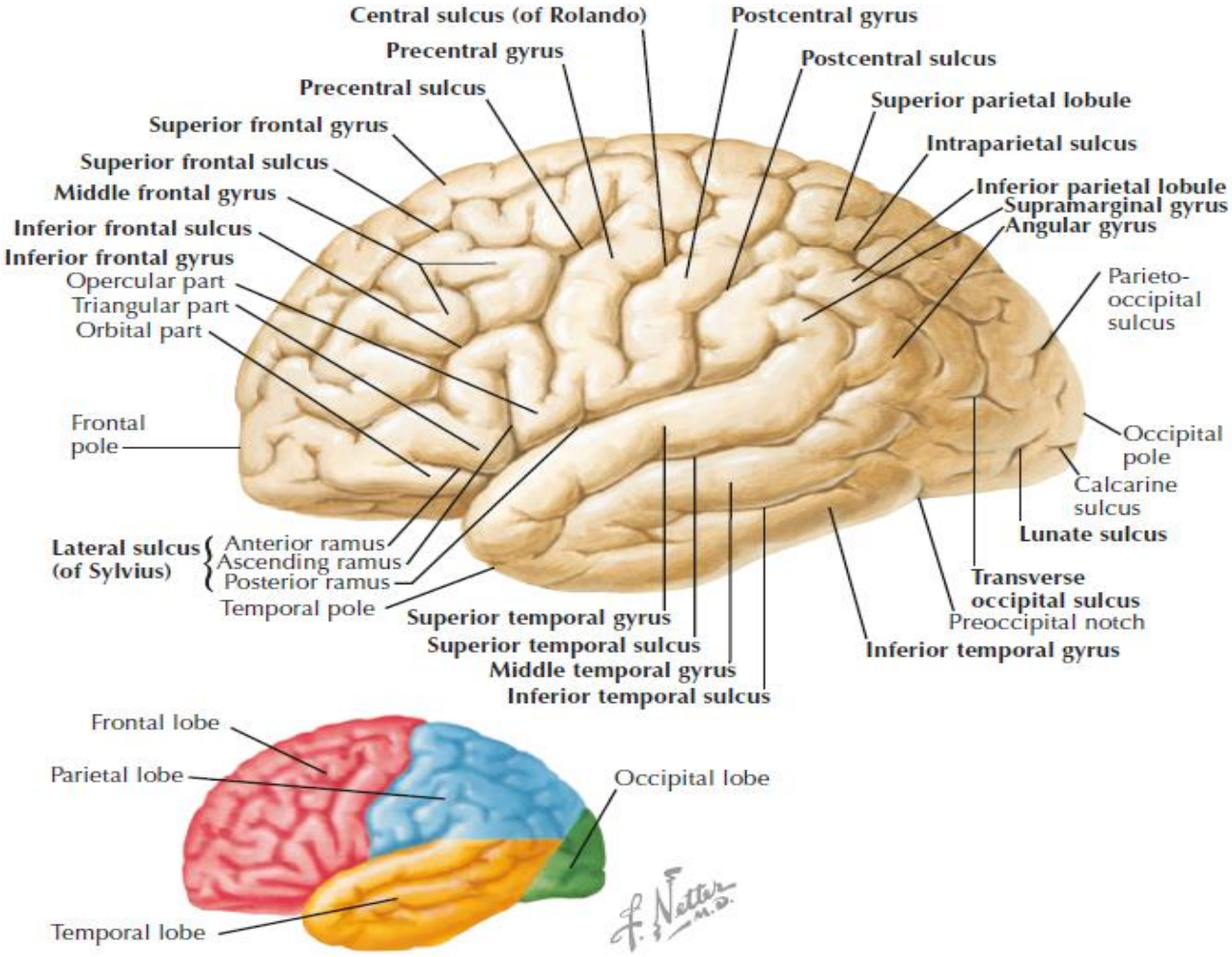


Organization of cerebrum

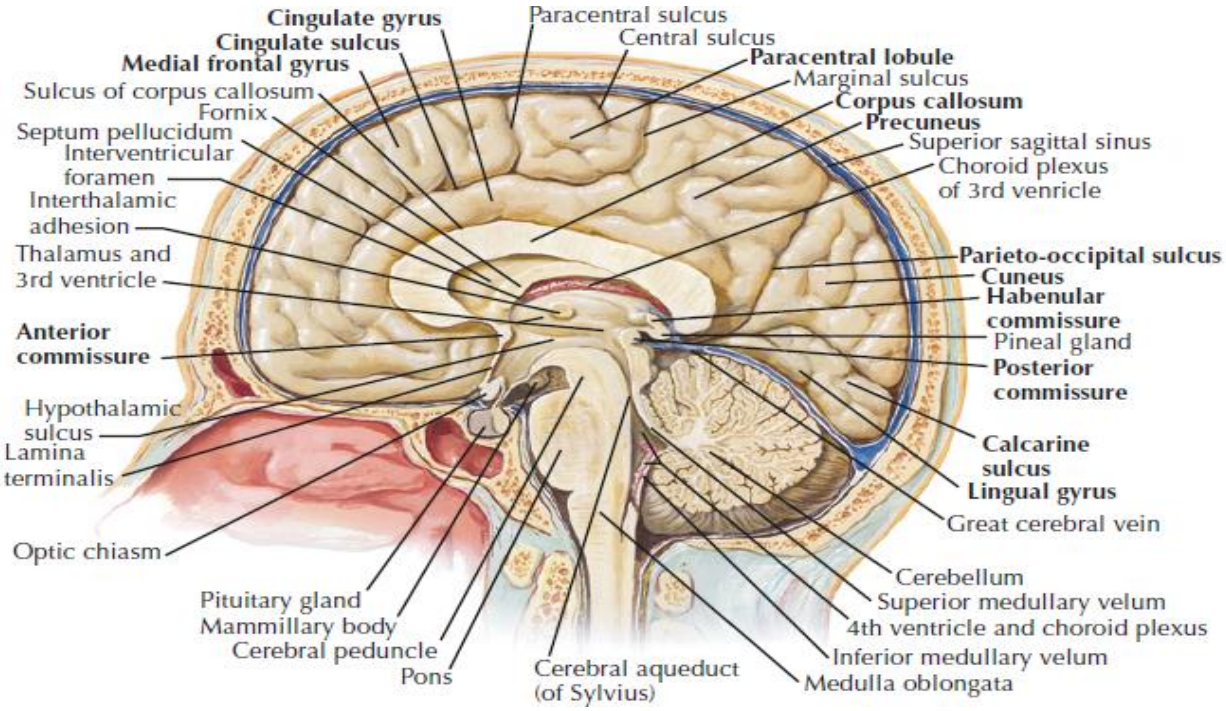
According to section, it can be seen that the surface of the brain has a gray color, and most of the area is based deeper, lighter, due to the denser position of the bodies of neurons of the surface area (cortex). precentral) gyrus praecentralis (gyrus praecentralis) reaches 5 mm in certain areas.

2.1. Sulci and gyrus of the hemispheres of brain cortex

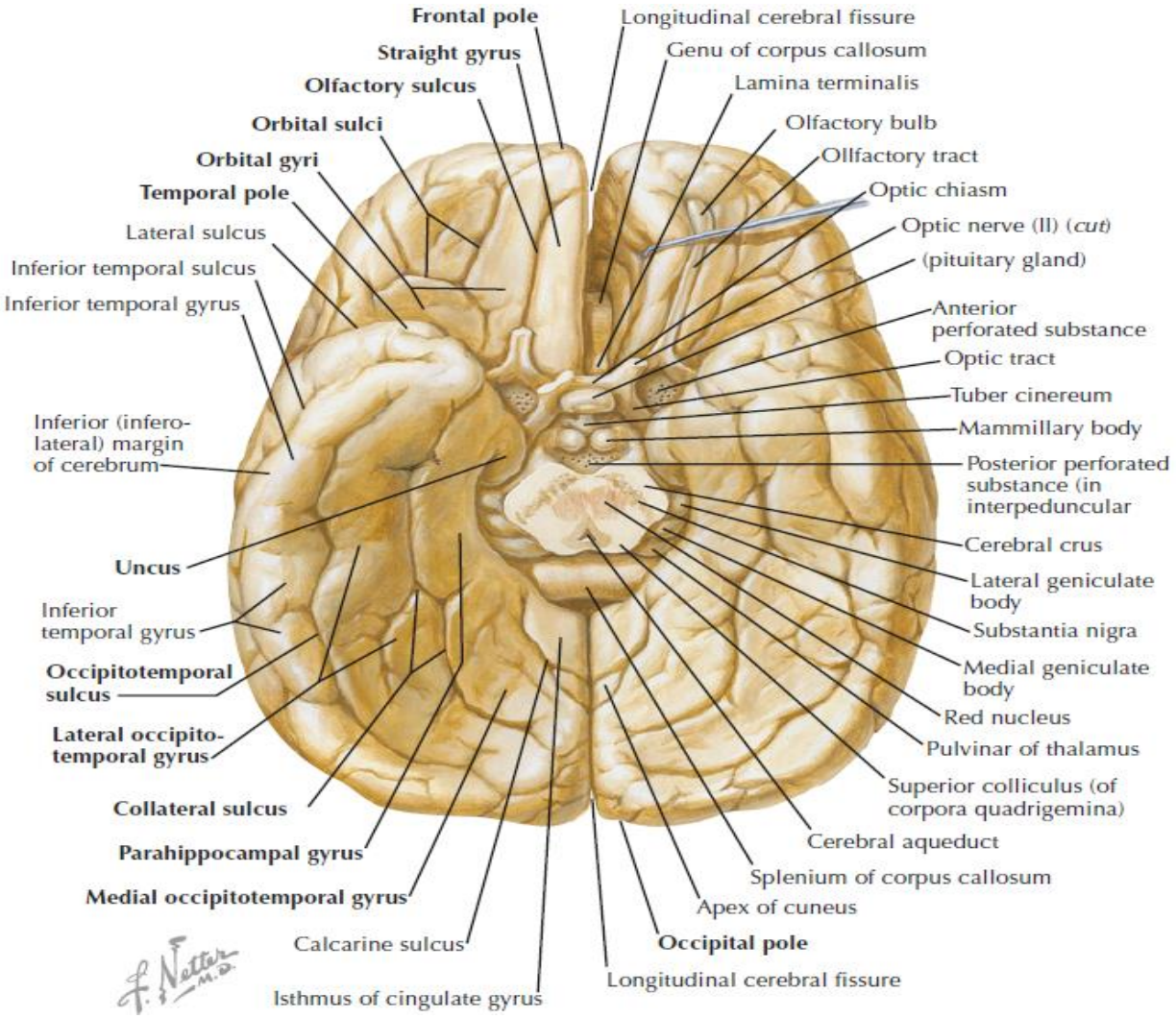
I. **Dorso-lateral surface**



II. **Medial surface**

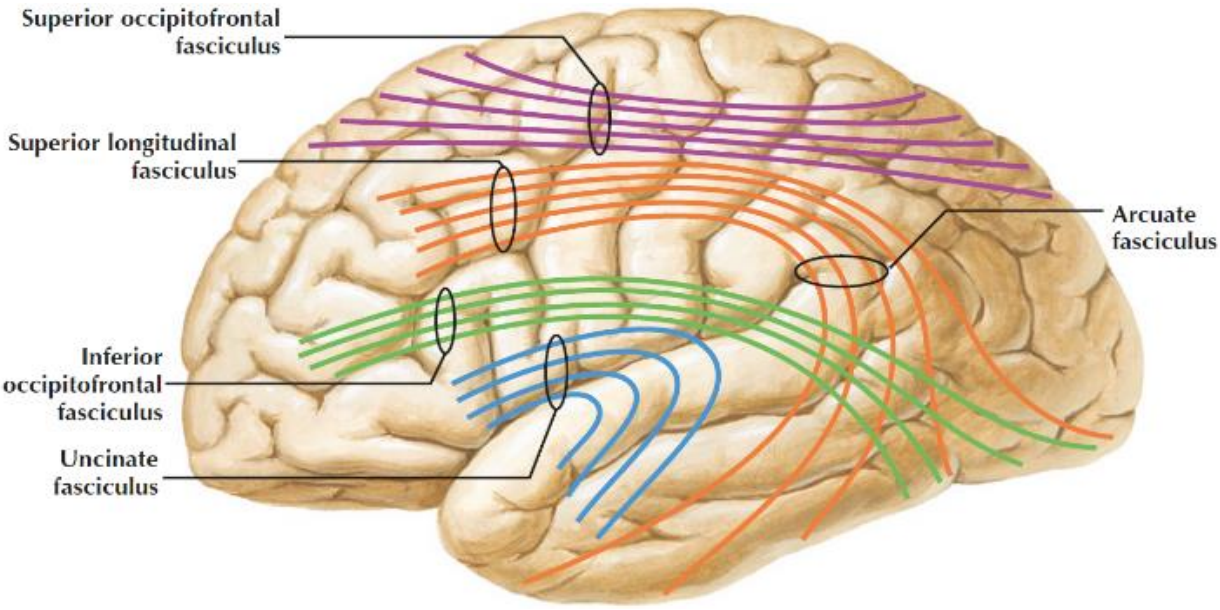


III. **Lower surface**



2.2. White matter of cerebrum

I. **Associative fibers**

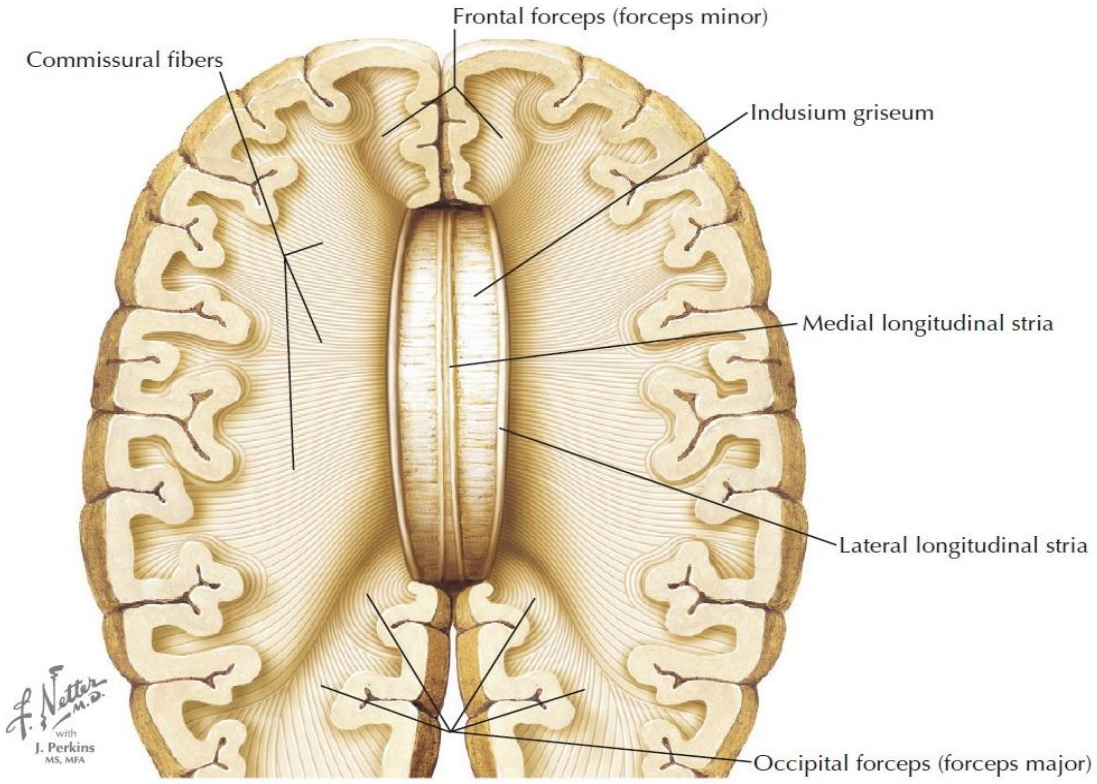


1. Upper longitudinal bundle (fasciculus)

2. Lower longitudinal bundle (fasciculus)
3. Unciform fasciculus (uncinate fasciculus)
4. Other important associative fibers

II. **Commissural fibers**

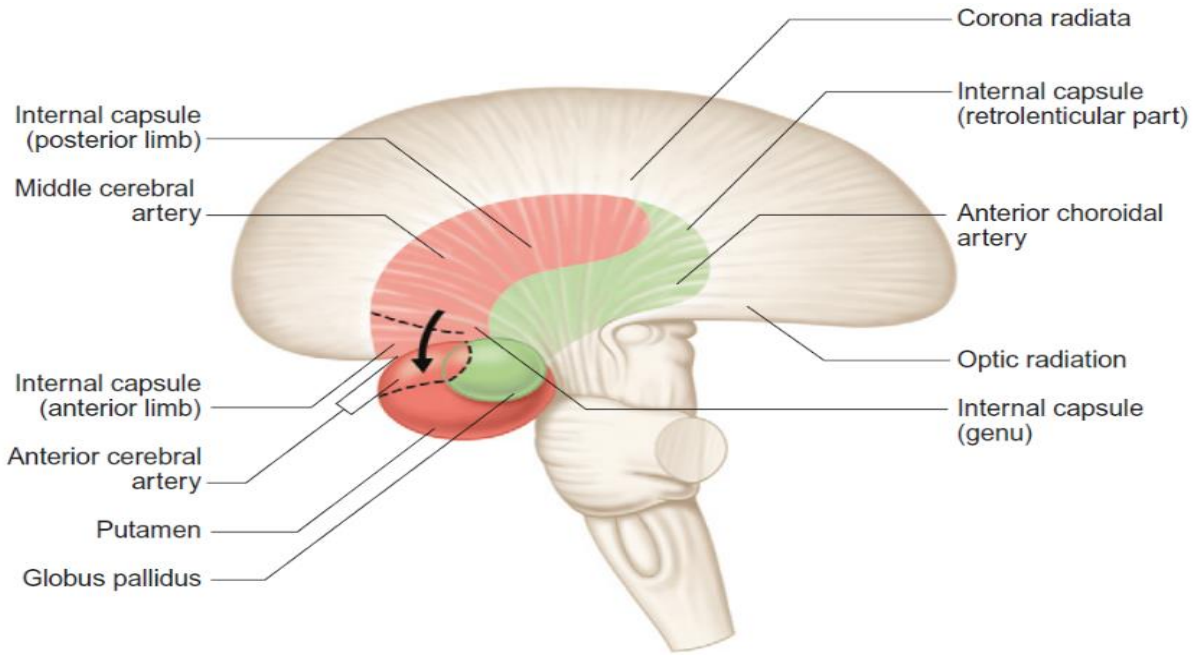
1. Corpus callosum



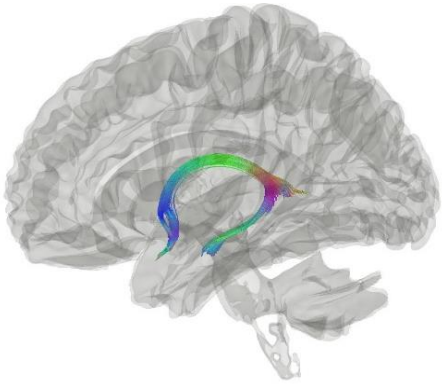
Schematic view of the lateral extent of major components

2. Anterior commissure (commissura anterior, precommissure)

III. **Projection fibers' system**



Arterial supply to the internal capsule and parts of the basal ganglia of the left cerebral hemisphere. The outer layers of the hemisphere have been removed to reveal these structures. The putamen and globus pallidus are displaced downward to display the internal capsule. Territory supplied by branches of the anterior and middle cerebral arteries is shown in red. Territory supplied by the anterior choroidal artery is shown in green. (Pic. from GRAY'S CLINICAL NEUROANATOMY: THE ANATOMIC BASIS FOR CLINICAL NEUROSCIENCE, ISBN: 978-1-4160-4705-6)



1. Psalterium (Lyra)
2. Fornix
3. Transparent membrane
4. Internal capsule (capsula interna)
5. Corona radiata

Corona radiata **Corona radiata**

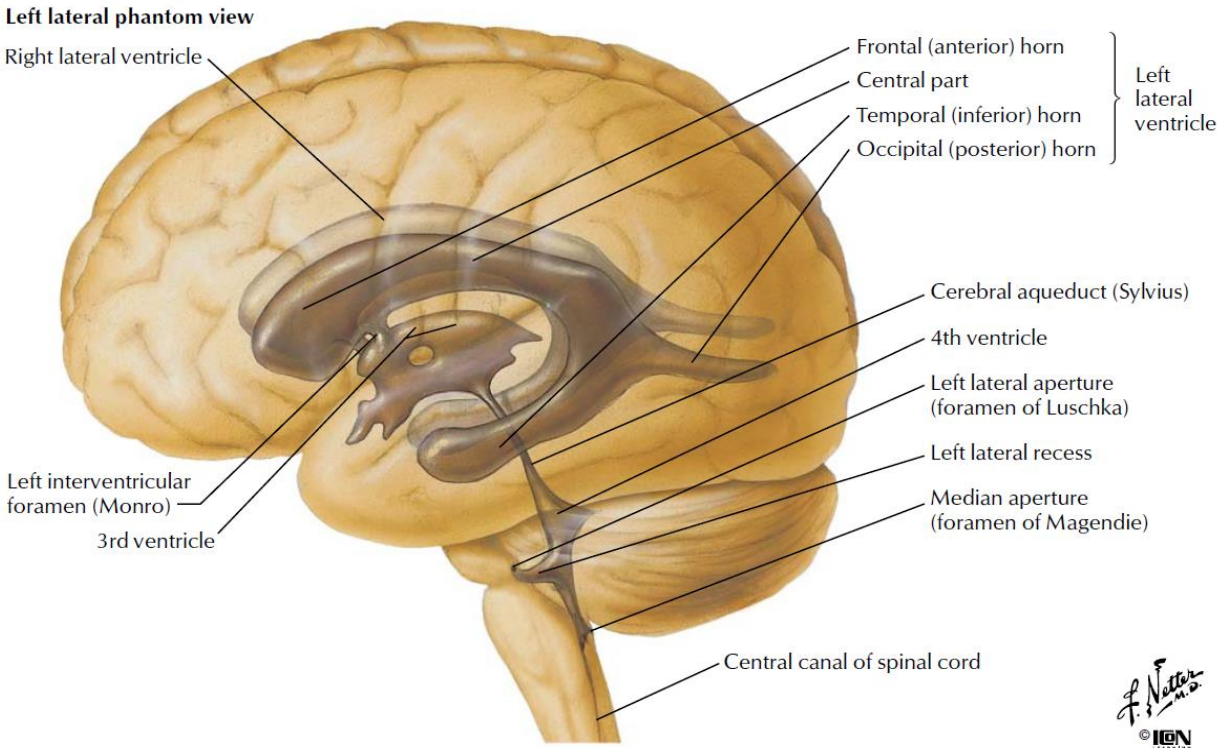
1. Lateral ventricles

a. Front horn

b. Rear horn

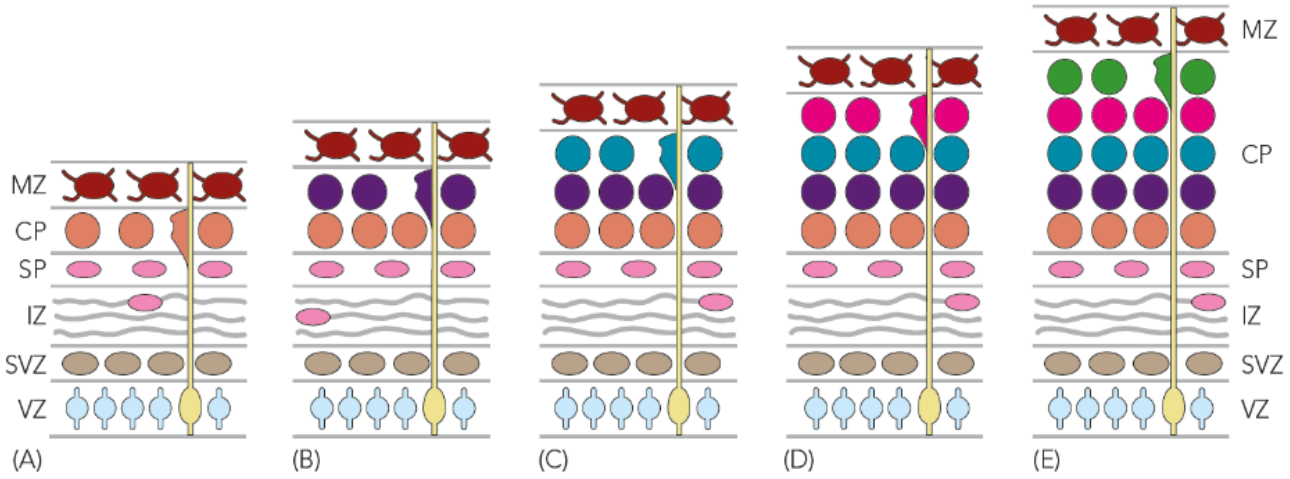
c. Lower horn

d. Ammon's Horn



3. Cytoarchitectonics and myeloarchithectonics of the cortex of the large hemispheres (layered structure)

All neurons, and neurons of the cerebral cortex, are laid in the ependym next to the cavity of the neural tube (a precursor to the ventricles), so the so-called preliminary plate (pre plastic) is formed,which is further divided into the edge zone and subplastic,and it is between these two formations that the six-layer layer of the cerebral cortex develops to phylogenetically the oldest are the V and VI layers, thenewest cells are localized in the first layers. "Young" cells bypass the "old" passing from the ependym to the cortical plate along the radial glial fibers. The process is called neuronal migration. The regulators of the process are the Cajal-Retzius cells, which are based in the marginal zone of the cerebral cortex and synthesize pandlin (Eng.: reelin) – glycoprotein, which directsnon-tear stem cells during migration along radial glial fibers. Violation of neuronal migration is a generalized concept for the pathology of the formation of neurons, a violation of the process of their migration or the process of their separation from radial glial fibers.

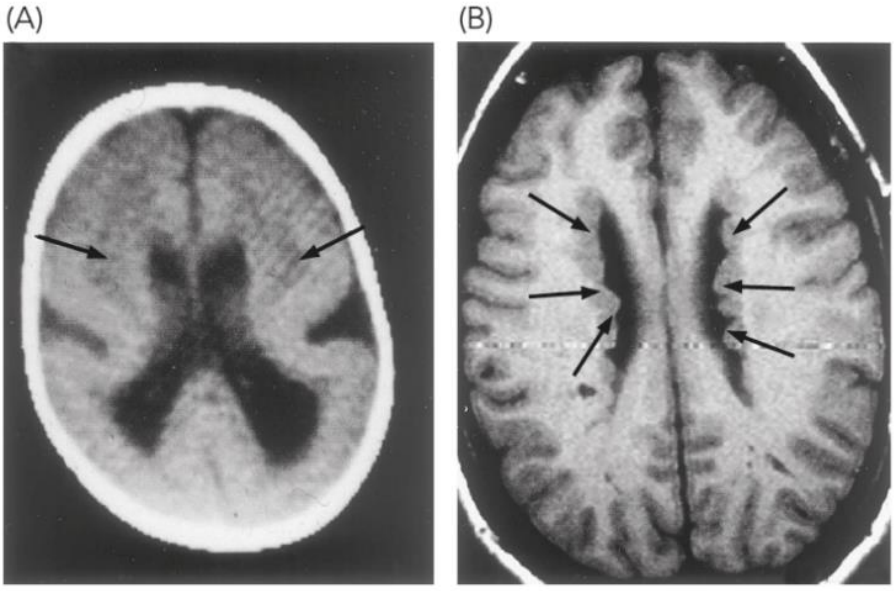


Cerebral cortical neurons rely on inside-out patterning to form layers. (A) *As neurons leave the ventricular zone (VZ) by attaching to radial glial cells (RG, yellow), they begin to accumulate in the cortical plate (CP), detaching from the RG prior to reaching the marginal zone (MZ). The first cells to arrive at the CP will form the deepest, innermost layer (future layer VI). (B) The next layer of neurons (future layer V) will migrate past the first layer to form a new layer closer to the pial surface. (C) The third layer of cells to form (future layer IV) will migrate past the first and second layers.*

Similarly, the next layers (future layers III and II) will continue to migrate past existing layers until all six cortical layers have formed using this inside first, outside last pattern (D and E)(Pic. from Developmental neurobiology, ISBN 9780815344827).

Defects in neuronal migration results in congenital disorders in humans

(A) A magnetic resonance imaging (MRI) scan illustrating the smooth brain appearance characteristic of lissencephaly. Compared to a typical brain or a brain with periventricular heterotopia (PH), the outer surface of the brain appears smooth and lacks the characteristic gyri and sulci. In addition, the ventricles are enlarged and some areas of misplaced neurons are observed in the white matter (arrows). (B) A brain scan illustrating PH. The arrows indicate nodules of neurons that cluster along the ventricles. (From Gressens P [2000] Pediatric Res 48:725–730.)



3.1. Layers of the cerebral cortex, histology of the cortex

Differentiation into layers in the cortex in some areas is noticeable even in the section (for example, Gennari's strip), but in mostcases, the layer structure of the cerebral cortex can be seen only under a microscope.

1874 – was year of the discovery of Betz cells, named after the author of the study Volodymir Oleksievich Bets, Ukrainian anatomist and histologist, professor of the Universityof Kiev, who foundgiant pyramidal cells in the cortex of the anterior central gyrus of man and in the motor region of the animal cortex,and also proved the absence of cell data in other areas of the cerebral cortex by electrostimulation. in an attempt to elicit a motor response.

Betz's research became the basis of thedoctrine of cytoarchitectonics and the work of such prominent scientists as the German anatomist Corbyniana Brodmann (Brodmann fields), the French neurophysiologist and neurologistof Cecil and Oskar Vogt(concept of isocortex). – areas of the neocortex,which are characterized by a six-layer structure).

The cortex consists of two main types of neurons:

- Pyramidal cells – excitatory projection neurons (form communications over long distances).
- Non-firamide cells – galtormenting neurons (granular cells and interneurons; form communications at short distances).

Isocortex and alokortex

Isocortex, as mentioned earlier, is an area of the neocortexthat is characterized by a six-layer structure, and the allocortex or heterogenetic cortexis an older area of the neocortex,which is divided into paleocortex and archicortex,which weredescribed in more detail earlier in these methodological recommendations.

3.2. Type of cortex structure

1. Homotype cortex - withtandart six-layer bark structure

A. Molecular layer.

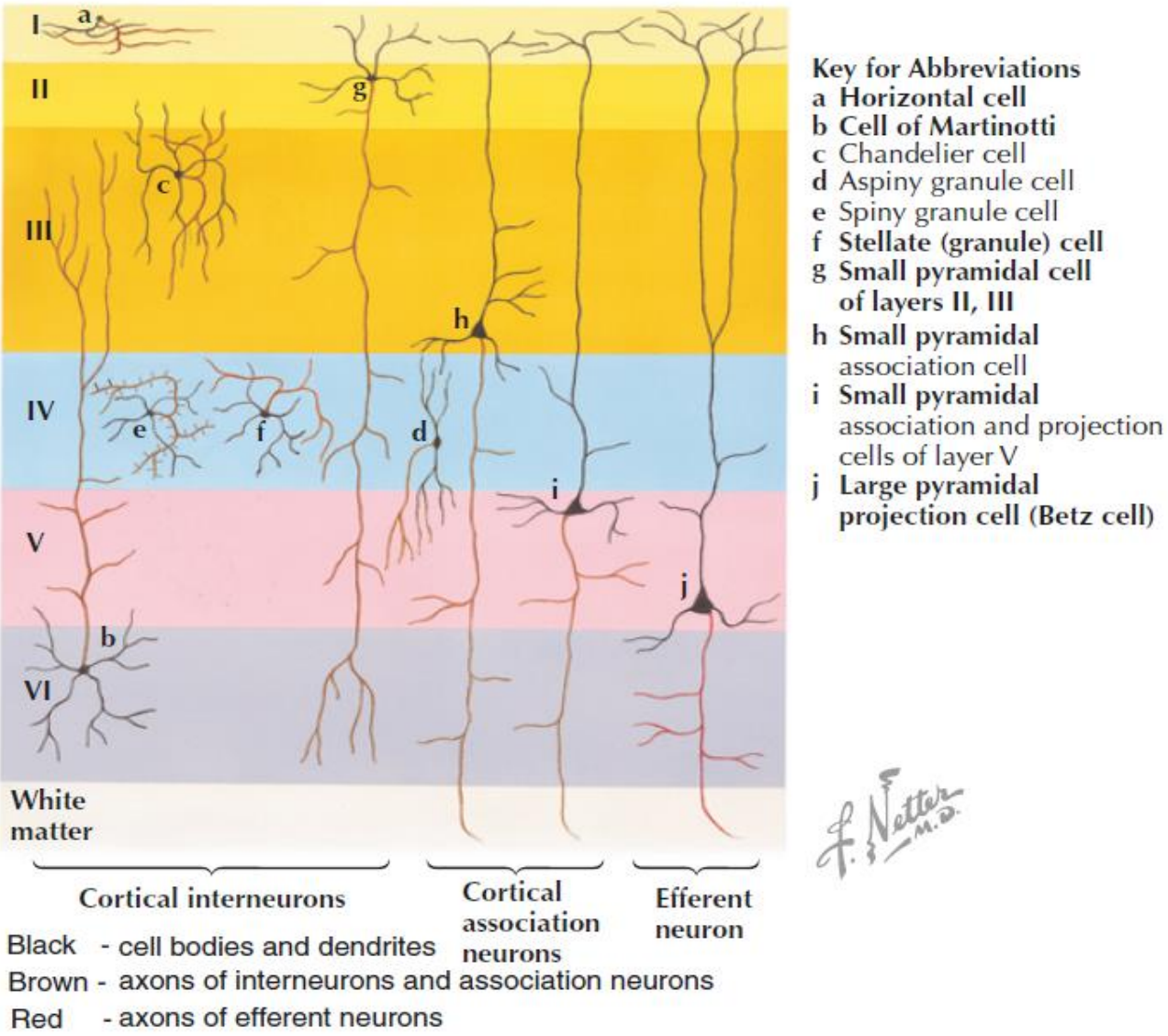
Also, according to the new classification, it is called "plate I". Contains relatively few cells, mainly small cells kahal-Ratzius, which have already been discussed in previous units. dendrites of these cells pass in the thickness of the cortex tangentially.

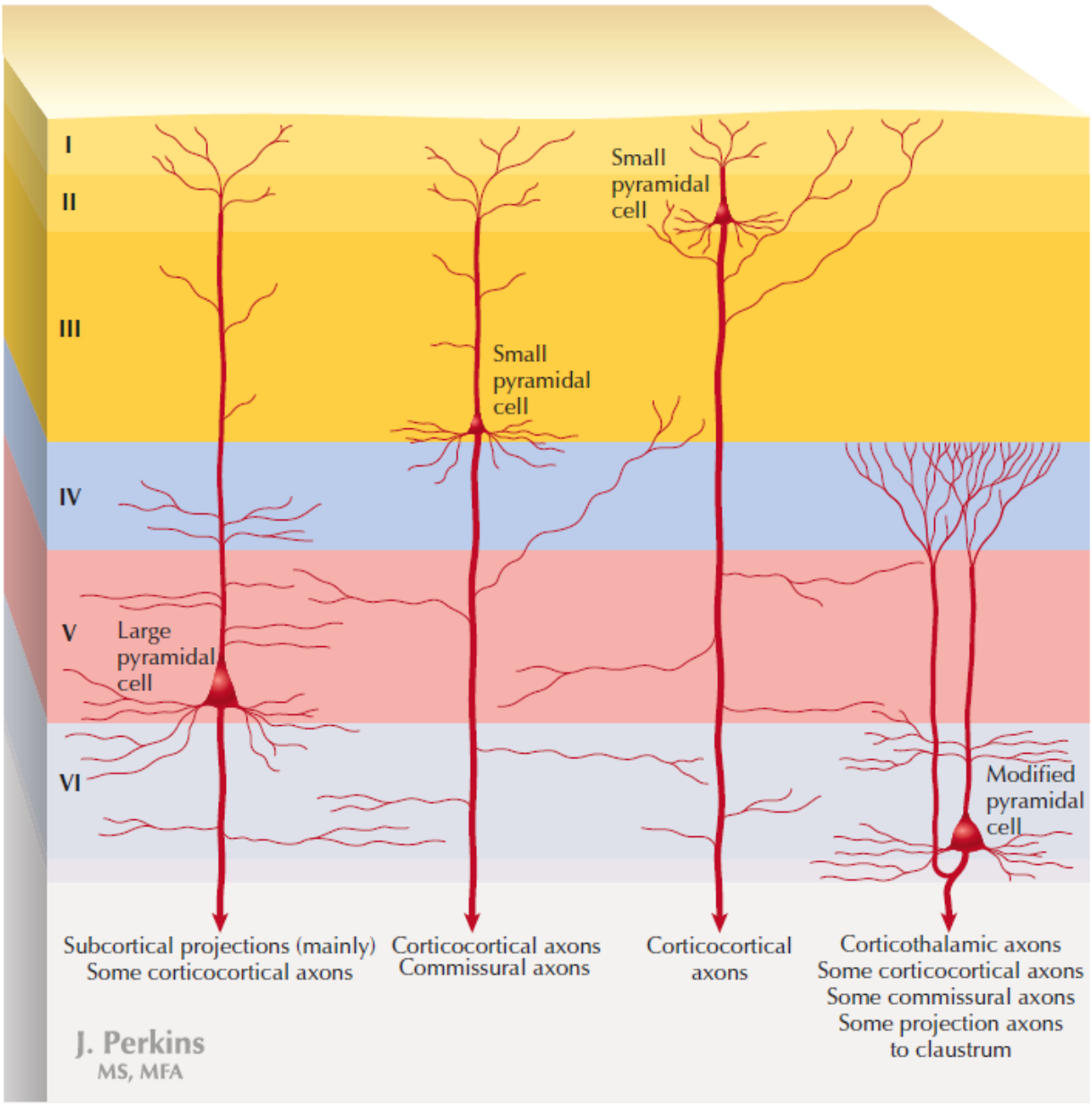
B. External granular layer.

Plate II: at this level there are mainly granular "non-pyramidal" cells (GABA-ergic tormose neurons) and a small number of pyramidal neurons (glutamate-dependent activating cells).

C. External pyramidal layer.

Plate III: smaller in size than representatives of the IV-V plates pyramidal cells prevail. The axons of these cells are covered with a significant layer of myelin already at the level of departure from the body of the neurocytes.





D. Internal granular layer.

Plate IV:many non-firamide cells (granular afferent cells that perceive information from thalamic neurons).

E. Internal pyramidal layer.

Plate V: a layer of medium and large pyramidal cells, the largest of which are called Betz cells, described above. along these pathways is high due to the thick layer of myelin on the axons of neurons compared to the above layers. The place of formation of the inner strip of bayarge (stria laminae ganglionaris).

F. Multiform layer (layer of polymorphic cells).

Plate VI:alayer of polymorphic cells with an inner part with small tightly equipped cells and with an outer part consisting of relatively larger cells.

2. Heterotype cortex - and thetypical structure ofthe cortex, where there is no standardofan pt number of layers

In heterotype areas of the isocortex, the six-layer structure ofthe cortex is generally disturbed. Agranull are completely or partially devoid of external and internal granular layers with the replacement of cells of these layers with pyramidal cells of different sizes. Granular heterotype is a radically opposite histological picture that can be observed in the fissurae calcarinae region, where the pyramidal cells of the III and V layers are replaced by granular cells “eight-layer cortex”. The granular area is called the "dusty cortex".

Agranular heterotype is more characteristic of the motor cortex, granular heterotype - for the sensory cortex.

3.3. Myeloarchitectonic

Myeloarchithectonics of the brain also has 6 layers:

- The first layer is lamina tangentialis (layer of tangential fibers);
- The second layer is lamina disfibrosa (layer with a small amount of myelin);
- The third layer is lamina suprastriata (the layer lying above the Baillargers’ tangential bands);
- The fourth layer – stria Baillargeri externa (Baillarger's external tangential band);
- The fifth layer – stria Baillargeri interna (Baillarger's internal tangential band);
- The sixth layer is lamina infrastriata (layer under the Baillargers’ tangential bands).

3.4. Areas and fields of the cerebral cortex

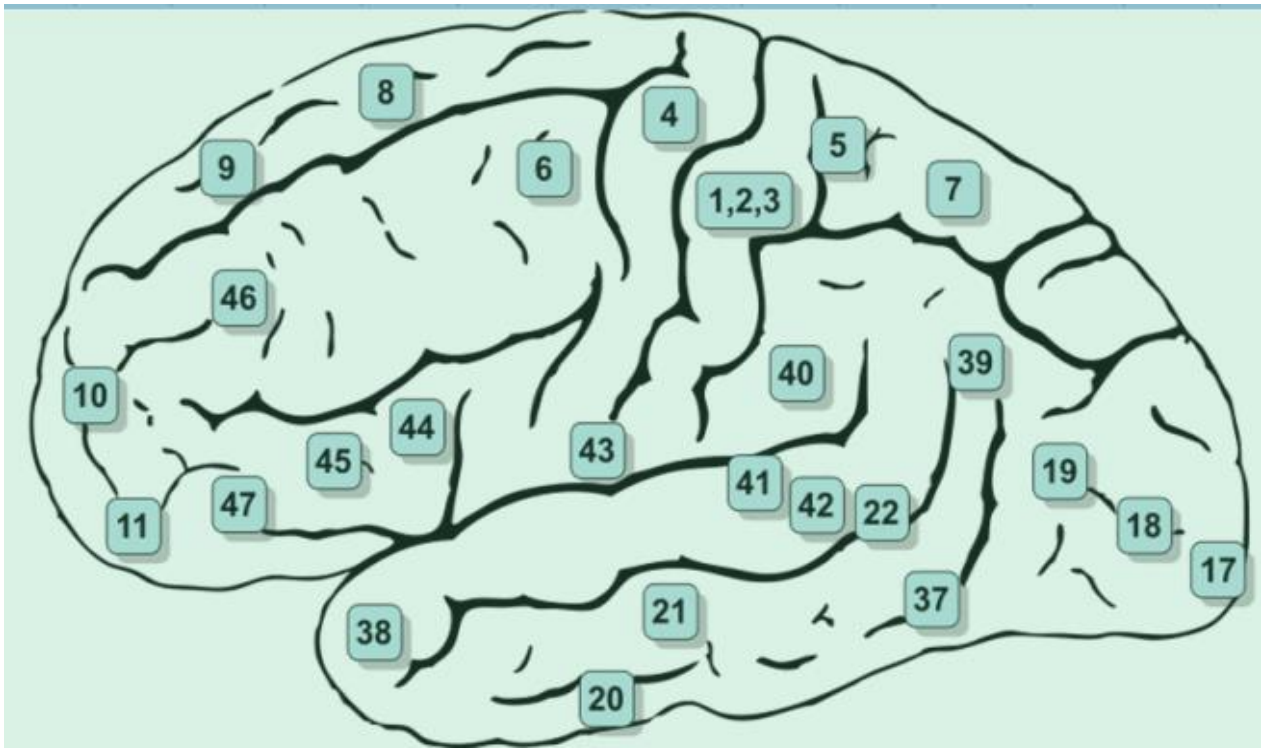
The Cytoarchitectonic Map of Korbinian Brodmann

Plasticity of cortical architectonics

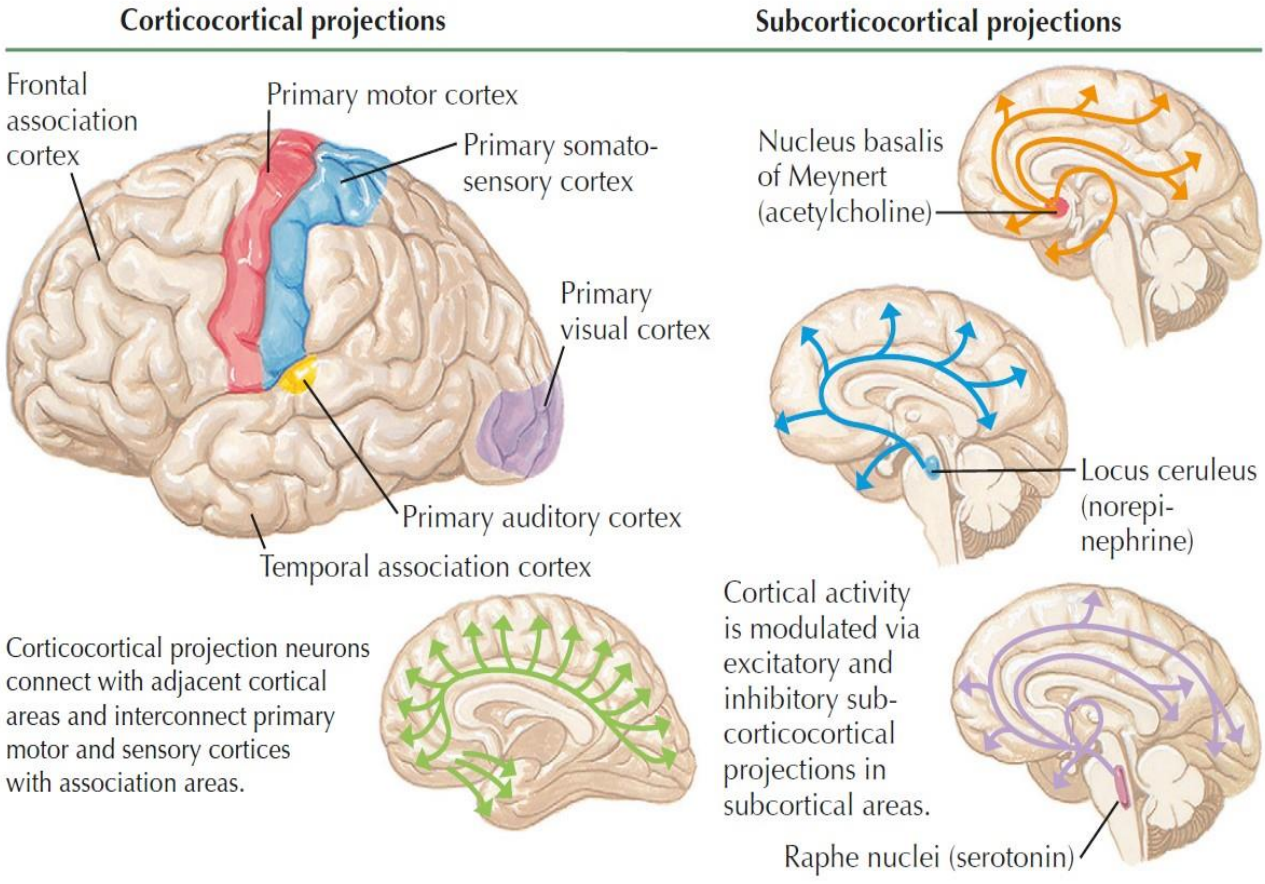
Physiology of the cerebral cortex.

Localization of functions in the cortex

Brodmann's projection areas



Primary cortical fields



A. Primary somatosensory cortex.

Areas 3,1,2. It's located in the post central gyrus. The primary somatosensory cortex is responsible for the conscious perception of pain and temperature as well as somatic sensation and proprioception, mainly from the contralateral half of the body and face. Its afferent input is derived from the ventral posterolateral and posteromedial nuclei of the thalamus. Histologically, this area would consist of granular cortex. The sensory homunculus includes cortical representation of the body based on the degree of sensory innervation. Very sensitive areas such as the lips and the fingertips have a huge representation. Neurons within each cortical site (particularly layer IV) are arranged in columns representing specific body regions. If a region is amputated (such as a finger) there is reorganization with neurons responding to stimulation of adjacent body parts. This can also happen as the result of increased use of a body part. Damage to the sensory cortex results in **decreased sensory thresholds**, an inability to discriminate the properties of tactile stimuli or to identify objects by touch.

B. Primary visual cortex.

Area 17. It's located on the lips of the calcarine fissure and adjacent portions of the cuneus above and the lingual gyrus below. The cortex is granular in type and extremely thin. Layer 4 is relatively thick with a prominent outer band of Baillarger (line or band of Gennari), which is visible grossly and gives the area its designation of striate cortex. Area 17 receives the geniculocalcarine projection, which is retinotopically organized The striate area receives primary visual impressions: color, size, form, motion, and illumination. Ictal activity or electrical stimulation of the calcarine cortex produces unformed visual hallucinations, such as scotomas and flashes of light. Destructive lesions cause defects in the visual field supplied by the affected areas [DeJong's the neurologic examination. 8th edition (2020)].

The parastriate region (area 18) and the peristriate region (area 19) receive and interpret impulses from area 17.

C. Primary auditory cortex.

The transverse temporal gyri are the primary auditory cortex (areas 41 and 42). Immediately adjacent to the primary auditory cortex is the auditory association cortex

(area 22); in the dominant hemisphere, part of this is the Wernicke's speech area. The primary auditory cortex is tonotopically organized with high frequencies medial and low frequencies lateral [DeJong's the neurologic examination. 8th edition (2020)].

D. Primary cortex of taste.

E. Primary vestibular cortex.

F. Primary motor cortex.

The pyramidal (corticospinal) system arises from the primary motor cortex in the precentral gyrus. The premotor and supplementary cortices control the planning and preliminary preparation for movements, which the primary motor cortex in the precentral gyrus then executes. The primary motor cortex also receives input from the basal ganglia and the cerebellum.

Area 4 (area gigantopyramidalis) of the precentral gyrus is the primary motor cortex (M-I); it is the region having the lowest threshold for stimulation to cause contraction of muscles of the opposite side of the body. The cortex of M-I is agranular and heterotypical; its most characteristic feature is the presence of giant pyramidal neurons (Betz cells) in lamina V. The localization of function within the precentral gyrus is depicted by the motor homunculus.

There are reciprocal connections between the primary motor cortex and the primary somatosensory cortex in the postcentral gyrus. M-I receives association fibers from the premotor and supplementary motor areas and from the insula. These connections are involved in the preparation and planning for voluntary movements that are then executed by the primary motor cortex. There are also connections between the primary motor cortices in the two hemispheres. The posterior division of the ventral lateral nucleus of the thalamus receives input from the cerebellum and projects to area 4 [DeJong's the neurologic examination. 8th edition (2020)].

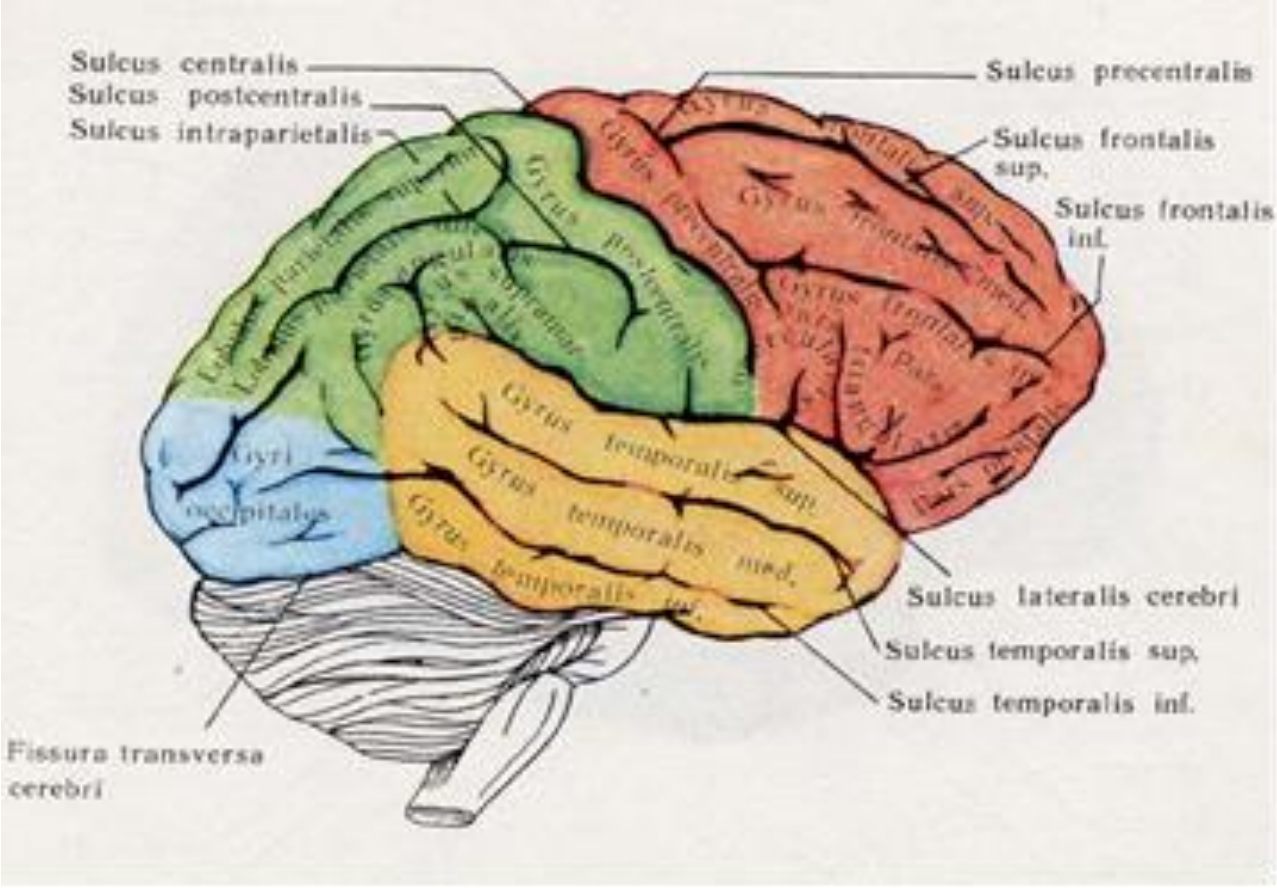
Associative zones of the cortex.

1. Secondary cortical fields
2. Tertiary cortical fields

4. Localization of function in the brain cortex.
Motor and sensory representations in the cortex.
Syndromes of defect of some parts of hemispheres: the frontal, temporal, parietal, occipital lobes, the limbic cortex.

Localization of function in the brain cortex.

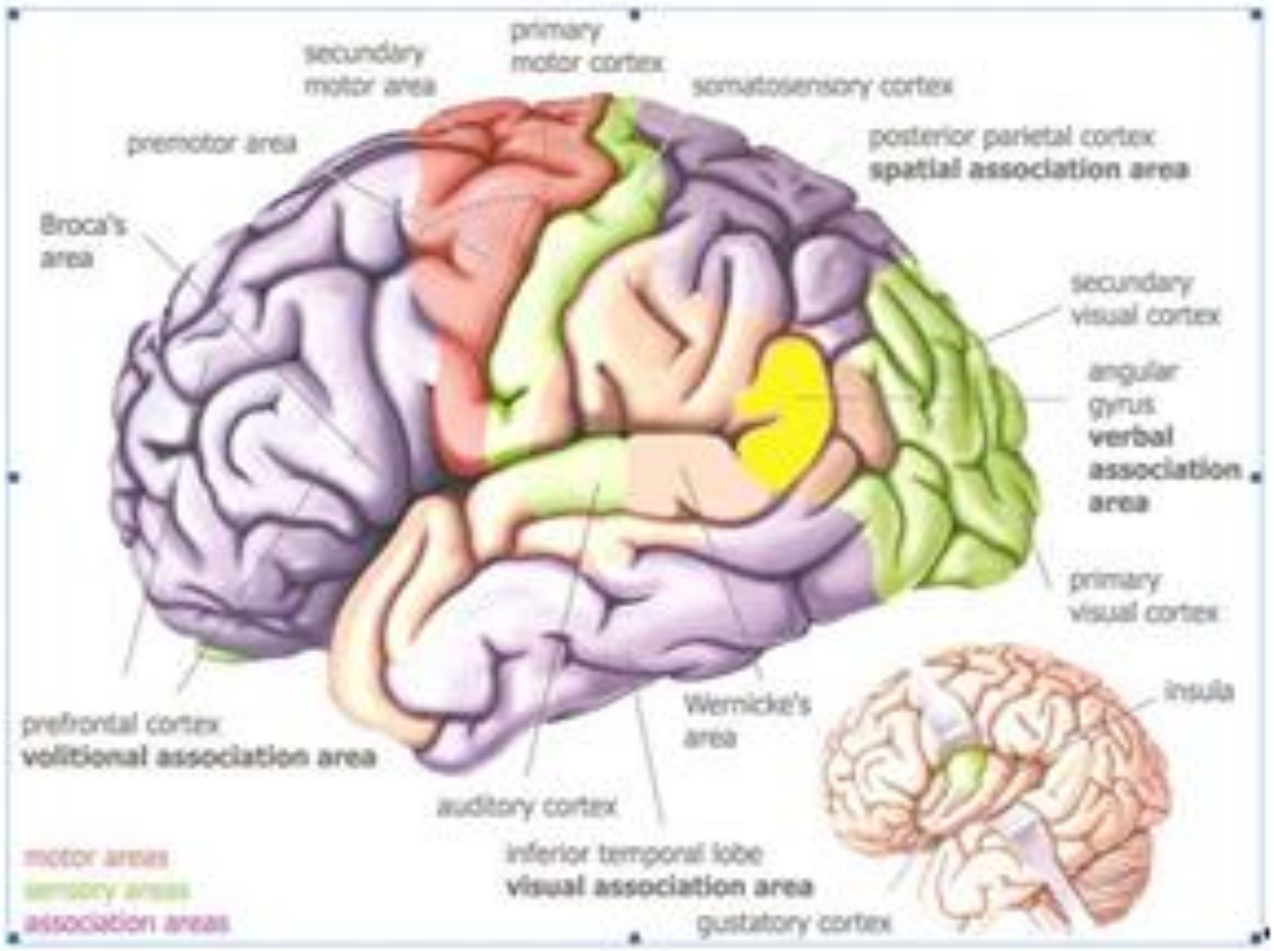
The cerebral cortex is the outer covering of gray matter over the hemispheres. This is typically 2–3 mm thick, covering the gyri and sulci. Certain cortical regions have somewhat simpler functions, termed the primary cortices. These include areas directly receiving sensory input (vision, hearing, somatic sensation) or directly involved in production of limb or eye movements. The association cortices subserve more complex functions. Regions of association cortex are adjacent to the primary cortices and include much of the rostral part of the frontal lobes also regions encompassing areas of the posterior parietal lobe, the temporal lobe and the anterior part of the occipital lobes. These areas are important in more complex cortical functions including memory, language, abstraction, creativity, judgment, emotion and attention. They are also involved in the synthesis of movements.



Frontal lobe (FL). The FL contains most of the dopamine-sensitive neurons in the cerebral cortex. The dopamine system is associated with attention, short-term memory tasks, planning, and motivation, select sensory information arriving from the thalamus to the fore-brain. A report from the National Institute of Mental Health says a gene variant that reduces dopamine activity in the prefrontal cortex is related to inefficient functioning of the brain, to slightly increased risk for schizophrenia.

The prefrontal cortex (PFC) is the anterior part of the frontal lobes, lying in front of the motor and pre-motor areas. The disorder of back part of the third frontal gyres (Broca`s zones) causes an efferent motor aphasia. Often accompanied by agraphia (isolated defeat of back part of the second frontal gyres on the left). A motor aphasia and agraphia are types of apraxia.

Neuroanatomical connections



Frontal ataxia. The frontal way of ponds, relating to the cerebellum system, begins in a frontal lobe, is characterized by disorders of standing and walking-astasia-abasia (falling and impossibility of walking). In extremities we can see an incorrect

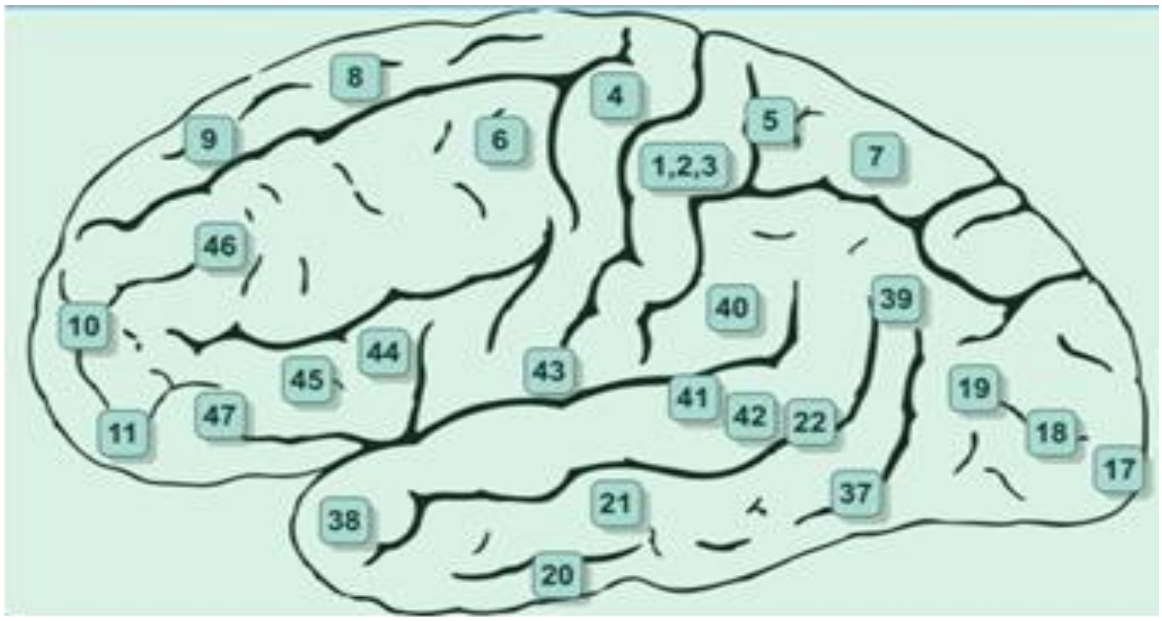
finger-nose test (a finger don`t touch the nose by opposite to the lesion hemisphere hand) or finger-hammer test.

Disorders of mental: emotionally-volitional sphere (apathetic and abulia syndrome), incompleteness of plan of actions (frontal apraxia), torpid mind, euphoria, no understanding of seriousness of own state, absence of criticism to the own behavior, depression, aggressiveness.

Frontal apraxia – it is not only absence of plan of action but also don`t finish of it to the end or perversion.

The irritation of frontal lobe by the tumor or hematoma causes the attacks of Jackson epilepsy(usually the attacks begin with a convulsive turn the head and eye in an opposite side). Symptoms of closing of eyelids (Kohanovsky), grabbing phenomenon of Yanishevsky.

Brodmann's projection areas



The primary projection areas are those that receive most of their sensory impulses:

• directly from the thalamic relay nuclei;

• (primary somatosensory cortex; Brodman areas 1, 2, 3), the visual (area 17), or the auditory (areas 41, 42) pathways. The primary motor cortex (area 4) sends motor impulses directly down the pyramidal pathway to somatic motor neurons;

• within brainstem and the spinal cord.

The secondary projection areas (motor, areas 6, 8, 44; sensory, areas 5, 7a, 40; visual, area 18; auditory, area 42), which subserve higher functions of coordination and information processing, and the tertiary projection areas (motor, areas 9, 10, 11; sensory, areas 7b, 39; visual, areas 19, 20, 21; auditory, area 22), which are responsible for complex functions such as voluntary movement, spatial organization of sensory input, cognition, memory, language, emotion.

Types of fibers

• The association fibers integrate information from different receptors or sensory areas, sends nerve impulses to the motor areas to give responses and located within one hemisphere.

• Commissural fibers conjunct both hemisphere (corpus callosum).

• Projection fibers are located within different parts of hemisphere and conjunct up and down located structure.

Disorders of corpus callosum

• Interruption of the corpus callosum can produce various disconnection syndromes.

• Total callosal transection causes splitbrain syndrome, in which the patient can`t name an object, felt by the left hand when the eyes are closed, or one seen in the left visual hemifield (tactile and optic anomia), and can`t read words projected into the left visual hemifield (left hemialexia), write with the left hand (left hemiagraphia), or make pantomimic movements with the left hand (left hemiapraxia).

• Anterior callosal lesions cause alien hand syndrome (diagonistic apraxia), in which the patient can`t coordinate the movements of the two hands. Disconnection syndromes are usually not seen in persons with congenital absence (agenesis) of the corpus callosum.

Temporal lobe

Sensory aphasia – arises up at the defeat of the left temporal lobe for right-handed(back part of superior temporal gyres – zone of Wernike), understanding of the speech is lost, including and writing, control after own speech, speech of patient as “salad from words". Amnesic aphasia – it is the disorder of back part of temporal

lobe and lower part of parietal lobe – loses ability to determine the names of objects, understanding their destiny(What it is a pencil patient don`t knows and speak:"it is for the writing").

Symptoms of disorders

• Parietal-temporal region: results in semantic aphasis – understanding of semantic value of order of words in the sentence is disorder in suggestion (for example, father of brother and brother of father).

• **Temporal ataxia**- attacks of temporal-cortex dizziness.

• **Quadrant hemianopsia**-fall of opposite fields of the vision.

• **Symptoms of irritation** – are olfactory, taste, auditory auras as an **attacks of temporal epilepsy**.

Parietal lobe. The defeat of the left parietal lobe for right-handed(dominant hemisphere) in area of g. supramarginalis lead to the motor apraxia – it is lost ability to reproduce difficult purposeful motions in the case of absent of paralyses and has ability of elementary motions(can`t fasten buttons, loss of skills of symbolic motions). Constructive apraxia causes if the lesion in a lower-parietal lobe on the left.

Alexia – is a loss of capacity for understanding of written in the defeat of the g.angularis. Astereognosis – defeat of area behind of postcentral gyres.

Afferent motor aphasia – the lesion is behind of lower part postcentral gyres – blockade of afferent impulses behind of a lower part postcentral gyres from the speech part of postcentral gyres. We can see the elements of oral apraxia. An articulation is violated, instead of one word the similar on sound speaks to him.

Autotopagnosia – unrecognition of parts of own body or distorted perception him. Pseudomelia (feeling of presence of the third hand-leg, a hand can seem continuation of shoulder, but not forearm. A defeat of right parietal lobe is combination of autotopagnosia and anosognosia – absence of realization of the own defect, paralysis).

Lesion on border of parietal, temporal and occipital lobes cause syndrome of Gertsman – is acalculation-disorder of account, digital agnosia and right – to the left orientation. Lesions in deep parts of parietal lobe cause down - quadrant hemianopsia.

Occipital lobe. The lesions in area of s.calcarina on the internal surface of occipital lobe causes the homonimus hemianopsia-fall of opposite eyes fields. The partial defeat of this area causes a **quadrant hemianopsia** – higher than s.calcarina – in opposite lower quadrants(cuneus), below – g. lingualis is a defeat of opposite overhead quadrants. **Scotomas** are as a insular falls of eyes fields in opposite of the same name quadrants.

Metamorphopsia is violation of correct recognition of contours of the subjects – at a defeat on the left side.

Visual agnosia - loss of ability of recognition of objects by appearance (without the loss of eyesight and ability to know objects by touch).

Syndromes of damage to certain areas of the cerebral cortex

I. Violation of speech. Aphasia and its types

1. Non-fluent aphasia (motor, Broca's aphasia)
2. Wernicke's aphasia
3. Conduction aphasia
4. Total aphasia
5. Amnesic aphasia
6. Transcortical motor aphasia
7. Disengagement Syndromes

a. Disengagement Syndromes in smell analysis system

b. Disengagement Syndromes in the visual system

II. Apraksia

1. Motor apraxia: ideomotor and ideatory
2. Constructive apraxia
3. Amnestic apraxia
4. Apraxia of gait
5. Sensory apraxia
6. Apraxia of speech

III. Agnosia

1. Visual agnosia
2. Somatosensory agnosia

a. Astereognosis

b. Asomatognosia

c. Gerstmann syndrome

d. Balint syndrome

IV. Ignoring. The phenomenon of suppression

V. Behavioral disorders

1. Lesions of the prefrontal convexital cortex
2. Damage to the orbitofrontal cortex

5. The cognitive functions and their disturbances.

Cognitive functions encompass the mental activities of thinking, learning, and remembering. Different types of mental activity are called cognitive functions. These are individual mental processes required for human behaviors. Cognitive functions are used for remembering, reasoning, planning, problem-solving, and decision-making. They can be divided into declarative and procedural functions.

Learning may be classified as introverted or extraverted and as sensing, intuition, thinking, or feeling. Cognition is the process of acquiring and understanding knowledge through thoughts, experiences, and senses. Learning requires a high level of attention and to focus on audible or printed language.

Memory plays an important role in intelligent behavior. It is divided into short-term and long-term forms as well as episodic and working memory.

Intelligence can be defined as a general mental ability for reasoning, problem solving, and learning. Intelligence integrates cognitive functions such as perception, attention, memory, language, and planning. Intelligence consists of learning from experience, adapting to new situations, handling abstract concepts, and manipulating the environment. The many types of intelligence include linguistic, logical-mathematical, spatial, musical, bodily kinesthetic, interpersonal, and intrapersonal. Individual intellectual development includes the sensorimotor, preoperational, concrete-operational, and formal-operational periods.

Simple and highly complex behaviors can be learned throughout the lifespan. There are widespread individual differences in the ability to reason, solve problems, and learn, which lead to differences in the general ability to cope with challenging situations.

Executive skills are higher-level examples of cognition that include organization and regulation. When there are problems with executive skills, signs and symptoms often resemble those of attention-deficit/hyperactivity disorder.

Comprehension is the action or capability of understanding, connecting objects or attributes to appropriate memories.

Mild cognitive impairment is the stage between the expected decline in memory and thinking that happens with age and the more serious decline of dementia. It may include problems with memory, language, or judgment. Cognitive disorders often begin subtly but progress until they impede the quality of life. Cognitive disorders include dementia, amnesia, and delirium more. Cognitive disorders that affect language, reading, or writing include aphasia, alexia, and agraphia.

It is important to understand the various cognitive disorders, their symptoms, and relevant treatment options.

6. The syndrome of chronic vegetative state

A vegetative state is when a person is awake but showing no signs of awareness. On recovery from the coma state, VS/UWS is characterised by the return of arousal without signs of awareness. In contrast, a coma is a state that lacks both awareness and wakefulness. Absence of awareness can only be inferred by lack of responsiveness to the environment and not as lack of consciousness that we may not be able to detect by behavioural measures. For this reason, many authors have suggested that the term 'Unresponsive Wakefulness Syndrome' (UWS) (Laureys et al., 2010) or 'post-coma unresponsiveness' (NHMRC, 2004) are more accurate descriptive terms for VS.

A person in a vegetative state may open their eyes, wake up and fall asleep at regular intervals and have basic reflexes, such as blinking when they're startled by a loud noise, or withdrawing their hand when it's squeezed hard. They're also able to regulate their heartbeat and breathing without assistance.

However, a person in a vegetative state doesn't show any meaningful responses, such as following an object with their eyes or responding to voices. They also show no signs of experiencing emotions nor of cognitive function.

VS/UWS patients' eyes might be in a relatively fixed position, may track moving objects (visual pursuit), or move in a completely unsynchronised manner. Sleep-wake cycles may resume or patients may appear to be in a state of chronic

wakefulness. They may grind their teeth, swallow, smile, shed tears, grunt, moan, or scream without any apparent external stimulus. VS/UWS patients do not respond to sound, hunger, or pain. Patients cannot obey verbal commands and lack local motor responses. Additionally VS/UWS patients cannot talk in comprehensible terms and may become noisy, restless, and hypermobile.

One of the most challenging tasks facing clinicians is that of differentiating VS/UWS from minimally conscious (MCS) states. These are both disorders of consciousness, so they can have similar presentations.

Whilst neuroimaging such as MRI is widely used in assessing brain damage and functional abilities, behavioural assessment has, until now, been the "gold standard" for detecting signs of consciousness and thereby for determining diagnosis.

If a person is in a vegetative state for a long time, it may be considered to be:

- a continuing vegetative state – when it's been longer than four weeks
- a permanent vegetative state – when it's been more than six months if caused by a non-traumatic brain injury, or more than 12 months if caused by a traumatic brain injury

If a person is diagnosed as being in a permanent vegetative state, recovery is extremely unlikely but not impossible.

Careful, ongoing assessment of the patient, using empirically validated assessment tools (eg the Coma Recovery Scale-Revised) is essential in order to evaluate and measure signs of progress, improvement or deterioration. Treatment is addressed at presenting symptoms and the patient's needs. VS/UWS patients require constant monitoring and assistance with feeding, hydration, hygiene, assisted movement and physical therapies (to help prevent ulcers and blood clots in the legs), and elimination of waste products.

Currently no treatment for VS/UWS exists that would satisfy the efficacy criteria of evidence-based medicine. Pharmacological methods, surgery, physical therapy, and various stimulation techniques have been suggested. Pharmacological therapy mainly uses activating substances such as tricyclic antidepressants or methylphenidate (Dolce et al. 2002). Surgical methods (eg. deep brain stimulation)

are used infrequently due to the invasiveness of the procedures. Stimulation techniques include sensory stimulation, sensory regulation, music and musicokinetic therapy, social-tactile interaction, etc.

Treatment can't ensure recovery from a state of impaired consciousness, however supportive treatment is used to give the best chance of natural improvement. This can involve;

- providing nutrition through a feeding tube
- making sure the person is moved regularly so they don't develop pressure ulcers
- gently exercising their joints to prevent them becoming tight
- keeping their skin clean
- managing their bowel and bladder – for example, using a catheter to drain the bladder
- keeping their teeth and mouth clean
- efforts should be made to establish functional communication and environmental interaction when possible. Offering opportunities for periods of meaningful activity – such as listening to music or watching television, being shown pictures or hearing family members talking

Sensory stimulation:

- visual – showing photos of friends and family, or a favourite film
- hearing – talking or playing a favourite song
- smell – putting flowers in the room or spraying a favourite perfume
- touch – holding their hand or stroking their skin with different fabrics

While not empirically validated, families have reported benefits from arousal regimes, such as those implemented by Dr Ted Freeman (eg Coma Arousal Therapy). This intensive therapy involves family members taking the patient through a regimen of controlled auditory, visual and physical stimulation for up to six hours a day every day.

Prognosis. Many patients emerge spontaneously from VS/UWS within a few weeks. Some people improve gradually, whereas others stay in a state of impaired consciousness for years. Many people never recover consciousness.

The chances of recovery depend on the extent of injury to the brain and age, with younger patients having a better chance of recovery than older patients. Generally, adults have about a 50 percent chance and children a 60 percent chance of recovering consciousness from VS/UWS within the first 6 months in the case of traumatic brain injury. For non-traumatic injuries such as strokes, the recovery rate falls within the first year. After this period the chances that VS/UWS patient will regain consciousness are very low and, of those patients who do recover consciousness, most experience significant disability. The longer a patient is in VS/UWS the more severe the resulting disabilities are likely to be.

Some patients who have entered a vegetative state go on to regain a degree of awareness (see Minimally Conscious State). The likelihood of significant functional improvement for VS/UWS patients diminishes over time. There are only isolated cases of people recovering consciousness after several years. The few people who do regain consciousness after this time often have severe disabilities caused by the damage to their brain.

7. The syndrome of the «Locked – In» patient

The locked-in syndrome (pseudocoma) describes patients who are awake and conscious but selectively deefferented, i.e., have no means of producing speech, limb or facial movements. Acute ventral pontine lesions are its most common cause. People with such brainstem lesions often remain comatose for some days or weeks, needing artificial respiration and then gradually wake up, but remaining paralyzed and voiceless, superficially resembling patients in a vegetative state or akinetic mutism. In acute locked-in syndrome (LIS), eye-coded communication and evaluation of cognitive and emotional functioning is very limited because vigilance is fluctuating and eye movements may be inconsistent, very small, and easily exhausted. It has been shown that more than half of the time it is the family and not the physician who first realized that the patient was aware. Distressingly, recent studies reported that the diagnosis of LIS on average takes over 2.5 months. In some cases it took 4-6 years before aware and sensitive patients, locked in an immobile body, were recognized as being conscious. Once a LIS patient becomes medically

stable, and given appropriate medical care, life expectancy increases to several decades. Even if the chances of good motor recovery are very limited, existing eye-controlled, computer-based communication technology currently allow the patient to control his environment, use a word processor coupled to a speech synthesizer, and access the worldwide net. Healthy individuals and medical professionals sometimes assume that the quality of life of an LIS patient is so poor that it is not worth living. On the contrary, chronic LIS patients typically self-report meaningful quality of life and their demand for euthanasia is surprisingly infrequent. Biased clinicians might provide less aggressive medical treatment and influence the family in inappropriate ways. It is important to stress that only the medically stabilized, informed LIS patient is competent to consent to or refuse life-sustaining treatment. Patients suffering from LIS should not be denied the right to die – and to die with dignity – but also, and more importantly, they should not be denied the right to live – and to live with dignity and the best possible revalidation, and pain and symptom management. In our opinion, there is an urgent need for a renewed ethical and medicolegal framework for our care of locked-in patients.

8. The syndrome of death of the brain

Brain stem death, determined by clinical examination with or without instrumental confirmation, should remain the mainstay of death definition. Legal rulings on brain death should be reviewed every 3 years to take into consideration new developments in medical knowledge and technology.

Brain stem death is a clinical syndrome defined by the absence of reflexes with pathways through the brain stem – the "stalk" of the brain, which connects the spinal cord to themid-brain, cerebellum and cerebral hemispheres – in a deeply comatose, ventilator-dependent patient. Identification of this state carries a very grave prognosis for survival; cessation of heartbeat often occurs within a few days although it may continue for weeks or even months if intensive support is maintained.

1982 articles in the British Medical Journal– and advanced a new definition of human death as the basis for equating this syndrome with the death of the person. The suggested new definition of death was the "irreversible loss of the capacity for

consciousness, combined with irreversible loss of the capacity to breathe". It was stated that the irreversible cessation of brain stem function will produce this state and "therefore brain stem death is equivalent to the death of the individual".

In the UK, the formal rules for the diagnosis of brain stem death have undergone only minor modifications since they were first published in 1976. The most recent revision of the UK's Department of Health Code of Practice governing use of that procedure for the diagnosis of death reaffirms the preconditions for its consideration. These are:

• There should be no doubt that the patient's condition – deeply comatose, unresponsive and requiring artificial ventilation – is due to irreversible brain damage of known aetiology.

• There should be no evidence that this state is due to depressant drugs.

• Primary hypothermia as the cause of unconsciousness must have been excluded, and

• Potentially reversible circulatory, metabolic and endocrine disturbances likewise.

• Potentially reversible causes of apnoea (dependence on the ventilator), such as muscle relaxants and cervical cord injury, must be excluded.

With these pre-conditions satisfied, the definitive criteria are:

• Fixed pupils which do not respond to sharp changes in the intensity of incident light.

• No corneal reflex.

• Absent oculovestibular reflexes – no eye movements following the slow injection of at least 50ml of ice-cold water into each ear in turn (the caloric reflex test).

• No response to supraorbital pressure.

• No cough reflex to bronchial stimulation or gagging response to pharyngeal stimulation.

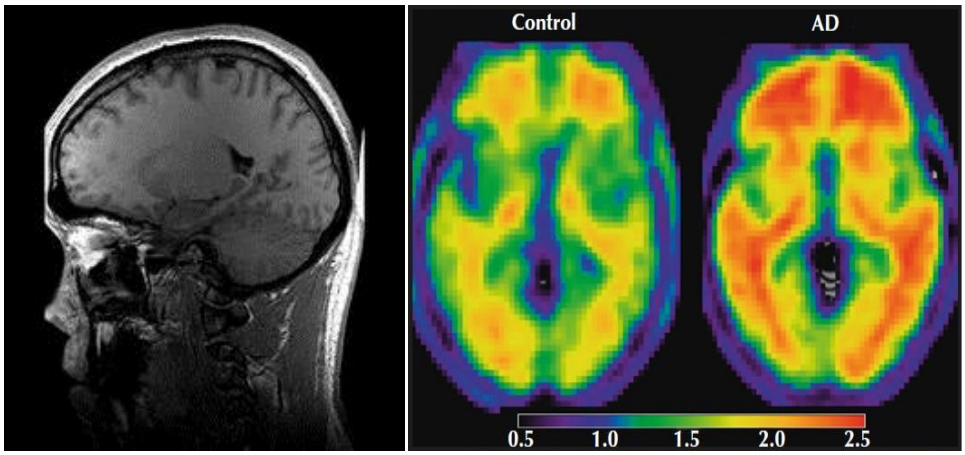
• No observed respiratory effort in response to disconnection of the ventilator for long enough (typically 5 minutes) to ensure elevation of the arterial partial pressure ofcarbon dioxide to at least 6.0 kPa (6.5 kPa in patients with chronic carbon

dioxide retention). Adequate oxygenation is ensured by pre-oxygenation and diffusion oxygenation during the disconnection (so the brain stem respiratory centre is not challenged by the ultimate, anoxic, drive stimulus). This test - the apnoea test - is dangerous – and may prove lethal.

Two doctors, of specified status and experience, are required to act together to diagnose death on these criteria and the tests must be repeated after "a short period of time ... to allow return of the patient's arterial blood gases and baseline parameters to the pre-test state". These criteria for the diagnosis of death are not applicable to infants below the age of two months.

9. Methods for studying the functional dignostic of the cortex of the large hemispheres

9.1. Methods of neurovisualization



PET imaging with florbetapir reveals the presence of amyloid plaque deposits in the brain of an individual with a clinical diagnosis of Alzheimer disease (shades of red) compared to a cognitively normal older adult with little to no evidence of amyloid (lighter red and yellow).

1. Magnetic resonance imaging (MRI)
2. Positronemission tomography (PET)

1. Magnetic resonance imaging (MRI)

Magnetic resonance imaging (MRI) is a medical imaging technique used in radiology to form pictures of the anatomy and the physiological processes of the body. MRI scanners use strong magnetic fields, magnetic field gradients, and radio waves to generate images of the organs in the body. MRI does not involve X-rays or the use of ionizing radiation, which distinguishes it from CT and PET scans. MRI is a medical application of nuclear magnetic resonance (NMR) which can also be used for imaging in other NMR applications, such as NMR spectroscopy.

MRI is widely used in hospitals and clinics for medical diagnosis, staging and follow-up of disease. Compared to CT, MRI provides better contrast in images of soft-tissues, e.g. in the brain or abdomen. However, it may be perceived as less comfortable by patients, due to the usually longer and louder measurements with the subject in a long, confining tube. Additionally, implants and other non-removable metal in the body can pose a risk and may exclude some patients from undergoing an MRI examination safely.

MRI was originally called NMRI (nuclear magnetic resonance imaging), but "nuclear" was dropped to avoid negative associations. Certain atomic nuclei are able to absorb radio frequency energy when placed in an external magnetic field; the resultant evolving spin polarization can induce a RF signal in a radio frequency coil and thereby be detected. In clinical and research MRI, hydrogen atoms are most often used to generate a macroscopic polarization that is detected by antennae close to the subject being examined. Hydrogen atoms are naturally abundant in humans and other biological organisms, particularly in water and fat. For this reason, most MRI scans essentially map the location of water and fat in the body. Pulses of radio waves excite the nuclear spin energy transition, and magnetic field gradients localize the polarization in space. By varying the parameters of the pulse sequence, different contrasts may be generated between tissues based on the relaxation properties of the hydrogen atoms therein.

Since its development in the 1970s and 1980s, MRI has proven to be a versatile imaging technique. While MRI is most prominently used in diagnostic medicine and biomedical research, it also may be used to form images of non-living objects, such as mummies. Diffusion MRI and Functional MRI extends the utility of MRI to capture neuronal tracts and blood flow respectively in the nervous system, in addition to detailed spatial images. The sustained increase in demand for MRI within health systems has led to concerns about cost effectiveness and over diagnosis.

2. Positronemission tomography (PET)

This is a method of imaging the brain, the basis of which is the use of radionuclides, which makes it possible to assess the metabolism of different parts of the brain tissue. The technique involves intravenous administration of a substance

with labeled radioactive isotopes, which allows you to determine the level of oxygen and glucose, as well as visualize intracerebral synaptic activity, the spread in the space of receptors.

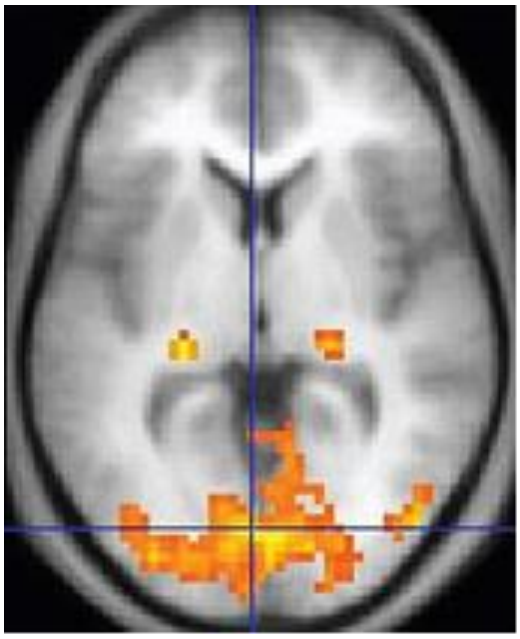
3. Functional magnetic resonance imaging or functional MRI (fMRI). measures brain activity by detecting changes associated with blood flow. This technique relies on the fact that cerebral blood flow and neuronal activation are coupled. When an area of the brain is in use, blood flow to that region also increases.

Primary form of fMRI uses the blood-oxygen-level dependent (BOLD) contrast, discovered by Seiji Ogawa in 1990. This is a type of specialized brain and body scan used to map neural activity in the brain or spinal cord of humans or other animals by imaging the change in blood flow (hemodynamic response) related to energy use by brain cells. Since the early 1990s, fMRI has come to dominate brain mapping research because it does not involve the use of injections, surgery, the ingestion of substances, or exposure to ionizing radiation. This measure is frequently corrupted by noise from various sources; hence, statistical procedures are used to extract the underlying signal. The resulting brain activation can be graphically represented by color-coding the strength of activation across the brain or the specific region studied. The technique can localize activity to within millimeters but, using standard techniques, no better than within a window of a few seconds. Other methods of obtaining contrast are arterial spin labeling and diffusion MRI. Diffusion MRI is similar to BOLD fMRI but provides contrast based on the magnitude of diffusion of water molecules in the brain.

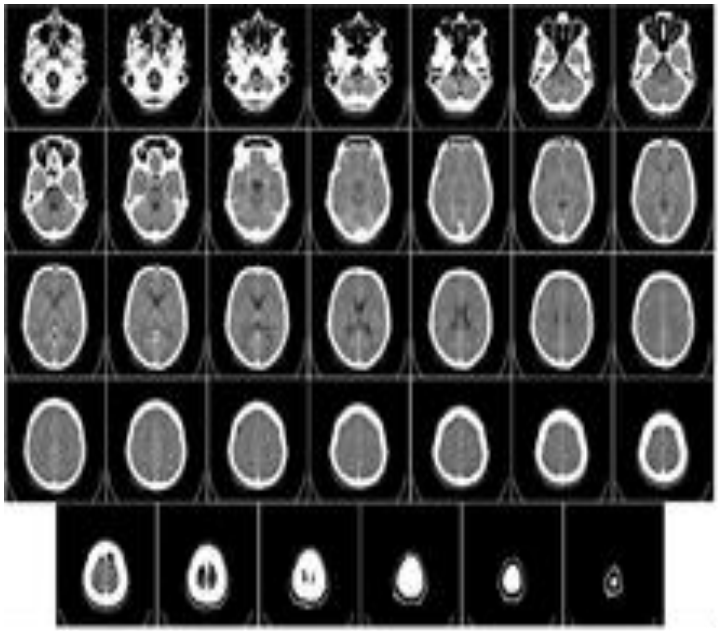
In addition to detecting BOLD responses from activity due to tasks or stimuli, fMRI can measure resting state, or negative-task state, which shows the subjects' baseline BOLD variance. Since about 1998 studies have shown the existence and properties of the default mode network, a functionally connected neural network of apparent resting brain states.

fMRI is used in research, and to a lesser extent, in clinical work. It can complement other measures of brain physiology such as electroencephalography (EEG), and near-infrared spectroscopy (NIRS). Newer methods which improve both

spatial and time resolution are being researched, and these largely use biomarkers other than the BOLD signal. Some companies have developed commercial products such as lie detectors based on fMRI techniques, but the research is not believed to be developed enough for widespread commercial use.



3. Functional magnetic resonance imaging (fMRI)



4. Computed tomography (CT)

4. Computed tomography (CT)

A CT scan or computed tomography scan (formerly known as computed axial tomography or CAT scan) is a medical imaging technique used in radiology to obtain detailed internal images of the body noninvasively for diagnostic purposes. The personnel that perform CT scans are called radiographers or radiology technologists. CT scanners use a rotating X-ray tube and a row of detectors placed in the gantry to measure X-ray attenuations by different tissues inside the body. The multiple X-ray measurements taken from different angles are then processed on a computer using reconstruction algorithms to produce tomographic (cross-sectional) images (virtual "slices") of a body. The use of ionizing radiation sometimes restricts its use owing to its adverse effects. However, CT can be used in patients with metallic implants or pacemakers, for whom MRI is contraindicated.

Since its development in the 1970s, CT has proven to be a versatile imaging technique. While CT is most prominently used in diagnostic medicine, it also may be used to form images of non-living objects. The 1979 Nobel Prize in Physiology or Medicine was awarded jointly to South African-American physicist Allan M. Cormack and British electrical engineer Godfrey N. Hounsfield "for the development of computer-assisted tomography".

CT scanning of the head is typically used to detect infarction (stroke), tumors, calcifications, haemorrhage, and bone trauma. Of the above, hypodense (dark) structures can indicate edema and infarction, hyperdense (bright) structures indicate calcifications and haemorrhage and bone trauma can be seen as disjunction in bone windows. Tumors can be detected by the swelling and anatomical distortion they cause, or by surrounding edema. CT scanning of the head is also used in CT-guided stereotactic surgery and radiosurgery for treatment of intracranial tumors, arteriovenous malformations, and other surgically treatable conditions using a device known as the N-localizer.

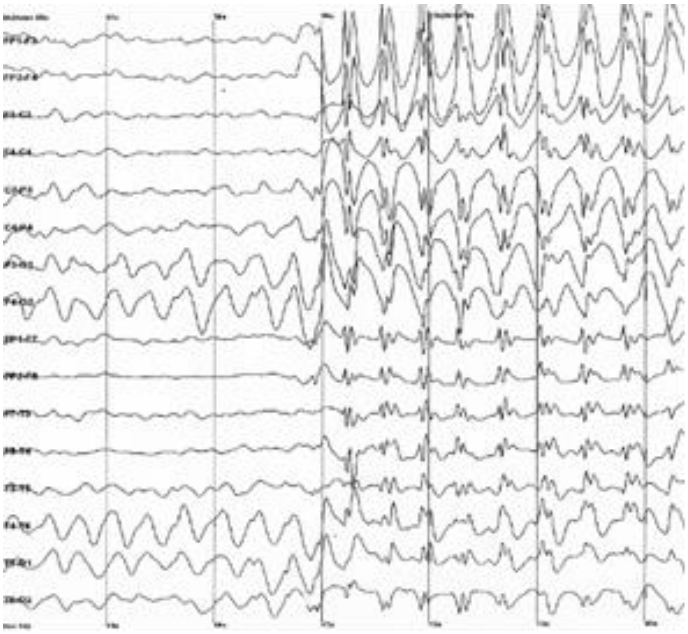
9.2. Electrophysiological methods

1. Electroencephalography (EEG) is a method to record an electrogram of the electrical activity on the scalp that has been shown to represent the macroscopic activity of the surface layer of the brain underneath. It is typically non-invasive, with the electrodes placed along the scalp. Electrocorticography, involving invasive electrodes, is sometimes called intracranial EEG. EEG measures voltage fluctuations resulting from ionic current within the neurons of the brain. Clinically, EEG refers to the recording of the brain's spontaneous electrical activity over a period of time, as recorded from multiple electrodes placed on the scalp.[1] Diagnostic applications generally focus either on event-related potentials or on the spectral content of EEG. The former investigates potential fluctuations time locked to an event, such as 'stimulus onset' or 'button press'. The latter analyses the type of neural oscillations (popularly called "brain waves") that can be observed in EEG signals in the frequency domain.

Is most often used to diagnose epilepsy, which causes abnormalities in EEG readings. It is also used to diagnose sleep disorders, depth of anesthesia, coma,

encephalopathies, and brain death. EEG used to be a first-line method of diagnosis for tumors, stroke and other focal brain disorders, but this use has decreased with the advent of high-resolution anatomical imaging techniques such as magnetic resonance imaging (MRI) and computed tomography (CT). Despite limited spatial resolution, EEG continues to be a valuable tool for research and diagnosis. It is one of the few mobile techniques available and offers millisecond-range temporal resolution which is not possible with CT, PET or MRI.

Derivatives of the EEG technique include evoked potentials (EP), which involves averaging the EEG activity time-locked to the presentation of a stimulus of some sort (visual, somatosensory, or auditory). Event-related potentials (ERPs) refer to averaged EEG responses that are time-locked to more complex processing of stimuli; this technique is used in cognitive science, cognitive psychology, and psychophysiological research.



1. Electroencephalography (EEG) 2.Magnetoencephalography

2. Magnetoencephalography

This study allows you to measure the magnetic fields of the cerebral cortex. Not to be confused with an electroencephalogram (EEG), which shows changes in electrical potentials. In the study of the functional state of the brain, it is preferable to perform MEG, since the attenuation of electrical potentials during the passage through bone tissue is more pronounced than magnetic potentials.

10. Materials for self-control

10.1. The questions for self-control work

1. The methods of investigation of individual parts cortex and symptoms of injury, function of individual parts of the cortex.

2. Differential diagnostic of defect of different part of thre brain cortex.

10.2. Typical tasks

1. A 60-year-old right-handed man acutely develops a loss of understanding of the speech, including and writing, control after own speech, speech of patient as “salad from words". What is the type disorder of the speech? What lobe and zone is affected?

2. A 50-year-old right-handed women acutely develops motor aphasia and agraphia. What is the type disorder? What lobe and zone is affected? What of diseases can cause such kind of pathology?

3. Clear Cerebrospinal fluid, pressure of 2, 45 kPa (250 mm of water. In. Reaction Pandy ++, protein - 0.96 g/l, cytosis 786x10/6 in 1 liter (dominated by lymphocytes). If any pathological processes can be observed such changes cerebrospinal fluid? What additional research is needed to clarify the etiology of the process? What autotopoagnoziya, psevdomeliya, anosognosia? Defeat of the Department of the cerebral hemispheres, they may be due to?

4. Cerebrospinal fluid is clear, colorless, pressure – 2.16 kPa (220 mm of water), The reaction of Pandy ++ Protein – 0.87 g / l, cytosis – 830x 10/6 in 1 liter (lymphocytes), glucose – 3.05 mmol / L (55 mg%). On standing, the cerebrospinal fluid during the day fibrin film is not dropped. Wasserman negative. Rate changes in the composition of the liquid and let us know what your guess as to the nature of the disease.

5. Central hemiplegia, central facial paralysis of the hypoglossal nerve, and the motor aphasia. The patient – a right-hander. Where localized lesion? Why do you think so?

6. Clear Cerebrospinal fluid, pressure of 2, 45 kPa (250 mm of water. In. Reaction Pandy ++, protein - 0.96 g/l, cytosis 786x10/6 in 1 liter (dominated by lymphocytes). If any pathological processes can be observed such changes

cerebrospinal fluid? What additional research is needed to clarify the etiology of the process? What autotopoagnoziya, psevdomeliya, anosognosia? Defeat of the Department of the cerebral hemispheres, they may be due to?

7. Cerebrospinal fluid is clear, colorless, pressure – 2.16 kPa (220 mm of water), The reaction of Pandy ++ Protein – 0.87 g / l, cytosis – 830x 10/6 in 1 liter (lymphocytes), glucose – 3.05 mmol / L (55 mg%). On standing, the cerebrospinal fluid during the day fibrin film is not dropped. Wasserman negative. Rate changes in the composition of the liquid and let us know what your guess as to the nature of the disease.

8. Central hemiplegia, central facial paralysis of the hypoglossal nerve, and the motor aphasia. The patient – a right-hander. Where localized lesion? Why do you think so?

9. The attacks of Jackson epilepsy begin with a convulsive turn the head and eye in an left side and accomplish paresthesia of right extremities. Where is zone of lesion?

10. The a right-hander patient has total aphasia(sensory and motor), right hemiparesis. Where is zone of lesion?

10. In right-handed people the patient has total aphasia (sensory and motor), right hemiparesis. Where is the affected area?

11. Establish indications and contraindications for lumbar puncture.

12. Perform lumbar puncture with cerebrospinal fluid tests. To give a clinical assessment of the analysis of cerebrospinal fluid.

13. Taking into account the data of clinical examination and with the help of the obtained data of cerebrospinal fluid diagnostics to make a differential diagnosis of a patient with emergency pathology and to establish a clinical diagnosis.

10.3. Tests for students you can see in the addition

1. In localization of the lesion focus in the area of the inferior frontal gyrus of the dominant hemisphere in the patient is observed;

a. the sensory aphasia

b. the motor aphasia

c. the semantic aphasia

d. the amnestic aphasia

e. the scanning speech

2. The main ingibitory mediator isa.

a. acetylcholine

b. GABA

c. norepinephrine

d. epinephrine

e. glutamate

3. The patient is determined the right-side hemihypesthesia, astereognosis, apraxia, alexia, and acalculia. Where is the lesion center located?

a. the frontal lobe of the left cerebral hemisphere

b. the temporal lobe of the left cerebral hemisphere

c. the internal capsule of the left cerebral hemisphere

d. the parietal lobe of the left cerebral hemisphere

e. the parietal lobe of the right cerebral hemisphere

4. What kind of crisis is evidenced by the sudden malaise, headache, general weakness, unpleasant feeling in abdomen, lowering of BP, bradycardia, and flushing of the face?

a. the vagoinsular crisis

b. the sympathoadrenal crisis

c. the mixed crisis

d. the syncopal state

e. the phasic vegetative crisis

5. The disturbance of performing the complex usual purposeful movements means

a. agnosia

b. apraxia

c. anosognosia

d. autotopagnosia

e. astereognosis

6. The sudden headache, fear of death, cold extremities, increased BP to 155|100 mm Hg art., heart rate 102 per minute, and the pale face testify to the development of

a. the vagoinsular crisis

b. the sympathoadrenal crisis

c. the mixed crisis

d. the syncopal state

e. the phasic vegetative crisis

7. The patient's articulation is lost. What term designates this syndrome?

a. dystonia

b. dysarthria

c. the sensitive aphasia

d. the motor aphasia

e. alalia

8. For the assesment of wich function of the autonomic nervous system is the clinoorthostatic test used?

a. the vegetative ensuring of the physical activity

b. the vegetative reactivity

c. the vegetative tonus

d. the trophotropic function

e. all answers are right

9. What term designates the syndrome of disturbance of the body scheme?

a. autotopagnosia

b. anosognosia

c. apraxia

d. astereognosis

10. Periodically the patient has the twilight conditions of consciousness, the gustatory and olfactory hallucinations, and the left-side quadrant hemianopsia. Specify the process localization

a. the frontal lobe of the right cerebral hemisphere

b. the occipital lobe of the left cerebral hemisphere

c. the temporal lobe of the right cerebral hemisphere

d. the temporal lobe of the left cerebral hemisphere

e. the visual radiance

Answers:

1. B 2.B 3.D 4.A 5.B 6.B 7. B 8. A 9.A 10.C

INFORMATION SOURCES

1. Color Atlas of Neurology Reinhard Rohkamm, Germany Thieme Stuttgart New York – 2th ed. – 2004. P. 6–10, 24–26.

2. Atlas of Neuroanatomy and Neurophysiology Selections from the Netter Collection of Medical Illustrations Copyright ©2002 Icon Custom Communications. P. 52, 61–62, 70.

3. Essential Neurology Iain Wilkinson, Graham Lennox. – 4th ed. P. 55–65.

4. Atlas of Neuroanatomy and Neurophysiology, Selections from the Netter Collection of Medical Illustrations by Frank H. Netter, MD John A. Craig, MD James Perkins, MS, MFA Text by John T. Hansen, PhD Bruce M. Koeppen, MD, PhD.– 2002, P. 71.

5. Hryhorova I.A., Sokolova L.I., Herasymchuk R.D. et all. Neurology: textbook – Kyiv: AUS Medicine Publishing, 2017. – 624 p.

6. Mishchenko T.S., Mishchenko V.M., Kabachna I.B. Vascular disease of the brain. Kharkiv «Tytul», 2022. – 50p. Laureys S, Pellas F, Van Eeckhout P, Ghorbel S, Schnakers C, Perrin F, Berré J, Faymonville ME, Pantke KH, Damas F, Lamy M, Moonen G, Goldman S.The locked-in syndrome: what is it like to be conscious but paralyzed and voiceless? Prog Brain Res. 2005; 150: 495–511.

7. Vinychuk S.M., Dubenko E.G., Macheret Ye.L. et all. Neurological Diseases. Textbook. Kyiv “Zdorovya”, 2001. – 696 p.

8. Conference of Medical Royal Colleges and their Faculties in the UK. BMJ 1976; 2:1187–Conference of Medical Royal Colleges and their Faculties in the UK. BMJ 1979; 1:332.

9. Hoffmann M. Assembly of the Executive Mind Evolutionary Insights and a Paradigm for Brain Health. Information on this title: www.cambridge.org/9781108456005. DOI: 10.1017/9781108589246. © Cambridge University Press 2019. First published 2019. ISBN 978-1-108-45600-5 Paperback.

10. Tatum, William O., IV, editor. Title: Atlas of artifacts of clinical neurophysiology / editor, William O. Tatum, IV. Description: New York : Demos, 2018. | Includes bibliographical references and index. Identifiers: LCCN 2018028988| ISBN 9780826169341 (alk. paper) | ISBN 9780826169358 (ebook). Classification: LCC RC387.5 | NLM WL 141 | DDC 616.8/0475—dc23 LC record available at https://lccn.loc.gov/2018028988.

11. William H.Olson, Roger A. Brumback, Gascon G., Ieer V. Hand-book of symptom-oriented Neurology. Year book medical publisher, inc. – 1981. P. 185–188, 205–207, 358–360.

12. Laureys S, Pellas F, Van Eeckhout P, Ghorbel S, Schnakers C, Perrin F, Berré J, Faymonville ME, Pantke KH, Damas F, Lamy M,Moonen G, Goldman S.The locked-in syndrome: what is it like to be conscious but paralyzed and voiceless. Prog Brain Res. 2005; 150: 495–511.

13. Kasule O.H. Brain death: criteria, signs, and tests. Journal of Taibah University of Medical Science. Volume 8, Issue 1, April 2013, Pages 1–6.

14. Moini J., LoGalbo A., Ahangari R. Book: Foundations of the Mind, Brain, and Behavioral Relationships. Chapter 13 – Cognitive functions. Publisher: Elsevier, 2024, PP. 211–228. https://doi.org/10.1016/B978-0-323-95975-9.00002-0

15. Oxford American handbook of neurology / edited by Sid Gilman ; with Hadi Manji ... [et al.]. p. ; cm.—(Oxford American handbooks). Adapted from: Oxford handbook of neurology / Hadi Manji ... [et al.]. 2007. Includes bibliographical references and index. 2010. ISBN 978-0-19-536979-3.

16. Topical issues of neurology: interdisciplinary relationships [Text]: materials of the conf. / ed. V. A. Groisman; SamSMU, Department of Health of the City Hall Togliatti, MUZ GKB № 1. – Togliatti: Format, 2007. – 178 p. : ill.

17. Jamie Ward. The Student's Guide to Cognitive Neuroscience. 4th Edition. 2020. ISBN: 978-1-351-03518-7 (ebk), ISBN: 978-1-138-49052-9 (hbk), ISBN: 978-1-138-49054-3 (pbk).

18. Brodal, Per. The central nervous system : structure and function / Per Brodal. — 4th ed. p. ; cm. Includes bibliographical references and index. 2010. ISBN-13: 978-0-19-538115-3 (alk. paper) ISBN-10: 0-19-538115-7 (alk. paper).

19. Christopher H. Hawkes, Richard L. Doty. Smell and Taste Disorders. Cambridge University Press. 2017. ISBN 978-0-521-13062-2 Paperback.

20. Atlas of Neuroanatomy and Neurophysiology. Selections from the Netter Collection of Medical Illustrations. Copyright ©2004 Icon Custom Communications.

21. Bear, Mark F., author. Neuroscience : exploring the brain / Mark F. Bear, Barry W. Connors, Michael A. Paradiso. — Fourth edition. p. ; cm. Includes bibliographical references and index. 2016. ISBN 978-0-7817-7817-6 (hardback : alk. paper)

22. Neurobiology of disease / edited by Michael V. Johnston, Harold P. Adams Jr., Ali Fatemi. — 2nd edition. p. ; cm. Preceded by Neurobiology of disease / edited by Sid Gilman. 2007.

23. Barkhof F., Jäger H.R., Thurnher M.M., Rovira A. Clinical Neuroradiology. The ESNR Textbook. ISBN 978-3-319-68535-9. ISBN 978-3-319-68536-6 (eBook). ISBN 978-3-319-68537-3 (print and electronic bundle). https://doi.org/10.1007/978-3-319-68536-6 .© Springer Nature Switzerland AG 2019.

24. Misulis, Karl E., author. | Head, Thomas C. (Thomas Channing), 1963-author. | Netter, Frank H. (Frank Henry), 1906-1991, illustrator. Title: Netter's concise neurology/Karl E. Misulis, Thomas C. Head ; illustrations by Frank H. Netter ; contributing illustrators, John A. Craig, Carlos A. G. Machado. Other titles: Concise neurology Description: Updated edition. | Philadelphia, PA : Elsevier, [2017] | Includes index. Identifiers: LCCN 2016017028 | ISBN 9780323482547 (pbk. : alk. paper). Classification: LCC RC355 | NLM WL 39 | DDC 616.8—dc23 LC record available at https://lccn.loc.gov/2016017028.

25. Felten, David L. Netter’s atlas of neuroscience / David L. Felten, Anil N. Shetty ; illustrations by Frank H. Netter ; contributing illustrators, Carlos A.G. Machado, James A. Perkins, John A. Craig. — 2nd ed. p. ; cm. Includes index. 2010. Rev. ed. of: Netter’s atlas of human neuroscience / David L. Felten, Ralph Jyzefowicz. 1st ed. c2003. ISBN 978-1-4160-5418-4.

26. Rubin, Michael, 1957 - author. | Safdieh, Joseph E., author. | Netter, Frank H. (Frank Henry), 1906-1991, illustrator. Title: Netter's concise neuroanatomy/Michael Rubin, Joseph E. Safdieh ; illustrations by Frank H. Netter ; contributing illustrators John A. Craig, Carlos A.G. Machado, James A. Perkins. Description: Philadelphia, PA : Elsevier, [2017] | Includes index. Identifiers: LCCN 2016016617 | ISBN 9780323480918 (pbk. : alk. paper). Classification: LCC QP361 | NLM WL 17 | DDC 612.8—dc23 LC record available at https://lccn.loc.gov/2016016617.

27. Mason, Peggy (Neurobiologist), author. Title: Medical neurobiology / by Peggy Mason. Description: Second edition. | New York, NY : Oxford University Press, [2017] | Includes bibliographical references and index. Identifiers: LCCN 2016028872 | ISBN 9780190237493 (alk. paper) | eISBN 9780190237516. Classification: LCC QP355.2 | NLM WL 102 | DDC 612.8--dc23 LC record available at https://lccn.loc.gov/2016028872.

28. R. Nieuwenhuys, J. Voogd, C. van Huijzen. The Human Central Nervous System. Fourth Edition. ISBN 978-3-540-34684-5. Springer-Verlag Berlin Heidelberg New York. ISBN 3-540-13441-7. Auflage Springer-Verlag Berlin Heidelberg New York. Springer Berlin Heidelberg 1978, 1981, 1988, 2008.

29. Elliott L. Mancall, David G. Brock. GRAY'S CLINICAL NEUROANATOMY: THE ANATOMIC BASIS FOR CLINICAL NEUROSCIENCE. ISBN: 978-1-4160-4705-6. Copyright © 2011 by Saunders, an imprint of Elsevier Inc.

30. Mattle H., Mumenthaler M., Gralla J., Schroth G.. Fundamentals of Neurology. An Illustrated Guide 2nd Revised and Updated Edition. ISBN 978-3-13-136452-4.

31. Franklin R. Amthor, PhD, Anne B. Theibert, PhD, David G. Standaert, MD, PhD, John N. Whitaker Erik D. Roberson, MD, PhD, Rebecca Gale Endowed. Essentials of Modern Neuroscience. © 2020 by McGraw Hill. ISBN: 978-1-25-986104-8. MHID: 1-25-986104-X. The material in this eBook also appears in the print version of this title: ISBN: 978-0-07-184905-0, MHID: 0-07-184905-X.

32. Bianchi Lynne. Developmental neurobiology / Lynne M. Bianchi. Description: New York, NY: Garland Science, Taylor & Francis Group, LLC, 2018. Identifiers: LCCN 2017034851 | ISBN 9780815344827. Classification: LCC QP363.5 .B563 2018 | DDC 612.6/4018--dc23. LC record available at

https://lccn.loc.gov/2017034851 . Published by Garland Science, Taylor & Francis Group, LLC.

33. DeMyer's. The Neurologic Examination: A Programmed Text, Seventh Edition. Copyright © 2017 by McGraw-Hill Education. Previous editions copyright © 2011, 2004, 1994 by The McGraw-Hill Companies, Inc. ISBN 978-0-07-184161-0. MHID 0-07-184161-X.

34. Campbell, William W., Jr. (William Wesley), author. | Barohn, Richard J., author. Title: DeJong's the neurologic examination / William W. Campbell, Richard J. Barohn. Other titles: Neurologic examination Description: Eighth edition. Philadelphia : Wolters Kluwer, [2020] | Includes bibliographical references and index. Identifiers: LCCN 2018059855 | ISBN 9781496386168 (hardback). Classification: LCC RC348 | NLM WL 141 | DDC 616.8/0475—dc23 LC record available at https://lccn.loc.gov/2018059855.

35. Radtke-Schuller S. Cyto- and Myeloarchitectural Brain Atlas of the Ferret (Mustela putorius) in MRI Aided Stereotaxic Coordinates. ISBN 978-3-319-76625-6, ISBN 978-3-319-76626-3 (eBook). https://doi.org/10.1007/978-3-319-76626-3. © Springer International Publishing AG, part of Springer Nature 2018.

36. Current Diagnosis & Treatment Neurology. Second Edition. Edited by John C.M. Brust, MD. New York, New YorkCopyright © 2012 by The McGraw-Hill Companies, Inc. ISBN: 978-0-07-171797-7. MHID: 0-07-171797-8. The material in this eBook also appears in the print version of this title: ISBN: 978-0-07-170118-1, MHID: 0-07-170118-4.

37. Clinical signs in neurology : a compendium / William W. Campbell. p. ; cm. Includes bibliographical references and index. ISBN 978-1-4511-9445-6. eISBN 978-1-4963-2814-4.

38. Jos J. Eggermont. BRAIN OSCILLATIONS, SYNCHRONY, AND PLASTICITY. Basic Principles and Application to Auditory-Related Disorders. Academic Press is an imprint of Elsevier, Inc. ISBN 978-0-12-819818-6.

Електронне навчальне видання комбінованого використання
Можна використовувати в локальному та мережному режимі

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ЛОКАЛІЗАЦІЯ ФУНКЦІЙ У КОРІ ГОЛОВНОГО МОЗКУ. СИНДРОМИ УРАЖЕННЯ

Методичні рекомендації
для здобувачів вищої освіти 4-го року навчання
з дисципліни «Neurology»

(Англ. мовою)

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