Ryazan State Medical University

Faculty of neurology and neurosurgery

O.V. EVDOKIMOVA, V.A. ZHADNOV, R.A. ZORIN

CLINICAL ANALYSIS OF THE NEUROLOGIC SYNDROMES

The educational and methodical manual for students
Of the stomatologic faculty
«Neurology»

Ryazan, 2022

UDK 616.8-07(075.1)
BBC 56.12
E 155

Reviewers:
S.N. Trushin, MD, the head of the department of faculty surgery, anesthesiology;
E.A. Nazarov, MD, the head of the department of orthopedy, traumatology and sport medicine;
L.F. Eltcova, the head of the department of foreign languages.

Authors:
O.V. Evdokimova, MD, the assistant professor of the department of neurology and neurosurgery;
V.A. Zhadnov, MD; the head of the department of neurology and neurosurgery;
R.A. Zorin, MD, the assistant professor of the department of neurology and neurosurgery

E 155 Evdokimova O.V. Clinical analyses of the neurologic syndromes: The educational and methodical manual for students of stomatologic faculty «Neurology» / Auth.: O.V. Evdokimova, V.A. Zhadnov, R.A. Zorin; RyazSMU. – Ryazan: DTS&OP, 2022. – 122 p.

The educational and methodical manual presents theoretical data of various neurological syndromes, as well as questions for preparing for classes, clinical tasks on the syndrome of neurological disorders, and a scheme for a clinical neurological examination of a patient in neurology. "Clinical analysis of neurological syndromes" is an addition to the basic educational literature for students of the stomatologic faculty.

UDK 616.8-07(075.1)
BBC 56.12

Федеральное государственное бюджетное образовательное
учреждение высшего образования
«Рязанский государственный медицинский университет
имени академика И.П. Павлова»
Министерства здравоохранения Российской Федерации

Кафедра неврологии и нейрохирургии

О.В. Евдокимова, В.А. Жаднов, Р.А. Зорин

Клинический анализ неврологических синдромов

Учебно-методическое пособие
для студентов стоматологического факультета
по дисциплине «Неврология»

Рязань, 2022

УДК 616.8-07(075.1)
ББК 56.12
Е 155

Рецензенты:
С.Н. Трушин, д.м.н., проф., заведующий кафедрой факультетской хирургии с курсом анестезиологии и реаниматологии;
Е.А. Назаров, д.м.н., профессор, заведующий кафедрой ортопедии, травматологии и спортивной медицины;
Л.Ф. Ельцова, к.филол.н., заведующая кафедрой иностранных языков

Авторы:
О.В. Евдокимова, к.м.н., доцент кафедры неврологии и нейрохирургии***;***
В.А. Жаднов, д.м.н., заведующий кафедрой неврологии и нейрохирургии;
Р.А. Зорин, д.м.н., доцент кафедры неврологии и нейрохирургии;

Е 155 Евдокимова О.В. Клинический анализ неврологических синдромов: Учебно-методическое пособие для студентов стоматологического факультета по дисциплине «Неврология» / О.В. Евдокимова, В.А. Жаднов, Р.А. Зорин; ФГБОУ ВО РязГМУ Минздрава России. – Рязань: ОТСиОП, 2022. – 122 с.

В учебно-методическом пособии представлены теоретические данные по различным неврологическим синдромам, а также вопросы для подготовки к занятиям, клинические задачи по синдромологии неврологических расстройств, схема клинического неврологического осмотра больного в неврологии. «Клинический анализ неврологических синдромов» являются дополнением к основной учебной литературе для студентов стоматологического факультета.

УДК 616.8-07(075.1)
ББК 56.12

Feature of diagnostics in each specific area of medicine expresses attachment of a specialty to organic laws of knowledge and logic constructions. Feature of clinical neurology will consist in use of fundamental logic and gnosiological concepts, such as concept, judgment, conclusion, use of classical laws of formal logic. The obligatory stage in clinical education is a mastering by laws of clinical logic, skill to allocate logic clinical structures, such as attributes, symptoms, tests, syndromes, topical diagnosis, criteria of diagnostics. To these purposes there correspond the problems submitted in the manual and examples. Training to the decision of clinical tasks provides allocation of symptoms, attributes, tests in clinical aspect, the formulation of neurologic syndromes, definition and formulation of the topical diagnosis, an establishment of clinical criteria of the diagnosis and the formulation of the preliminary clinical diagnosis. In the third part of the manual clinical trouble-shooting tests which purpose is except for higher listed tasks of the differential diagnostics are submitted.

CONTENT

GENERAL ANALISIS OF THE NEUROLOGIC DISORDERS

Study of neurology begins by learning methods of clinical diagnosis.

Value of study of neurology for clinical education of the modern doctor will consist in the following.

1. The fundamental concepts of clinical neurology are basis of dialogue between different specialists.

2. The classical neurology has the special logic. It allows to transform diagnosis into performance of fixed algorithm.

Ultimate goal of diagnostic is build-up of the clinical diagnosis. The clinical diagnosis is systemic exposition of the causes, mechanisms, clinical forms, localizations and character of processes, clinical manifestations and flow of disease, its complications on a background of concomitant diseases and the special states.

The clinical diagnosis is positioned during process of diagnosis. Process of diagnosis begins with definition of initial attributes of disease at immediate contact with the patient. There are symptoms, signs and tests.

The symptom is subjectively estimated infringement which the patient brings to the doctor as the complaint. The doctor transforms value judgment of health by the patient into an objective sign of disease. For example, the pain is subjective sensation. The pain estimates on several standard parameters. Features of pain are localization, duration, time of originating and petering, the cause of appearance and relief, the diffusion, facilitating factors. At the strict versatile description the pain becomes the objective attribute of disease.

Second amounting initial examinations of patients is termed an sign. The sign is a external figure of disorder of functions appeared by the doctor. For example, sign of pain at patients show as a pain facial expression and limitative position.

The third amounting external patterns of the disease is a test. The test is the attribute detected at the special positions of the patient or artificial change of his state. For example, at adoption the patient a vertical position blindly it is possible to see infringement of coordination. This test is termed the Romberg's test.

The initial disorders estimated at bed of the patient allow to establish a clinical syndromes. The clinic-neurologic set of symptoms is

not simple concurrence of symptoms and signs, but the plurality of infringements bounded by intrinsic mechanisms. Clinic-neurologic syndrome defines gravity of patients, dynamics of disease, localization of disease process. When on the establishment of clinic neurologic set of symptoms it is located lesions of nervous system, it is termed "the topical diagnosis".

The topical diagnosis defines where pathological process is in nervous system, how long it’s extent, interaction with environmental tissues of nervous system is posed.

On the basis of the topical diagnosis the provisional clinical diagnosis which includes the basic clinical form of disease and localization of a lesion is set. The following stage of clinical diagnosis the differential diagnosis. Carrying out of the differential diagnosis provides performance of tool examination differently builds-up of the plan of padding examination. On the establishment of the differential diagnosis the closing clinical diagnosis which is valid and actual basis for treatment of patients is stated.

Thus the clinical diagnosis in medicine does not come up suddenly in head of the doctor, and is drawn up during diagnostic procedure.

MOTOR DISORDERS

On the establishment of algorithm of build-up of the diagnosis we shall consider **motor disorders**.

Locomotion is a basic function of a human body. All manifestations of vital activity of organism are reduced to locomotion.

Locomotions are ensured with anatomical safety of locomotion, nervous system, psychics.

Therefore disorders of locomotions arise in dependence on lesion of one of the basic systems of organism. Disorders of locomotions are osteo- and arthrogenic, myogenetic, neurogenic and psychogenic. The clinical neurology surveys neurogenic and myogenetic infringements of locomotions.

In dependence on anatomical basis of disorder neurogenic motor disorders are pyramidal, extrapyramidal and cerebellar.

Neurogenic disorders of locomotions are in the following forms. 1. Paresises and paralyses.

2. Pathological muscle weariness
3. Convulsions.
4. Ataxia.
5. Hypokinesia.
6. Hyperkinesias.

The clinical manifestation of paresis is the fundamental neurologic concept. The paresis is decrease of muscle force and restriction of volume of the voluntary movements.

Paresises are different. In dependence on localization on body paresis named as monoparesis, biparesis, paraparesis - upper or inferior, hemiparesis, triparesis, tetraparesis, akinetic mutism.

In dependence on state of muscle tone paresises are spastic and flaccid. In dependence on the mechanism of development paresises are central, peripheral, mixed and psychogenic.

In dependence on acuteness of development paresis is acute, the reduction and residual.

Syndrome of Central Spastic Paralysis

1. decrease in strength associated with loss of subtle movements
2. spastic increase in tone (hypertonia)
3. exaggerated proprioceptive reflexes with or without clonus

4. decrease in or loss of exteroceptive reflexes (abdominal, cremasteric, plantar reflexes)

5. appearance of pathologic reflexes (Babinski's, Oppenheim's, Gordon's, Mendel-Bechterew, and others)

6. no degenerative muscular atrophy.

The symptomatology varies according to the location of the lesion along the course of the pyramidal tract. Involvement of the tract at eight different levels, indicated by black bars identified by the letters a through h.

a) **Subcortical lesion** Contralateral monoparesis, not monoplegia, occurs. The paresis results from the almost total preservation of extrapyramidal fibers. A small lesion of the cortex of area 4 produces quite often focal epileptic attacks (Jacksonian epilepsy).

b) **Internal capsule lesion**: Contralateral spastic hemiplegia occurs, because the pyramidal and extrapyramidal fibers are close to each other. Because the corticonuclear tract is also involved, there is contralateral paralysis of facial and possibly of hypoglossal nerves. c) Peduncle lesion: The result of this lesion is a contralateral spastic hemiplegia, which may be associated with ipsilateral paralysis of the oculomotor nerve.

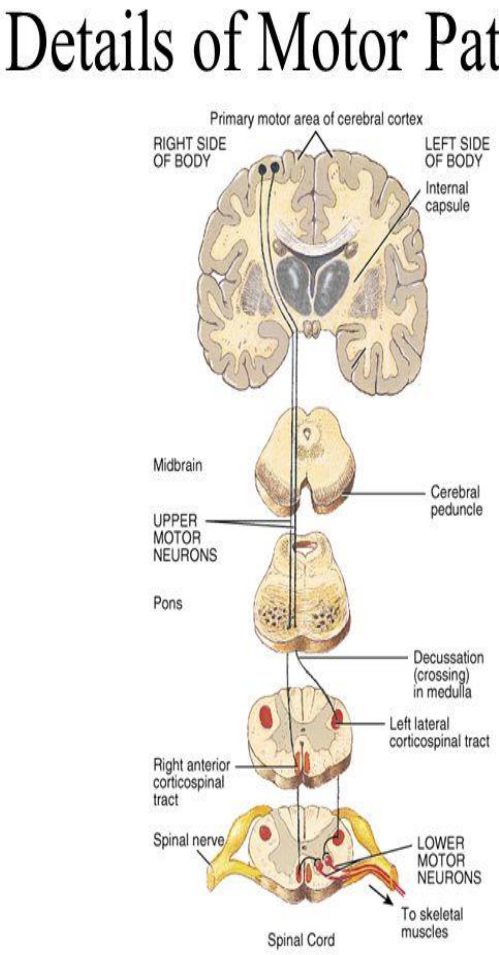
d) **Pons lesion:** The result of this lesion is a contralateral hemiplegia. Often, not all pyramidal fibers are damaged. Because the fibers descending to the facial and hypoglossal nuclei are more dorsally located, the facial or hypoglossal nerves may be spared. On the other hand, there may be an ipsilateral paralysis of abducens or trigeminal nerves.

Syndrome of Flaccid Paralysis

The syndrome of flaccid paralysis is composed of the following symptoms:

1. decrease in overall strength
2. hypotonia or atonia of the muscle
3. hyporeflexia or areflexia
4. neurogenic muscle degeneration

Schema of the pathways of the voluntary movements



Picture 1.

Principle schema of the localization of the pathological focus and motor syndromes

Table 1.

Localization of the lesion	Neurological syndrome
Motor cortex (precentral gyrus)	Central contralateral monoparesis of extremities ore hemiparesis (according to extension of the lesion)
Corona radiate in white matter of the brain hemisphere	Central nonsymmetrical contralateral hemiparesis
Internal capsule	Central symmetrical contralateral hemiparesis
Half of the brainstem	Alternating syndrome (peripheral ipsilateral paresis of the muscles of the face and central contralateral paresis of extremities)
Lateral funiculus of the spinal cord above the cervical thickening	Central ipsilateral biparesis
Lateral funiculi of the spinal cord above the cervical thickening	Central tetraparesis, central neurogenic bladder
Cervical thickening	Peripheral paresis of the arms, central paresis of the legs, central neurogenic bladder
Lateral funiculus of the spinal cord below the cervical thickening	Central ipsilateral paresis of the leg
Lateral funiculi of the spinal cord below the cervical thickening	Central paraparesis of the legs, central neurogenic bladder
Lumbar thickening	Peripheral paraparesis of the legs, central neurogenic bladder

SENSORY DISORDERS

The sensory system places the individual in relationship with the environment. Every sensation depends upon impulses which are excited by adequate stimulation of receptors, or end-organs. These impulses are carried to the central nervous system by means of afferent or sensory nerves, and are then conveyed through fiber tracts to higher centers for conscious recognition, reflex action, or other consequences of sensory stimulation. Receptors of various types are situated in the skin, subcutaneous tissues, muscles, tendons, periosteum, and visceral structures. Traditionally these receptors were considered to be specific for individual sensations, each responding only to certain stimuli.

Abnormalities of sensation may be characterized by increase, perversion, impairment, or loss of feeling. Increase in sensation is usually manifested by pain- an unpleasant or disagreeable feeling that results from excessive stimulation of certain sense organs, fibers, or tracts. It may result from a stimulus that partially injures the sense organs, acting to make the patient aware of noxious stimuli and thus to protect him from them. The severity of pain depends upon a number of factors: the tissues affected; the duration, extent, and quality of the stimulus; the personality of the individual and his powers of discrimination. Pain is accompanied by an emotional state as well as by other physical reactions so that the entire pain experience is complex in nature. Often the patient's description of the symptom is the sole guide to its character and severity. Perversions of sensations take the form of paresthesias, dysesthesias, and phantom sensations. Some of these are associated with irritation of receptors, fibers, or tracts, whereas others are release phenomena. Impairment and loss of feeling result from lessening of the acuity of the sense organs, decrease in the conductivity of the fibers or tracts, or dysfunction of higher centers with a consequent decrease in the powers of recognition or of perception.

The sensory examination is performed to discover whether areas of absent, decreased, exaggerated, perverted, or delayed sensation are present. The quality and type of sensation that is affected, the quantity and degree of involvement, and the localization of the change should be determined. There may be any of the following: loss, decrease, or increase of one or of all types of sensation; dissociation of sensation with loss of one type but not of others; loss in ability to recognize dif-

ferences in degrees of sensation; misinterpretations of sensation; areas of localized tenderness or hyperesthesia. More than one of these may occur simultaneously. The presence of trophic changes, especially painless ulcers and blisters, is also an indication for careful tests of sensation; these may be the first manifestations of a sensory disorder of which the patient is not aware.

Before starting the investigation, the examiner should determine whether the patient is aware of subjective changes in sensation or is experiencing spontaneous sensations of an abnormal type. The patient should be asked whether he notices pain, paresthesias, or loss of feeling; whether any part of the body feels numb, dead, hot, or cold; whether he has perceived sensations such as tingling, burning, itching, "pins and needles," pressure, distention, or feelings of weight or constriction. If such symptoms are present, the examiner should attempt to determine their type and character, intensity, exact distribution, duration, and periodicity, as well as factors that accentuate, produce, and decrease them. Subjective pain sensations should be differentiated from tenderness, which results from touch or pressure. It should be recalled that pain and numbness may exist together, as they do in the thalamic syndrome and in peripheral neuritis. The patient's manner of describing the pain or sensory disturbance and the associated affective responses, the nature of the terms used, the localization, and the precipitating and relieving factors may aid in differentiating between organic and psychogenic disturbances. Psychogenic pains are often associated with inappropriate affect (either excessive emotion or indifference) and are vague in character or location, and reactions to them are not consistent with the degree of disability.

Types of sensations

Sensations may be classified into various categories. Anatomists differentiate between somatic and visceral sensation, with general and special varieties of each. Most of those that can be tested clinically, however, fall into the general somatic group. The terms epicritic and protopathic sensibility were used by Head.

These terms do have a place in clinical neurology, but a more practical classification is that of Sherrington, who listed exteroceptive, proprioceptive, and interoceptive sensations, basing his types on the location of the end organs and the types of stimuli that they mediate. It

is this classification that will be used in the followings. To these is added the so-called combined sensations, or those which for their recognition require integrative cerebral functions.

The exteroceptive sensation

The exteroceptive sensations are those which originate in sense organs in the skin or mucous membranes in response to external agents and changes in the environment. They may also be designated the superficial or the cutaneous and mucosal varieties of sensation. There are three major types: pain, temperature (hot and cold), and tactile (light touch).

Clinical evaluation of superficial sensation

Many different procedures for testing superficial pain sensation have been described. Perhaps the simplest method, one as reliable as any, is the use of a common pin. The latent time in the response to stimulation is eliminated and the delineation is most accurate if the examiner proceeds from areas of lesser sensitivity to those of greater sensitivity, rather than the reverse. If there is hyperalgesia, for example, the examiner should proceed from the normal to the hyperalgesic area. If the stimuli are applied in too close proximity and if they follow each other too quickly, there may be summation of impulses; on the other hand, if conduction is delayed the patient's response may refer to a previous stimulation. Algesia and algesthesia are the terms used to indicate pain sensibility. In recording the response to pain stimulation, alganesthesia and analgesia designate areas insensitive to pain; hypalgesia, those areas having decreased sensitivity; and hyperalgesia, those showing increased sensitivity.

Temperature sensation may be tested by the use of test tubes containing cold water (or cracked ice) and hot water, or, better, by the use of cold or warm metal tubes or other metal objects, since glass is a poor conductor. Changes in temperature sensibility are recorded by the terms thermanesthesia, thermhypesthesia, and thermhyperesthesia, modified by the adjectives hot and cold. Sometimes, following cordotomy or lesions at high spinal cord levels, the patient perceives either cold or warm stimuli as warm; this is termed isothermagnosia.

Various means are available for evaluating the **tactile sensations**. General tactile sensibility is tested by the use of a light stimulus such

as a camel's hair brush, a wisp of cotton, a feather, a piece of tissue paper, or even a very light touch with a fingertip. The terms anesthesia, hypesthesia, and hyperesthesia are used to designate changes in tactile sensation, but, unfortunately, these terms also denote changes in all types of sensation. Thigmanesthesia denotes loss of light touch. Loss of sensation on stimulation to or movement of the hairs is known as trichoanesthesia. Topoanesthesia may be used to indicate loss of tactile localization. Pressure sensation, or touch-pressure, may be regarded as a distinct type of tactile sensation, involving more gross pressure from the skin. Most pressure impulses, however, arise from subcutaneous structures rather than from the skin, and pressure sense is herein considered to be a variety of proprioceptive rather than of exteroceptive sensation.

Locating the Site of Neurologic Lesions.

In delineating and recording alterations in superficial sensations, it is important to differentiate between changes due to lesions of the peripheral nerves, the nerve roots, the spinal cord, or higher centers of the brain.

In **peripheral nerve lesions** the areas of anesthesia, hypesthesia, or hyperesthesia correspond to the areas of sensory distribution of specific nerves. All types of sensation, including the proprioceptive sensations, are altered within the distribution of the affected nerve or nerves. It is necessary to bear in mind, however, that individuals show variations in the areas supplied by the peripheral nerves, and in one patient the resulting change will differ from that in another, as shown in variations in the radial nerve supply. It is also important to recall that there are areas of algesic overlap for pain and temperature sensations. The demonstrable area of loss of pain and temperature perception in a lesion of a specific nerve is usually smaller than the distribution of the anatomically described cutaneous supply of the nerve. Consequently, with careful testing, one can identify an area of slight hypalgesia, with loss of ability to distinguish a slight difference in pain and thermal stimuli; then there is an area of marked hypalgesia and hypesthesia, within which, however, the patient may identify ordinary tactile stimuli; and finally an area of complete anesthesia and analgesia. Occasionally there is spread of sensory loss beyond the field of an injured nerve. Those nerves supplying the face and body have a certain amount of "crossing" at the midline, more on the body

than on the face. Therefore, an organic anesthesia usually ends before the midline is reached.

In lesions confined to the **nerve roots**, areas of anesthesia or hypesthesia may be detected which are limited to the segmental distribution of these roots. All types of sensation are affected. Instead of sensory loss there may be hyperesthesia and radicular, or girdle, pains in the same distribution. The skin areas innervated by specific segments of the cord or their roots or dorsal root ganglia are called dermatomes. The distribution of these has been studied by Head, who based his observations on herpetic lesions and traumatic involvement of the spinal cord and the cauda equina, and later by Sherrington and Foerster, who performed isolated posterior root sections and noted the remaining, or unaltered, sensibility after certain roots were sectioned. Foerster also determined the distribution of the dermatomes by making use of antidromic responses (reverse conduction of impulses) and noting the vasodilation that followed stimulation of the cut end of a dorsal root.

On occasion it may be necessary to differentiate between peripheral nerve and radicular lesions on the one hand and the more complex type of involvement that is found in lesions of the cervical, brachial, lumbar, or sacral plexuses on the other hand. A lesion of the upper trunk or the posterior cord of the brachial plexus, for instance, will result in changes in sensation which differ from those found in segmental or radicular involvement and from those arising from peripheral nerve lesions.

In **lesions of the brain stem and spinal cord**, or the cerebrospinal axis, the sensory changes are similar to those occurring with nerve root lesions. That is, they have segmental or dermatome distribution. The examiner can localize with fair accuracy the level of involvement if he recalls the following: the first cervical segment has no supply to the skin; the interaural or vortex- meatal line forms the border between the areas supplied by the trigeminal nerve and the second cervical segment; the fifth and sixth cervical segments supply the radial side of the arm, forearm, and hand; the eighth cervical and first thoracic, the ulnar side of the forearm and hand; the fourth thoracic, the nipple level; the tenth thoracic, the umbilicus; the twelfth thoracic and first lumbar, the groin; the first three lumbar, the anterior aspect of the thigh; the fourth and fifth lumbar, the anterior and lateral aspects of the leg; the first and second sacral, the little toe, most of the sole of the

foot, and the posterior aspect of the thigh and leg; the fourth and fifth sacral segments, the perianal region. In a spinal cord lesion there may be anesthesia of the body below the uppermost level of the lesion as a result of involvement of the ascending pathways. There may be hyperesthesia at the dermatome level of the lesion. There may be a zone of gradual transition. The level for pain and temperature sensations is most specific, and it may be difficult to delineate definite changes in tactile sensation. Furthermore, since the pain fibers from the lower portions of the body are lateral, pressure on the cord from one side may affect only the external fibers, and the resulting loss in pain sensation may be significantly below the level of the lesion.

It is also important to recall that the segments of the spinal cord and the spinal processes of the vertebrae are not on corresponding levels. In the upper cervical region the spinal cord level is about one segment higher than that of the corresponding spinal process; in the lower cervical and thoracic regions there is a difference of about two segments, while in the lumbar region there is a difference of almost three segments. The spinal cord ends between the bodies of the first and second lumbar vertebrae in adults. Furthermore, in lesions of the cerebrospinal axis there is often a dissociation of sensation, with loss, for instance, of pain and temperature sensations but little or no impairment of touch, owing to involvement of certain sensory pathways but the sparing of others.

The anatomic differentiation between the conduction of pain and temperature impulses on the one hand, and the conduction of tactile impulses on the other, is valuable in the diagnosis of neurologic disorders and in certain types of therapy. In syringomyelia, for instance, where the primary pathologic change is situated in the vicinity of the central canal of the spinal cord, the decussating pain and temperature fibers are interrupted. The earliest clinical manifestation may be a dissociation of sensation, with loss of pain and temperature modalities in the areas of the body supplied by the involved segments, whereas tactile sensation may not be affected. If the medial portions of the lateral spinnothalamic tract are later involved, there may be interference with the conduction of pain and temperature sensations from other parts of the body. Occasionally, deep pain sensation is lost, with otherwise normal sensory perception. In hemisection of the spinal cord of the Brown Squared variety, pain and temperature sensations are lost on

the opposite side of the body below the level of the lesion, whereas tactile sensation shows little if any evidence of change; usually a corticospinal paralysis and loss of proprioceptive sensations occur on the side of the lesion, often with anesthesia in the distribution of the involved segments on that side. With transverse lesions of the spinal cord all types of sensation are lost below the level of the lesion, the level for pain and temperature being the most distinct and well demarcated; the level is within one or two segments of the site of the lesion, owing to the immediate decussation of the fibers. There may be radicular pain in the distribution of the involved segments.

Special Varieties of Sensory Changes.

In the neuritis or in conditions where there is irritation of or pressure on the nerve roots, hyperesthesia may be present instead of anesthesia, together with demonstrable tenderness of the involved nerves. The ulnar, the radial, or the common peroneal nerves may often be palpated under the skin, and pressure on them may cause pain. Occasionally, as in the hypertrophic neuritis of Dejerine and Sottas and in leprosy, the hyperplasia of the nerves is palpable under the skin. In the neuritis there may also be pain on brisk passive stretching of the affected nerves.

There is increased susceptibility to ischemia in the presence of peripheral nerve and dorsal root lesions, and even in spinal and cerebral lesions, and constriction of a limb will accentuate both subjective and objective sensory changes; this can be used to evaluate both improvement and deterioration of nerve status. On the other hand, pressure and ischemia also cause paresthesia in the distribution of normal nerves, probably the result of impairment of conduction of some fibers and irritation of others. In conditions such as trigeminal neuralgia there is irritability of one or more branches of the nerves with resulting "trigger" or "dolorogenic" zones on the face. Hyperesthesia may precede the development of vesicles in herpes zoster. Hyperesthesia of the hands and the feet, in spite of demonstrable hypoesthesia, is a frequent finding in patients with peripheral neuritis. This is especially true of the soles of the feet, which may be almost anesthetic to testing, yet extremely sensitive to all types of stimuli.

Spontaneous, or central, pain occurs most frequently with lesions of the thalamus. Similar pain, however, has also been described with cortical lesions, brain stem involvement (thrombosis of the posterior

inferior cerebellar artery), and affections (usually post-traumatic) of the spinal cord. Either stimulation or section of the spinnothalamic tract may cause a brief, intense, burning pain on the opposite side of the body, and neoplasms of the spinal cord may also cause contralateral pain. Phantom, or spectral, sensations are spontaneous sensations referred to insensitive areas; these may occur with lesions of the spinal cord or cauda equina. A phantom limb, on the other hand, is a sensation of the continued presence of an absent portion of the body, or of pain, paresthesia, or movement in it.

The terms allachesthesia, allesthesia, and synesthesia are used when the sensation of touch is experienced at a site remote from the point of stimulation; allochiria means the referring of a sensation to the opposite side of the body. Sensation in an affected area may be dulled when it and a normal area, usually the corresponding one on the opposite side of the body, are stimulated simultaneously; this denotes a cutaneous sensory extinction, or suppression, in the involved area, even though sensation may appear to be normal at the affected site if it is the only area stimulated. Occasionally a painful stimulus of high intensity in an analgesic area may cause perception of pain in adjacent regions on the same or opposite side of the body, owing to spread of excitatory processes within the segments of the cerebrospinal axis. After a cordotomy the patient may perceive pain on the normal side when the analgesic side is intensely stimulated.

Among the special varieties of sensory changes that may be found in the extremities are the following: Causalgia is a neuritis characterized by a disagreeable, burning type of pain, often accompanied by trophic changes, and most frequently seen in lesions of the median and sciatic nerves; acroparesthesia is a disease characterized by tingling, numbness, burning, and pain of the extremities, chiefly of the tips of the fingers and toes, often accompanied by cyanosis; meralgia paresthetica is a painful paresthesia in the area of distribution of the lateral femoral cutaneous nerve; digitalgia paresthetica is an isolated neuritis of the dorsal digital nerve of one of the fingers; gonyalgia paresthetica is a sensory neuritis of the infrapatellar branch of the saphenous nerve.

The proptioceptive sensations

The proprioceptive sensations arise from the deeper tissues of the body, principally from the muscles, ligaments, bones, tendons, and joints. Kinesthesia is the sense by which muscular motion, weight, and

position are perceived. Bathyesthesia is deep sensibility, or that from the parts of the body which are below the surface, such as the muscles and the joints. Myesthesia is muscle sensation, or the sensibility of impressions coming from the muscles. The aforementioned terms are sometimes used as synonyms for proprioceptive sensation, but the latter is somewhat more inclusive and specific varieties of it will be described.

The proprioceptive sensations which can be tested clinically are those of motion and position, vibration, and pressure; considered with the proprioceptive sensations, but somewhat different in nature, is deep pain.

The sense of motion, also known as the kinetic sense, or the sensation of active or passive movement, consists of an awareness of motion in the various parts of the body. The sense of position, or of posture, consists of an awareness of the position of the body or its parts in space. Arthresthesia is used to designate the perception of joint movement and position, and statognosis to indicate the awareness of posture.

Sensations of motion and position may be tested by placing the fingers of one of the patient's hands in a certain position while his eyes are closed, then asking him to describe the position or to imitate it with the other hand. The foot may be passively moved while the eyes are closed, and the patient asked to point to his great toe or his heel. The patient may be asked to hold his hands outstretched; with loss of position sense one hand may waver or droop. One of the outstretched hands may be passively raised or lowered while the patient's eyes are closed, and the patient is asked to place the other extremity at the same level. One of the hands may be passively moved while the eyes are closed, and the patient asked to grasp the thumb or forefinger of that hand with the opposite hand. These latter tests, however, do not denote the side of involvement when a unilateral lesion is present.

Cerebral sensory functions

The term combined sensation has been used to describe those varieties of sensation for the recognition of which more than one of the senses is used. They are not mere combinations of sensation, however, and in most instances a cortical component is necessary for the final perception. This cortical component is a function of the parietal lobes,

which act to analyze and synthesize the individual varieties of sensation; to correlate, integrate, and elaborate the impulses; to interpret the stimuli; and to call out memories to aid in discrimination and recognition. The resulting manifestations are perceptual and discriminative functions rather than the simple appreciation of the stimulation of primary sensory nerve endings. The more important combined sensory functions will be described.

Stereognosis is a faculty of perceiving and understanding the form and nature of objects by touch, and of identifying and recognizing them. When this ability is lost, the patient has astereognosis, or tactile agnosia. Astereognosis can be diagnosed only if cutaneous and proprioceptive sensations are present, for if these are significantly impaired, the primary impulses cannot reach consciousness for interpretation. Various qualities or steps may be noted in the recognition of objects. First, the size is perceived. Then the appreciation of shape in two dimensions is noted, then form in three dimensions, and finally there is identification of the object. Size perception may be tested by the use of objects of the same shape but of different sizes.

Barognosis is the recognition of weight, or the ability to differentiate between weights. It is tested by the use of objects of similar size but of different weights, such as a series of plastic or wooden balls, or blocks loaded with different weights, which are appraised by holding them in the hand, either unsupported or resting on a table, but preferably the former. The senses of motion and position should be intact. Loss of ability to differentiate weight is known as baragnosis.

Topesthesia, or topognosia, is the ability to localize a tactile sensation. Its loss is known as topoanesthesia, or topagnosia. Localization is impossible if there is cutaneous anesthesia, but the loss of the sense of localization with an intact exteroceptive sensibility usually signifies the presence of a lesion affecting the parietal lobe.

Graphesthesia is a term used to designate the ability to recognize letters or numbers written on the skin. Letters or numbers 1 mm in height may be written on the finger pads, and up to 4 mm in height on the forearm and legs. A pencil or a dull pin is used to write the letters, and the patient is asked to identify them. Easily identifiable, dissimilar numbers should be used, e.g., 3 and 4 rather than 3 and 8. Loss of this sensation is known as graphanesthesia.

Two-point, or spatial, discrimination is the ability to differentiate cutaneous stimulation by one blunt point from stimulation by two points. A compass or a calibrated two-point esthesiometer is used, and the patient is stimulated randomly by a single point and by two points. Bending a paperclip to different distances between its two points is less quantitative but readily available for quick evaluation of differences between the two sides of the body. The patient's eyes should be closed during the test. It is best to start with the two points relatively far apart, and single and double points should be varied unpredictably; the points are approximated until the patient begins to make errors. One notes the minimum distance between two points that can be felt separately. The distance varies considerably in different parts of the body. Two points can be differentiated from one at a distance of 1 mm on the tip of the tongue, at 2 - 4 mm on the fingertips, at 4 - 6 mm on the dorsum of the fingers, at 8-12 mm on the palm, and at 20 - 30 mm on the dorsum of the hand. Greater distances are necessary for differentiation on the forearm, upper arm, torso, thigh, and leg. The findings on the two sides of the body must always be compared.

Autotopagnosia, or somatotopagnosia, is the loss of power to identify or orient the body or the relation of its individual parts - a defect in the body scheme. The patient may have complete loss of personal identification of one limb or of one half of the body. He may drop his hand from the table onto his lap and believe that some other object has fallen, or he may feel an arm next to his body and not be aware that it is his own. Lack of awareness of one half of the body is referred to as agnosia of the body half.

In the syndrome of finger agnosia of Gerstmann there is an inability to recognize, name, and select individual fingers when looking at the hands. This may apply to both the patient's and the examiner's fingers. Accompanying this, in the full-blown syndrome, is loss of awareness of the position and identity of the parts of the body, with disorientation for right and left, agraphia, and acalculia.

Anosognosia is defined as the ignorance of the existence of disease and has been used specifically to imply the imperception of hemiplegia, or a feeling of depersonalization toward or loss of perception of paralyzed parts of the body, either due to anesthesia of the paralyzed parts or to amnesia for them. The patient may believe that he is

able to use his paretic extremities in a normal manner. Anosognosia is most often found in lesions of the right parietal lobe.

The aforementioned are all complicated varieties of disturbances of cerebral sensory function, indicating involvement of parietal areas or their connections.

The parietal cortex receives correlates, synthesizes, and elaborates the primary sensory impulses. It is not concerned with the cruder sensations, such as recognition of pain and temperature, which are subserved by the thalamus (to be described later in this chapter). It is important in the discrimination of the finer or more critical grades of sensation, such as the recognition of intensity, the appreciation of similarities and differences, and the evaluation of the gnostic, or perceiving and recognizing, aspects of sensation. It is also important in localization, in the recognition of spatial relationships and postural sense, in the appreciation of passive movement, and in the recognition of differences in form and weight and of two-dimensional qualities. These elements of sensation are more than simple perceptions, and for their recognition it is necessary to integrate the various stimuli into concrete concepts as well as to call forth engrams. They are diminished or absent in lesions of the anterior portion of the middle third of the postcentral gyms of the parietal lobe. The loss of each of these varieties of combined sensation may be considered a variety of agnosia, or the loss of the power to recognize the import of sensory stimuli.

Occasionally loss of ability to recognize the size and shape of objects is encountered with lesions of the cervical portion of the spinal cord and even of the posterior nerve roots and brachial plexus. **Autotopagnosia** also has been observed with lesions in these sites. In such instances, however, there is severe involvement of proprioceptive as well as of cutaneous sensation. The term stereoanesthesia is occasionally used when the difficulty results from infracerebral lesions, and the term astereognosis is reserved for disturbances that follow interference with cortical synthesis.

Lesions of the parietal cortex are not associated with anesthesia or complete loss of sensation. There may be diminution in the appreciation of the various modalities, with a raising of the threshold on the opposite side of the body, but both exteroceptive and proprioceptive sensations are perceived. Sensation is often disturbed more in the upper than in the lower extremity, trunk, or face. The distal parts of the

extremities are affected more than the proximal portions, with a gradual transition to more normal perception as the shoulder and hip are approached. Perception becomes normal before the midline is reached on both face and trunk.

The threshold for pain stimuli is raised very little in parietal lesions, although a prick may feel less sharp than on the normal side; with deeper lesions the threshold is more definitely raised. The qualitative elements of heat and cold are present, but there is loss of discrimination for slight variations in temperature, especially in the intermediate ranges. Light touch perception is little disturbed, but tactile discrimination and localization may be profoundly affected. There often is severe impairment of postural sense; this results in sensory ataxia and athetoidlike movements. Vibratory sensation is only rarely affected. Astereognosis, baragnosis, graphanesthesia, and impairment of two-point discrimination may all be present. The period required for sensory adaptation is prolonged, and occasionally allachesthesia is experienced. Disorders of the body image such as autotopagnosia, anosognosia, and Gerstmann's syndrome occur with localized involvement.

Lesions between the thalamus and the cortex, especially those affecting the posterior limb of the internal capsule, cause more severe and extensive sensory loss than isolated cortical lesions. In this area the fibers are crowded closely together.

Detailed examinations of sensory perception and critical evaluation may be necessary to diagnose lesions of the **parietal lobe**. Both small and large objects may have to be used in testing for astereognosis; sometimes a delay in answering when objects are placed in the affected hand, with no delay when the other side is examined, may be a clue to minimal involvement. A similar detailed investigation of tactile localization and discrimination may be essential. Sensory inattention, or extinction, is often an early and important diagnostic finding in parietal lobe lesions. With involvement of one parietal area, the stimulus on the opposite side of the body will not be perceived, even though sensation on that side may be normal with routine testing. Bilateral simultaneous testing for stereognostic sense (placing identical objects in the patient's hands) may yield valuable information. The ability to distinguish two cutaneous stimuli separated by a brief time interval is also impaired with parietal lobe lesions. Double simultaneous stimulation

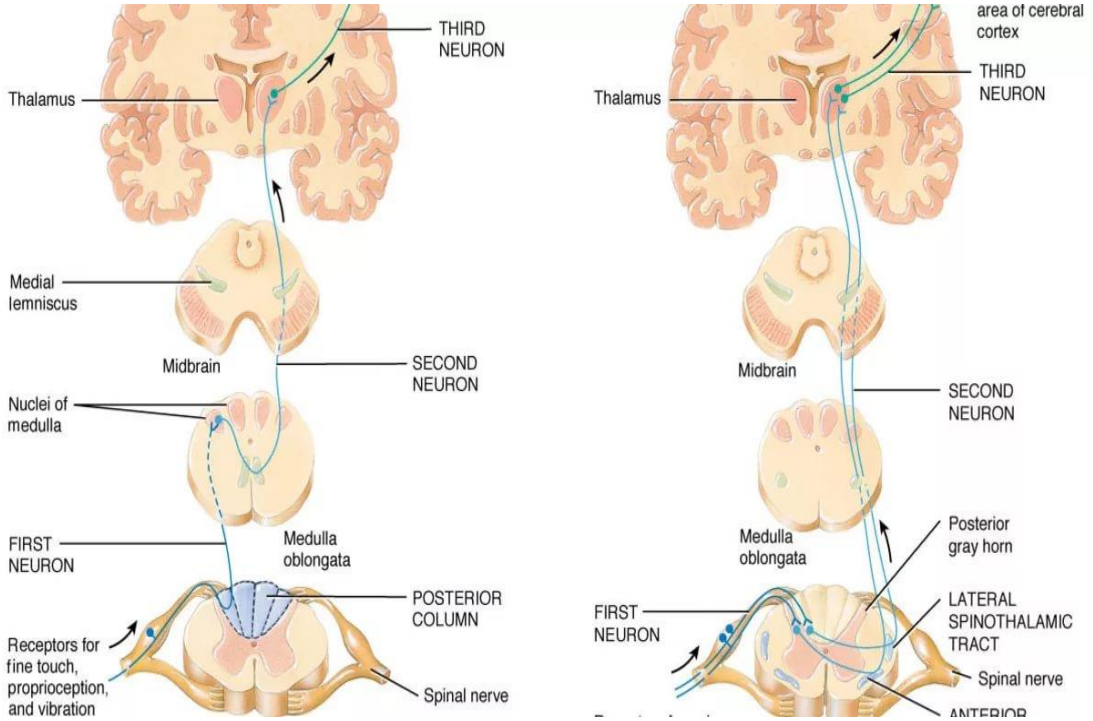
above and below the presumed level of a spinal cord lesion in which there is relative but not absolute sensory loss may aid in demonstrating the level of the lesion. If the upper stimulus only is perceived, the lower is moved more rostrally until the intensity of both is equal; this may indicate the segmental level of the lesion. Bilateral or homolateral simultaneous stimulation of two different segments of the body (heterologous areas) shows that even in normal subjects one stimulus "dominates" the other, at least for the initial tests. In young children, patients with organic disease of the brain, and the aged this phenomenon is demonstrable still more frequently, and it appears that in general the more rostral area is the dominant one; when face and hand are stimulated, there is extinction of the hand percept (the face-hand test).

Sensory impulses that enter consciousness for interpretation by the parietal cortex must first pass through the thalamus and then be redistributed. The thalamus is thought to be the end-station in the quantitative interpretation of pain, heat, cold, and heavy contact, and is a receptive center wherein sensory impulses produce a crude, uncritical form of consciousness. A lesion in or near the thalamus may cause loss of various sensations, owing to interruption of the impulses on which they depend. A severe and extensive lesion may cause gross impairment of all forms of sensation on the opposite side of the body, probably as a result of damage to the nucleus ventralis posterolateralis (body) and posteromedialis (face). Marked loss of appreciation of heavy contact, posture, passive movement, and deep pressure perception occurs, and the thresholds for light touch, pain, and temperature sensations are raised.

In the thalamic syndrome of **Dejerine and Roussy**, which occasionally accompanies a cerebral hemorrhage or thrombosis, there is a characteristic group of symptoms, the result either of damage which predominates in the nucleus ventralis posterolateralis or of an interruption of the pathways from the thalamus to the cerebral cortex. The responsible lesion need not be limited to the thalamus. There is blunting, or raising of the threshold, of all forms of sensation on the opposite side of the body, without true anesthesia. All stimuli, however, when effective, excite unpleasant sensations, and any stimulus, even the lightest, may evoke a disagreeable, often burning, type of pain response. Slight hot and cold stimuli, and also light cutaneous sensations, excite marked discomfort. The overreaction is termed hyperpa-

thia, and the diminution of all varieties of superficial and deep sensibility, accompanied by subjective intractable pain in the hypesthetic regions, is called anesthesia dolorosa. In addition to sensory changes, a hemiparesis or hemiplegia and a hemianopia usually occur and, less frequently, hemiataxia, choreoathetosis, and unmotivated emotional responses. Occasionally, pleasurable stimulation, such as that produced when a warm hand is applied to the skin on the affected side, may be markedly accentuated. This thalamic overreaction is due either to irritation of the thalamus or to release of thalamic function from normal cortical control by damage to higher centers. Every stimulus acting on the thalamus produces an excessive effect on the abnormal half of the body, especially as far as the affective element - the pleasant or unpleasant character in its appreciation - is concerned.

Schema of the sensory pathways



Picture 2

Diminution or loss of sensation may occur in the presence of lesions at various levels of the nervous system, as may abnormal sensations, such as pain or paresthesia.

Either diminution or perversion of sensation occurs in the presence of lesions involving the end-organs, or sensory receptors, such as the pain endings and Meissner's corpuscles. These are microscopic structures, and pathologic changes in them are difficult to evaluate. Quanti-

tative studies of Meissner's corpuscles, however, have shown that they decrease in number with advancing age, and that their cholinesterase activity is decreased in the distribution of under-functioning peripheral nerves. The pain that is perceived with irritation of the skin, traumatic denudements, and burns, as well as pruritus, paresthesias, and dysesthesias, may result from irritation of these distal structures or the nerve filaments to them, and decreased sensation in callosities and scars may result from involvement of the end-organs and smaller filaments. Infiltration of an area with a local anesthetic or freezing with ethyl chloride will cause anesthesia.

In lesions of the peripheral nerves there usually is a loss or diminution in all types of sensation in the distribution of the nerve or nerves affected. In peripheral neuritis, vibration is often the first modality to be found to be defective, but in severe cases all exteroceptive, proprioceptive, and combined modalities are impaired. An irritative lesion of a peripheral nerve may also cause abnormal sensation in the form of paresthesias or of pain that is either constant or lancinating in character. The nerves themselves may be hyperalgesic and sensitive, or tender to pressure, and there may be pain on brisk stretching of the affected nerves and increased susceptibility to ischemia. There sometimes is hyperalgesia in the cutaneous distribution of the nerves, even though the sensory threshold is raised. The unmyelinated fibers may be most affected, so the threshold for slow or aching pain is depressed, while the myelinated fibers may suffer less damage, and the threshold for fast or piercing pain is raised. In polyneuritis the distribution of sensory loss is variable, usually involving predominantly the distal segments. There may be what appears to be a glove and stocking distribution of altered sensation, but the margins of this area are poorly demarcated, and usually there is a peripheral blunting of sensation with no sharp border between the normal and hypesthetic areas.

The sensory examination is important in the diagnosis of peripheral nerve injuries and in the evaluation of progress in nerve regeneration. Pain in the distribution of a single nerve or group of nerves can be relieved by section of the nerves involved or injection with local anesthetic agents, alcohol, or phenol. Section or injection of a nerve peripheral to the dorsal root ganglion, however, may be followed by regeneration and return of pain.

Disease of the dorsal root ganglia (or corresponding ganglia of the cranial nerves) is also associated with sensory changes. In herpes zoster there is severe, lancinating pain in the distribution of the affected ganglia. In the now rare tabes dorsalis there is loss of deep pain, a delayed response to superficial painful stimulation, and, sometimes, impairment of superficial pain sensation. Transient, spontaneous "lightning" pains may develop. In hereditary sensory neuropathy the pathologic lesions are in the dorsal root ganglia; there is severe distal loss of all sensory modalities, along with trophic changes in the extremities.

The nerve root is in reality a part of the peripheral nerve, since it constitutes a part of the same neuron. Radicular lesions also are accompanied by diminution or loss of sensation, and by either pain or paresthesias, but the distribution is segmental in type. Irritation of the nerve roots causes pain in a radicular, sometimes girdle, distribution (i.e., encircling the body). The pain may be either constant or intermittent, but it is often of a sharp, stabbing, lancinating character. It is increased by movement, coughing, or straining. There may be either hypalgesia or hyperalgesia. Owing to algesic overlap, sensory changes may be difficult to demonstrate if but one root is involved.

Pain of a radicular distribution is sometimes relieved by injection or section of the root. If the nerve root is sectioned between the cerebrospinal axis and the ganglion, as in the rhizotomy or retrogasserian neurectomy done for the relief of pain in trigeminal neuralgia, no regeneration of the nerve occurs. Spinal anesthesia, caudal analgesia, or subarachnoid injection of alcohol or phenol also can relieve such pain by interrupting the conductivity of the nerve root.

With lesions of the spinal cord and brain stem, impairment of one or more modalities of sensation, or perversions of sensation in the form of either pain or paresthesia, may develop. The area of sensory diminution or loss, and the paresthesia as well, may involve the entire body below the level of the lesion, whereas the pain is usually segmental and involves only the derma-tomes supplied by centers at the level of the lesion. The sensory loss is usually dissociated, with impairment of certain modalities and sparing of others. Despite a raised threshold for pain stimuli, there may be an overreaction to rapidly repeated stimulation.

When lesions of the spinal cord are present, involvement of pain, temperature, discriminatory, and proprioceptive sensations occurs.

With brain stem lesions there may be ipsilateral sensory loss on the face and contralateral changes on the body. Lesions high in the cervical spinal cord and in the medulla may impair kinesthetic sensation in the upper extremities more than in the lower. As a result of the disturbance of proprioceptive sensations and a raised threshold for cutaneous senses there may be stereo-anesthesia, which is difficult to differentiate from astereognosis. Extinction and even autotopagnosia may be present with such lesions. "Central" pain is occasionally experienced in patients with pontine, medullary, and spinal cord involvement. Lhermitte's sign, which consists of sudden electric-like or painful sensations spreading down the body or into the back or extremities on flexion of the neck, may be present with either local lesions of the cervical cord, or multiple sclerosis or other degenerative processes; the phenomenon may be secondary to disease of the posterior columns. It has also been reported following head injury; in such instances it may be the result of subdural or sub-arachnoid adhesions.

The pattern of sensory return with recovering spinal lesions is variable; the impairment may recede downward in a segmental manner; the return may start in the sacral distribution and ascend, or there may be a gradual recovery of function over the entire affected area. Pressure sensation returns first and its recovery is usually the most complete, followed, in turn, by tactile, pain, cold, and heat sensibilities. Intractable pain may be relieved by section of the ascending pathways in the spinal cord, medulla, pons, or mesencephalon.

Lesions of the thalamus are followed by diminution of various sensory modalities on the opposite side of the body without loss of sensation. They may be associated with abnormalities of sensation, such as paresthesia and hyperesthesia, or painful hyperpathia. Pain of central origin is most often associated with thalamic lesions, although it may occasionally be caused by stimulation of ascending pathways in the cerebrospinal axis or lesions of the cortex. When associated with cerebral lesions it probably is not a "spontaneous" pain, but rather an overreaction to stimuli capable of exciting affective reactions and sensations.

Involvement of the sensory radiations in the internal capsule causes variable and sometimes extensive diminution of all types of sensation on the opposite side of the body. The changes are similar to those

which follow a thalamic lesion, and it may be difficult to differentiate between the two. Pain, however, is rarely experienced.

Lesions of the parietal cortex cause disturbances in the discriminatory sensations. There may be astereognosis, baragnosis, and sensory inattention or extinction, as well as autotopagnosia and anosognosia. Anesthesia is rare, but there is a raising of the threshold for both exteroceptive and proprioceptive sensations of the opposite side of the body (Part H, "Diagnosis and Localization of Intracranial Disease"). Irritative lesions rarely cause pain, but they frequently cause paresthesias on the opposite side of the body. These paresthesias are especially important clinically when they assume the manifestations of a sensory aura preceding a jacksonian convulsion or constitute a focal sensory seizure.

The pains of causalgia and of phantom limb, which to a certain extent appear to be due to a hypersensitive receptive mechanism at the cortical level, have been relieved by interruption of the centripetal sensory pathways leading to consciousness; this has been achieved by removal of the cortical sensory representation of the part (gyrectomy or topectomy). Abnormalities of sensation in the form of anesthesias, paresthesias, or pain may be present in the absence of organic etiology and may be of psychogenic origin. In addition to the use of measures mentioned previously, such as nerve and nerve root block and section, anterolateral cordotomy, and tractotomy, other means have also been used for the relief of intractable pain or the patient's reaction to it; these include prefrontal lobotomy (severing the corticothalamic connections), cingulumotomy, and either stereotaxic surgery or the use of implanted electrodes to stimulate the nucleus ventralis posteromedialis and posterolateralis of the thalamus. The internal capsule and other structures have also been stimulated for such purposes. Apparently the patient's awareness of or concern over pain is lessened, even though there may be little or no change in the pain threshold.

Universal insensitivity or indifference to pain is a rare condition that is usually congenital. A few cases have been reported in which the absence of pain sensation was a part of a congenital sensory neuropathy or was associated with the absence of organized nerve endings or of Lissauer's tract and small dorsal root axons, but in others detailed investigations have shown no abnormality. In these latter cases the sensory defect may result from some anatomic, physiologic, or chemi-

cal abnormality in the cerebral integration of pathways concerned with pain appreciation, or it may represent a form of sensory agnosia.

Principle schema of the localization of the pathological focus and sensory syndromes

Table 2

Localization of the lesion	Neurological syndrome
Brain cortex (postcentral gurus)	Sensory paroxysms on the contralateral half of the body. Disorders of the combined types of sensitivities, astereognosis, disorder of the «body schema».
Corona radiate	Conductory contralateral total hypoesthesia (monohypoesthesia).
Internal capsule	Conductory contralateral total hypoesthesia.
Thalamus	Contralateral total hypoesthesia, hyperpathia, thalamic pain, sensory contralateral ataxia.
Half of the brainstem	Alternating hypoesthesia (segmentary on the face ipsilataraly and conductory contralateral on the body and extremities)
Lateral funiculus of the spinal cord	Contralateral hypoesthesia of pain and temperature sensitivities from the level 2-3 segments below the pathological focus
Lateral funiculi of the spinal cord both sides	Conductory paraanesthesia of pain and temperature sensitivities from the level of the pathological focus
Posterior funiculus of the spinal cord	Conductory ipsilateral hypoesthesia of proprioception from the level of the pathological focus
Posterior funiculi of the spinal cord both sides	Conductory paraanesthesia of proprioception from the level of the pathological focus

Principle schema of the localization of the pathological focus and sensory syndromes

Table 2 (cont.)

Hemisection of the spinal cord	On the side of the lesion: central paresis below the lesion, - conductory hypoesthesia of proprioception below the lesion, on the opposite side conductory hypoesthesia of pain and temperature sensitivities from the level 2-3 segments below the pathological focus
Complete transection of the spinal cord	Total conductory anesthesia below the lesion
Posterior horn of the spinal cord	Dissociated segmentary disorder of sensitivity – pain and temperature in the correspondent dermatome
Posterior root	Pain, paresthesia, total hypoesthesia in the correspondent dermatome
Spinal ganglion	Pain, paresthesia, total hypoesthesia, herpetic rashes in the correspondent dermatome
Neural plexus	Pain, paresthesia, total hypoesthesia in the area of innervation of the affected plexus
Peripheral nerve	Pain, paresthesia, total hypoesthesia in the area of innervation of the affected nerve
Diffuse lesion of the peripheral nerves	Polyneural sensory disorder – hypoesthesia of all sensitivities in distal parts of extremities («gloves» and «stocking» distribution), pain, paresthesia, sensory ataxia

CRANIAL NRERVES AND BRAINSTEM LESION

The motor function of muscles of the face, eye globes, soft palate, pharynx, vocal cords and tongue, and also sensitivity of a skin of the face, mucosa of eye, oral cavity, nasopharynx and larynx is provided with cranial nerves. From 12 pairs of cranial nerves only sensitive are I, II and VIII pairs, motor - III, IV, VI, VII, XI and XII pairs and the mixed - V, IX, X pairs.

Some from them contain vegetative fibers (III, VII, IX and X pairs). Sensory nerves form periphery departments of analyzer: olfactory (I), visual (II), acoustical (VIII), vestibular (VIII) and gustatory (VII, IX). These nerves are transmitters of the information on environment in basic with the of receptors.

Two first of cranial nerves (olfactory and visual) on a constitution differ from others (they represent as though parts of a brain, borne on periphery).

Others 10 steam of cranial nerves, besides an originality of each of them, have also common features with spinal roots and nerves. We shall not be stop on anatomy as you are know it, but we shall discuss syndromology.

Principle schema of focus localization and syndromology of the cranial nerves

Table 3

Localization of the lesion	**Neurological syndrome**
Filla olfactoria, olfactory bulb, olfactory tract	Ipsilateral hyposmia (anosmia)
Parahyppocampal gyrus, uncus	Olfactory simple focal epileptic seizures
Optic nerve	Amaurosis (amblyopia), paracentral scotoma, simple atrophy of the optic disc, absence of direct and intact pupillary reaction to light
Optic chiasm intrinsic fibers	Bitemporal heteronymous hemianopia, amblyopia, optic disc atrophy
Optic chiasm extrinsic fibers	Binasal heteronymous hemianopia, amblyopia, optic disc atrophy
Optic tract, lateral geniculate body	Homonymous contralateral hemianopia, optic disc atrophy, positive scotoma, asymmetric visual field defects, absence of hemianopic pupillary response to light
Visual radiation (Gracile's bundle)	Homonymous contralateral hemianopia, no atrophy of the optic nerve discs, negative scotoma, symmetrical visual field defects, preservation of the hemianopic pupillary reaction to light
Primary visual cortex (medial surface of the occipital lobe, spur sulcus)	Quadrant visual field loss from the opposite side (wedge - lower quadrants, lingual gyrus - upper). Irritation - photopsies in the corresponding fields of view.
Associative visual cortex (lateral surface of the occipital lobe)	Visual agnosia. Irritation - complex visual hallucinations, macropsia, micropsia, metamorphopsia
Oculomotor nerve	Ptosis, diplopia, restriction of eyeball movements up, down, medially, mydriasis, accommodation paralysis

Principle schema of focus localization and syndromology of the cranial nerves

Table 3(cont)

Trochlear nerve	Mild convergent strabismus, diplopia when looking down
Abducens nerve	Converging strabismus, diplopia when looking towards the affected nerve
Frontal center of gaze (posterior part of the 2nd frontal gyrus)	Paresis of gaze in the opposite direction (eyes look at the focus)
Pontine center of gaze	Paresis of gaze in the direction of the lesion (eyes turn away from the focus)
Superior colliculli of the midbrain	Parino's syndrome - gaze paralysis upwards with the preservation of horizontal movements of the eyeballs, partial ptosis from 2 sides
Medial longitudinal bundle	Internuclear ophthalmoplegia
Trigeminal nerve	Trigeminal neuralgia of the face. Masticatory paresis. Segmental and neural anesthesia of the face.
Facial nerve	Mimic paresis ipsilaterally. Xerophthalmia. Dysacusia. Ageusia.
Cochleo-vestibular nerve	Defenses ipsilaterally. Vestibular ataxia.
Glossopharyngeal nerve.	Neuralgia of the pharynx. Throat anesthesia. Ageusia.
Vagus nerve	Dysphagia, dysphonia, paralysis of the pharynx, larynx, soft palate.
Accessory nerve	Paresis of the sternoclavomastiodeus and trapezius muscles ipsilaterally
Hypoglossal nerve	Paresis of half of the tongue ipsilaterally

Syndromes of lesion of the brainstem

The lesion of all diameter of brainstem incompatibles to life. In clinical practice it is necessary to meet patients with the locus of lesion in one half of brainstem. Almost always thus the nucleus or root any of cranial nerves is involved. Lockout of motor nucleus or axons of its cells produces flaccid paralysis of conforming muscles. Besides such locus routinely damages passing in neighborhood (pyramidal, spinnothalamic and bulbothalamic. There is paralysis of a cranial nerve on the side of the locus, hemiplegia or hemianaesthesia on counter. Such combination of neurologic distresses has received the name «an alternating set of symptoms » and allows to establish a lesion of cerebral fulcrum, and the lesion of cranial nerve determines level of the locus.

Lesion of the midbrain includes nucleus or roots of third cranial nerve the outside, intrinsic or total ophthalmoplegia educes; trochlear nerve - concurrent strabismus, diplopia at view downwards, vertical nystagmus (spontaneous vertical nystagmus, discoordinated locomotions of eyeglobes, an ophthalmoplegia, horizontal nystagmus, audition, a paralysis of oculomotor muscles, choreic hyperkynesias), paresis and paralyses of extremities, cerebellar distresses, cerebrate rigidity (it is connected to lesion of mesencephalon centers of regulating muscle tone of below red nucleus).

Parinau syndrome: vertical paresis stare, infringement of convergence of eyeglobes, particulate two-sided ptosis of eyelids. Horizontal locomotions of eyeglobes are not limited. The set of symptoms is observed at lesion upper colliculus roofs of mesencephalon and at tumor of epiphysis.

A red nucleus syndrome: intentional hemitremor, hemikiperkinesis;

Claoud syndrome (the inferior red nucleus syndrome): lesion of third cranial nerve (ptosis, divergent strabismus, mydriasis) on the side of locus; intentional hemitremor, hemiataxia and hypomyotonia - on the counter side.

Tegmental syndrome: on the side of the locus - ataxia, Bernard's syndrome, tremor, myoclonias; on counter to the locus side - hemianaesthesia, infringement colliculous reflexes (prompt rough locomotions in reply to unexpected visual and annoyances - starts - jerks).

Weber syndrome: a flaccid paralysis of third cranial nerve on the side of the locus and hemiparesis (hemiplegia) - on counter. The locus settles down in the establishment of brain leg and breaks pyramidal fibers.

Benedict's syndrome: paralysis of third cranial nerve on the side of locus (ptosis, divergent strabismus, mydriasis), intentional tremor and asteroids locomotions in extremities on counter to locus side. The locus damages fibers of third cranial nerve, red nucleus and cerebellar conductors suitable to it dental-rubral pathes.

Lesion of the pons the following alternating symptoms develop:

Mijar-Gubler syndrome: flaccid paralysis of mimic muscles on the side of locus and hemiplegia on counter side. The locus settles down in the base of bottom of the brain Pons, suffer nucleus n. facials and pyramidal fascicle.

Foville syndrome: flaccid paralysis of mimic muscles and outside direct muscle of eye (concurrent strabismus) on the side of locus, hemiplegia - on counter. This set of symptoms arises at lesion of bottom of the base of Pons Varolii. Are damaged pyramidal fascicle, nucleus facial and axons of abducent nerve.

Gasperini syndrome: a flaccid paralysis of abducent and facial nerve, weakening of audition, hypoesthesia in zone of trigeminal nerve on the side of locus and conduction hemianaesthesia on the counter side. Such symptoms educes at unilateral locus of cover of the Pons cerebri.

Brisso-Sikar syndrome is characterized by spastic stricture of mimic muscles on the side of lesion (hemispasm of facial muscles from irritation of facial nerve nucleus and spastic hemiparesis on counter to the locus side (lesion pyramidal system).

Raimon-Sestan syndrom is caused by combined lesion of medial longitudinal fascicle and Pons center of look, medial leg of cerebellum, medial loop and pyramidal path: are observed paresis a look aside the lesion locus, ataxia, choroatetoid hyperkinesia - on the side of the locus; and contralateral spastic hemiparesis and hemianaesthesia.

Graine: disorder of surface sensitivity on the face for segmentary type on the side of locus, contralateral hemianaesthesia of the surface sensitivity on trunk and extremities (a lesion of nucleus V cranial nerves and spinnothalamic path).

Medulla syndromes:

Avellis syndrome: flaccid paralysis of half of tongue, soft palate and vocal cords (IX, X, XII cranial nerves) on the side of the locus and hemiplegia - on counter. Educes at the locus in one half of medulla.

Jackson syndrome: the flaccid paralysis of muscles of tongue on the side of the locus and central paralysis of counter extremities arises at lesion of one pyramid of mesencephalon and a root of XII cranial nerves.

Wallenberg syndrome: lesion of vagus nerve on the side of the locus (unilateral paralysis of soft palate, vocal cords, dysphagia). On same side Horner's syndrome, ataxia of cerebellar type, anesthesia of the face, dissociated anesthesia on the counter side - an alternating hemianaesthesia. The symptoms arise at infringement of circulation in an inferior back cerebellar artery.

Schmidt's syndrome: on the side of the locus paresis of vocal cords, soft palate, trapezoid and sternocleidomastoid muscle; on counter - spastic hemiparesis, i.e. nucleus and fibers IX, X, XI, XII cranial nerves and pyramidal systems are damaged.

Babynsky – Naghotta syndrome on the side of the locus - cerebellar signs (ataxia, nystagmus, asynergia), Horner syndrome, hyperthermia; contralateral spastic hemiparesis, dissociated hemianaesthesia (drops out pain and a thermoesthesia).

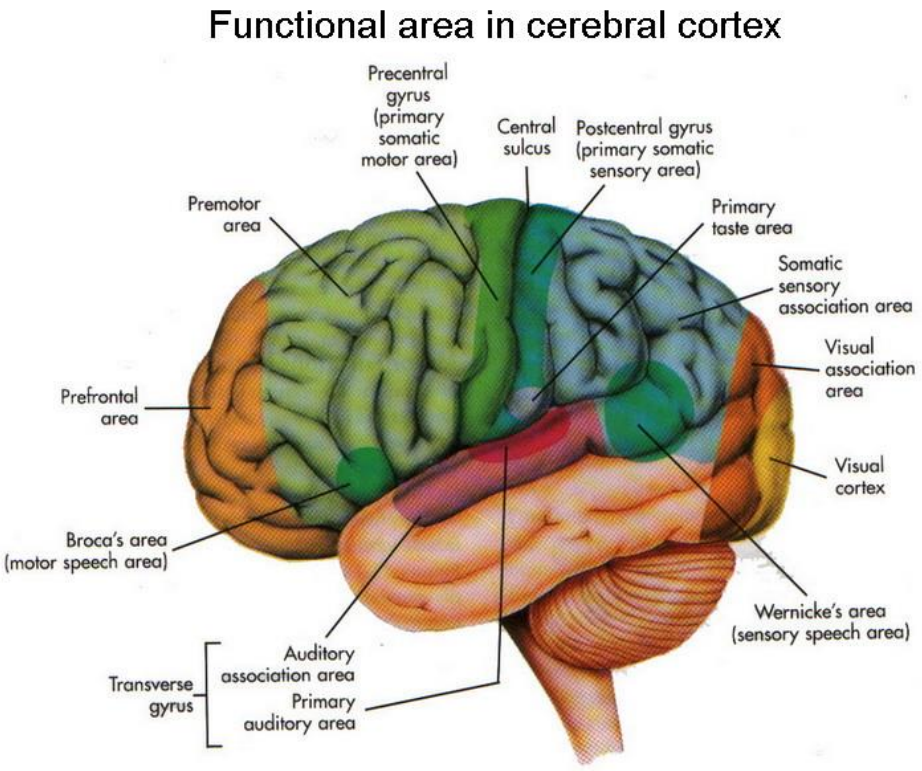
Two-sided lesion of nucleus and roots IX, X and XII cranial nerves produces bulbar paralysis. It is characterized by infringement of swallowing, hit of fluid nutrition in a nose, change of voice sonority, an aphonia, appearance of nasal shade of speech, a dysarthria. Are observed atrophy and fascicular twitching of tongue muscles. The gag reflex disappears. This syndrome arises at vascular and some degenerative diseases (lateral sclerosis, syringobulbia).

Pseudobulbar paralysis is central paralysis of muscles, innervated by IX, X, XII cranial nerves. Educes at two-sided lesion of corticonuclear pathes. The locuses settle down at different levels above myelencephalon, including in brainstem. Clinical exhibiting are similar to those at bulbar paralysis (infringement of swallowing, nasal shade of voice, dysarthria). At a pseudobulbar paralysis there are reflexes of oral automatism, violent laughter and crying. Attributes of lesion of

peripheral neuron (an atrophy, fascicular twitching, etc.) miss. The set of symptoms is linked to vascular lesions of brain more often.

At the pathological locuses outside of brainstem can suffer a little bit close posed nerves, there are characteristic syndromes. Among them it is important to note syndrome of pontocerrebelar angle - a lesion of acoustical, facial and trigeminal nerves.

SYNDROMES OF THE CEREBRAL COTTEX LESION



Picture 3

Aphasia

Left-hemisphere dominance for speech and language applies to more than 95% of all populations studied. Right-hemisphere dominance in a right-handed person is rare enough to prompt case reports in the literature. Most left-handed persons show some disturbance in speech and language from either left- or right-hemisphere lesions, making predictions for hemisphere dominance for left-handed persons difficult to predict on an individual basis. The most predictable site for disturbances in speech and language are the regions in and bordering on the sylvian fissure of the hemisphere controlling the hand preferred for skilled movements. The farther from this zone that the lesion occurs, the less the lesion disturbs speech and language. The disturbances in speech and language resulting from a lesion form a group of disorders known as the aphasias.

Motor aphasias

An acute focal lesion (the most frequent and best known being an infarct) involving any portion of the insula or the individual gyrus forming the upper banks of the opercular cortex (from the antero-inferior frontal region to the anterior parietal) acutely disrupts the ac-

quired skills involving the oropharyngeal, laryngeal, and respiratory systems that mediate speech, causing mutism. Writing may be preserved, although it is usually confined to a few simple words. Comprehension of words heard or seen is generally intact because these functions are largely subserved by posterior regions. The speech that emerges within minutes or days of the onset of motor aphasia consists mostly of crude vowels (dysphonia) and poorly articulated consonants (dysarthria). Disturbed coordination (dyspraxia) of speaking and breathing alters the rhythm of speech (dysprosody). This faulty intonation, stress, and phrasing of words and sentences is known collectively as speech dyspraxia. The language conveyed through this speech is usually only slightly disturbed, but the grammatic forms used in speaking or writing are sometimes simplified.

The more anterior that the lesion is along the operculum, the more speech dyspraxia predominates, especially with involvement of the inferior frontal region (Broca area) located adjacent to the sensorimotor cortex. When the sensorimotor cortex itself is affected, dysarthria and dysphonia are more prominent than dysprosody and dyspraxia. The errors in pronunciation may make it impossible to understand the language conveyed by the patient's speech, but they are not, strictly speaking, a language disorder.

When an acute lesion occurs more posteriorly along the sylvian operculum, the precise sensorimotor control over the positioning of the oropharynx may be impaired, causing unusual mispronunciations as well as mild dysphasia. The disturbed pronunciation is not simple dysarthria. Instead, the faulty oropharyngeal positioning yield sounds that differ from those intended (e.g., “dip” is said instead of “top”). The errors, analogous to the typing errors of a novice unfamiliar with the typewriter keyboard, are called literal paraphasias. The listener may mistake the utterances as language errors (paraphasias) or may be impressed with some of the genuine paraphasias and give the condition the name conduction aphasia (see the following). The patient's comprehension is intact despite the disordered pronunciation.

Stroke is the most common cause of acute lesions. The arrangement of the individual branches of the upper division of the sylvian artery favors the wide variety of focal embolic obstructions that produce this remarkable array of syndromes. The more specific that the speech abnormality is, the more limited is the focal infarction. Because the

sensorimotor cortex is part of the same arterial supply of the upper division of the middle cerebral artery, the larger infarcts and other disorders such as basal ganglia hemorrhages, abscesses, large tumors, and acute encephalitis usually cause accompanying contralateral hemiparesis and hemisensory syndromes, making the diagnosis of perisylvian disease fairly easy. One disorder, known as primary progressive aphasia, appears to be an unusual form of atrophy, causing mainly a relentless decline in speech and language function without the accompanying motor, sensory, visual, or other clinical evidence of a large lesion affecting the main pathways serving these functions.

For speech and language, the smaller and more superficial that the injury is, the briefer and less severe is the disruption. Rapid improvement occurs even when the lesion involves sites classically considered to cause permanent speech and language disturbances, such as the foot of the third frontal gyrus (Broca area). The larger the acute lesion, the more evident is dysphasia and the longer is the delay before speech improves. In larger sylvian lesions, dysphasia is evident in disordered grammar, especially when tests involve single letters, spelling, and subtleties of syntax. Problems with syntax occur not only in speaking and writing but also in attempts to comprehend the meaning of words heard or seen. For example, the word “ear” is responded to more reliably than is “are,” “cat” more than “act,” and “eye” more than “I.” The language content of spontaneously uttered sentences is condensed, missing many of the filler words, causing telegraphic speech, or agrammatism. Agrammatism is an important sign of a major lesion of the operculum and insula. When the causative lesion involves many gyri, as with large infarcts, hemorrhages, and neoplasms or abscesses large enough to produce unilateral weakness, the reduction of both speech and comprehension is profound and is called total aphasia. Within weeks or months in cases of infarction and hemorrhage, comprehension improves, especially for nongrammatic forms, and speaking and writing seem to be affected more than listening and reading. This last syndrome, in which dysphasia is most evident in speaking and writing, is known as motor aphasia; the eponym Broca aphasia is often used. This syndrome emerges from an initial total aphasia as a late residual. It is not the usual acute syndrome of a circumscribed infarction, even when the lesion is confined to the pars opercularis of the inferior frontal gyrus (Broca area).

Sensory aphasias

A different set of acute symptoms follows acute focal lesions of the posterior half of the temporal lobe and the posterior parietal and lateral occipital regions. Infarction is also the usual cause of the discrete syndromes, while hemorrhage, epilepsy, and acute encephalitis may account for sudden major syndromes. Even large lesions in these areas are usually far enough removed from the sensorimotor cortex so that hemiparesis and speech disturbances (e.g., dysprosody, dysarthria, or mutism) are only occasionally part of the clinical picture.

In patients with large posterior lesions, the effects are almost the reverse of the insular-opercular syndromes: Syntax is better preserved than semantics; speech is filled with small grammatic words, but the predicative words (i.e., words that contain the essence of the message) are omitted or distorted. Patients vocalize easily, engage in simple conversational exchanges, and even appear to be making an effort to communicate; however, little meaning is conveyed in the partial phrases, disjointed clauses, and incomplete sentences. In the most severe form, speech is incomprehensible gibberish. Errors take the form of words that fail to occur (omissions), are mispronounced as similar-sounding words (literal paraphasias), or are replaced by others that have a similar meaning (verbal paraphasias). A similar disturbance affects understanding words heard or seen. These language disturbances may require prolonged conversation to be revealed in mild cases. Because this disturbance in language contrasts with motor aphasia, it is often labeled as sensory aphasia, or Wernicke aphasia, but neither syndrome is purely motor or sensory.

The posterior portions of the brain are more compact than the anterior portions. As a result, large infarctions or mass lesions from hemorrhage, abscess, encephalitis, or brain tumors in the posterior brain tend to cause similar clinical disorders with few variations in syndrome type. Contralateral hemianopia usually implies a deep lesion. When hemianopia persists for longer than about 1 week, the aphasia is likely to persist.

Highly focal lesions are uncommon and, when present, usually mean focal infarction. Those limited to the posterior temporal lobe usually produce only a part of the larger syndrome of sensory aphasia. Speech and language are only slightly disturbed, reading for comprehension may pass for normal, but auditory comprehension of language

is grossly defective. This syndrome was classically known as pure word deafness. Patients with this disorder also usually reveal verbal paraphasias in spontaneous speech and disturbed silent reading comprehension. This syndrome might be better named the auditory form of sensory aphasia. It has a good prognosis, and useful clinical improvement occurs within weeks; some patients are almost normal.

A similarly restricted dysphasia may affect reading and writing, more so than auditory comprehension, because of a more posteriorly placed focal lesion that damages the posterior parietal and lateral occipital regions. Reading comprehension and writing morphology are strikingly abnormal. This syndrome has traditionally been known as alexia with agraphia, but spoken language and auditory comprehension are also disturbed (although less than reading and writing). A better label might be the visual form of sensory aphasia. It also has a good prognosis.

The more limited auditory and visual forms of Wernicke aphasia are rarely produced by mass lesions from any cause and tend to blend in larger lesions. Whether the major syndrome of sensory aphasia is a unified disturbance or a synergistic result of several separate disorders has not been determined.

Amnestic aphasia

Anomia or its more limited form dysnomia is the term applied to errors in tests of naming. Analysis requires special consideration because the mere occurrence of naming errors is of less diagnostic importance than is the type of error made. In all major aphasic syndromes, errors in language production cause defective naming (dysnomia), taking the form of paraphasias of the literal (e.g., “flikt” for “flight”) or verbal (e.g., “jump” for “flight”) type. For this reason, it is not usually of diagnostic value to focus a clinical examination on dysnomias alone, as they have little value as signs of focal brain disease.

A pattern known as amnestic dysnomia has a greater localizing value. Patients act as though the name has been forgotten and may give functional descriptions instead. Invoking lame excuses, testimonials of prowess, claims of irrelevance, or impatience, patients seem unaware that the amnestic dysnomia is a sign of disease. The disturbance is common enough in normal individuals, but in those with disease it is prominent enough to interfere with conversation. Amnestic aphasia, when fully developed, is usually the result of disease of the

deep temporal lobe gray and white matter. A frequent cause is Alzheimer disease, in which atrophy of the deep temporal lobe occurs early, and forgetfulness for names may be erroneously attributed to old age by the family. Identical symptoms may occur in the early stages of evolution of mass lesions from neoplasms or abscess but are rarely a sign of infarction in the deep temporal lobe. Other disturbances in language, such as those involving grammar, reading aloud, spelling, or writing, are usually absent, unless the responsible lesion encroaches on the adjacent temporal parietal or sylvian regions. When due to a mass lesion, the disturbance often evolves into the full syndrome of Wernicke aphasia.

Apraxia

The term apraxia (properly known as dyspraxia because the disorder is rarely complete) refers to disturbances in the execution of learned movements other than those disturbances caused by any coexisting weakness. These disorders are broadly considered to be the body-movement equivalents of the dysphasia and, like them, have classically been categorized into motor, sensory, and conduction forms.

Limb-kinetic or innervatory apraxia

This motor form of dyspraxia occurs as part of the syndrome of paresis caused by a cerebral lesion. Attempts to use the involved limbs reveal a disturbance in movement beyond that accounted for simply by weakness. Because attempted movements are disorganized, patients appear clumsy or unfamiliar with the movements called for in tasks such as writing or using utensils. Although difficult to demonstrate and easily overlooked in the presence of the more obvious weakness, innervatory dyspraxia is a useful sign to elicit because it indicates that the lesion causing the hemiparesis involves the cerebrum, presumably including the premotor region and other association systems. Dyspraxias of this type are thought to be caused by a lesion involving the cerebral surface or the immediately adjacent white matter.

Ideational apraxia

Ideational dyspraxia is a different type of disorder altogether. Movements of affected body parts appear to suffer from the absence of a basic plan, although many spontaneous actions are easily carried out. This disorder is believed to be analogous to sensory aphasia

(which features a breakdown of language organization despite continued utterance of individual words). The term is apparently derived from the simplistic notion that the lesion disrupts the brain region containing the motor plans for the chain of individual movements involved in complex behaviors such as feeding, dressing, or bathing. To the observer, patients appear uncertain about what to do next and may be misdiagnosed as confused. The lesion causing ideational dyspraxia is usually in the posterior half of the dominant hemisphere. The coexisting sensory aphasia often directs diagnostic attention away from the dyspraxia, which, like innervatory dyspraxia, is only rarely prominent enough to result in separate clinical recognition.

Ideomotor apraxia

This form of dyspraxia is frequently encountered. The term derives from the notion that a lesion disrupts the connections between the region of the brain containing ideas and the region involved in the execution of movements. The disturbance is analogous to conduction aphasia: Motor behavior is intact when executed spontaneously, but faulty when attempted in response to verbal command. For movements to be executed by the nondominant hemisphere in response to dictated commands processed by the dominant hemisphere, the lesion could involve the presumed white-matter pathways through the dominant hemisphere to its motor cortex, the motor cortex itself, or the white matter connecting to the motor cortex of the nondominant hemisphere through the corpus callosum. Because so many presumed pathways are involved, ideomotor dyspraxia is common. The syndrome is most frequently encountered in the limbs served by the nondominant hemisphere when the lesion involves the convexity of the dominant hemisphere. Concomitant right hemiparesis and dysphasia, usually of the motor type, often occupy the physician's attention so that the ideomotor dyspraxia of the nondominant limbs passes without notice. Dysphasia may make it impossible to determine whether ideomotor dyspraxia is present, but, when mild, dyspraxia can be demonstrated by showing that patients cannot make movements on command, although they can mimic the behavior demonstrated by the examiner and execute it spontaneously at other times. The disturbances are most apparent for movements that involve the appendages (e.g., fingers, hands) or oropharynx. Axial and trunk movements are often spared.

ATAXIAS. THE HYPERKYNETIC SYNDROME, HYPOKYNETIC SYNDROME

Ataxias

Ataxias - disorders of coordination - are described as a series of syndromes, which are characterized by nuclear (basic) and additional disorders. Allocate cerebellar, vestibular, sensitive, cortical, conductive, psychogenic ataxia. The function of the cerebellum is the reflex maintenance of muscle tone, balance, coordination and synergy of movements. With damage to the cerebellum, a number of motor disorders of atactic and asynergic nature occur.

1. Gait disorder. Atactic-cerebellar, or the so-called "drunk", gait is the result of not only imbalance, but also asynergy. The patient walks with his legs wide apart and staggering, which is especially pronounced when turning. Deviation to the side when walking, and in severe cases, a fall, are observed more often in the direction of the cerebellar lesion. 2. Intentional trembling is observed during movement and is absent at rest. It is detected most sharply at the end of the movement and is examined in the hands with the help of a finger-nose test, and in the legs with the help of a heel-knee test. The patient is given the task with closed eyes to hit the tip of his nose with his index finger; the closer to the target, the more clearly and sharply the trembling of the finger or the entire hand and arm is detected. Even better, intentional trembling in the hands is detected in a different way: the patient touches the hammer with his index finger or the finger of the examiner with open eyes, and the position of the hammer changes several times. A heel-knee test is performed in a lying patient, who is asked to first raise his leg high, then touch the heel of the other knee and draw the heel down the front surface of the leg. 3. Nystagmus (twitching of the eyeballs when retracting them), observed with damage to the cerebellum, is more often horizontal than vertical or rotatory; an indication that it is more pronounced when looking at the affected side is unreliable. 4. Adiadochokinesis is detected when trying to quickly perform alternately opposite movements. So, patients fail to quickly change pronation to supination of the hand: awkward, incorrect movements are obtained. 5. Dysmetria, or, more precisely, hypermetria of movements, can be easily detected by the following

method: the subject is asked to hold the hands with palms extended forward, with fingers apart; followed by an order to quickly turn the hands palms down; on the side where there are cerebellar disorders, this movement is made with excessive rotation of the hand. When offered to touch the heel of one leg to the knee of the other (in the supine position), the patient raises the legs above the knee and touches the thigh with the heel (hyperflexia phenomenon).

Missing, or missing, with the so-called “test of indications” is detected as follows: the patient is invited to hit the index finger 2-3 times with the finger of the examiner placed in front of him or in the hammer: after that, the patient closes his eyes and continues the same the task. In the hand, in which there are cerebellar disorders, there is a missed target, more often outwards. 7. Speech disorder is a particular manifestation of cerebellar movement disorder; speech loses its smoothness, becomes chanted, explosive, slowed down. 8. Muscle hypotension, which manifests itself in lethargy, flabbiness of muscles, in excessive excursion in the joints, is found in the study of passive movements. It may be accompanied by a decrease in tendon reflexes of the extremities. With lesions of the cerebellum, other symptoms may also be observed. a) Asynergy is expressed in a violation of the coordination of the work of a number of muscle groups necessary for the implementation of a particular movement. b) A symptom of the absence of a “reverse impulse”, which is also explained by hypotonia and a violation of antagonistic innervation; the patient holds his hand in front of him, bending it with force at the elbow joint, in which he is resisted; with a sudden cessation of resistance, the patient's hand strikes the chest with force. In a healthy person, this does not happen, since the rapid activation of the antagonists (extensors of the forearm) - the "reverse push" - prevents the blow. c) Handwriting disorder is a consequence of impaired coordination of fine movements and trembling; handwriting becomes uneven, lines zigzag, letters too large (megalography). d) Underestimation of the severity of the object held by the hand is a peculiar symptom observed on the side of the lesion. e) Dizziness is a fairly common symptom of acute cerebellar lesions. f) When the connections of the nucleus dentatus with the nucleus ruber are damaged, extrapyramidal hyperkinesis may occur; with damage to the lower olive or its connections with the nucleus dentatus, myoclonus of the tongue, pharynx, and soft palate is sometimes ob-

served. g) When the worm is affected, static and gait disorders prevail; when the hemispheres of the cerebellum are affected, the smoothness and accuracy of movements of the homolateral limbs (intentional trembling) are especially affected.

The hyperkinetic syndromes

The patient who presents with a hyperkinetic movement disorder experiences excessive movement. The patient may state that it interferes with the activities of daily living, or the patient may be unaware of the problem and the family may prompt the evaluation. The correct diagnosis depends on the character of the movement, that is, when it occurs, its speed, its location, and contributing factors. The more common disorders include dystonia, tremor, chorea, tics, myoclonus, and tardive dyskinesia.

Dystonia

Dystonia is a disorder consisting of intermittent or sustained, often painful, twisting, repetitive muscle spasms that may occur in one part of the body (focal dystonia) or throughout the entire body (generalized dystonia).

Initially, the movements may be triggered by a specific act, such as writing, and are made worse by movement of other parts of the body. There may be superimposed tremor or myoclonic jerks.

The patient may use a "sensory trick" - a tactile stimulus - that can decrease the muscle contractions. The movements may be painful.

Classification

Dystonia is classified by the part of the body affected (focal versus generalized dystonia), by the age of onset (adult-onset versus childhood-onset dystonia), and by contributing factors (idiopathic versus secondary dystonia).

Pathophysiology. Idiopathic dystonia is not associated with any particular brain lesion. Secondary dystonia is most often observed in patients with lesions in the basal ganglia, such as the putamen, and their connections with the thalamus and cortex.

Generalized dystonia. The spasms of generalized dystonia affect most of the body. They can manifest in one part of the body, particularly the foot, but rapidly spread to contiguous parts and usually involve the limbs, trunk, and neck. Generalized dystonia more common-

ly manifests among children and young adults. Onset is typically in the legs and spreads to contiguous body parts.

Focal dystonia is isolated to one part of the body

Dystonia can be idiopathic (primary) or have a known cause Secondary.

1. Idiopathic (primary) dystonia

Idiopathic dystonia has no known cause. Birth history is normal, and the examination findings are normal exifor dystonia. Ceruloplasmin and brain imaging are normal. Types of idiopathic dystonia include focal and generalized forms. Regardless of a focal or generalized presentation, dystonia can be genetic, such as autosomal dominant (DYT1), X-linked (Lubag syndrome), or sporadic.

2. Secondary dystonia. The most common known metabolic defect causing secondary dystonia is Wilson's disease.

3. Acquired dystonia occurs as a result of an injury, treatment, or other disease process. These can include

a. Prenatal injury resulting in an ischemic event manifesting as dystonia.

b. Exposure to toxins (carbon monoxide, manganese) resulting in structural changes to the basal ganglia.

c. Anoxic injury to the cerebral cortex or basal ganglia resulting in dystonic posturing.

d. Tardive syndrome from dopamine blockers (phenothiazine, metoclopramide). Tardive dystonia generally occurs during treatment or within 3 months of discontinuation of therapy.

e. Focal brain lesions (stroke, tumor, demyelinating, postinfectious, posttraumatic), regardless of the cause, can manifest as dystonia.

f. Peripheral nerve injury to the neck or limbs can result in dystonic posturing of that body part. Why this causes dystonia has not been determined.

g. Psychogenic dystonia remains a diagnosis of exclusion. Irregular spasms, unusual triggers, and bizarre postures may be clues.

Clinical manifestations of dystonia

1. Focal dystonia

a. Blepharospasm is a disorder that consists of uncontrollable involuntary spasms of the eyelids causing spontaneous closure. It often interferes with vision, resulting in functional blindness. It may be

worsened by bright light or stress.

b. Oromandibular dystonia consists of grimacing of the lower part of the face, usually involving the mouth, jaw, and platysma muscle. If associated with blepharospasm, it is called *Meige's syndrome.*

c. **Spasmodic torticollis** or cervical dystonia consists of intermittent, uncontrollable spasms of the neck muscles, often associated with severe pain. The neck may involuntarily turn, tilt, or rotate forward, sideways, or backward.

d. Spasmodic dysphonia involves only the vocal cords. There are two types of spasmodic dysphonia. With **adductor-type** spasmodic dysphonia, hyperadduction of the cords produces an intermittent strain and strangle quality to the voice. Often patients also report tightness in the throat during the spasms. With the more rare **abductor type** of spasmodic dysphonia, there is a whispering quality to the voice, similar to the movie star Marilyn Monroe's voice.

e. Occupational dystonia. Writer's cramp is the most common and most underdiagnosed form of limb dystonia. Dystonic posturing may be noticed in the hand or foot. Early in the course of the disease, the movement may be brought out by performing a specific task such as writing, typing, or playing a musical instrument. Examples of this include an auctioneer who has jaw dystonia only during an auction, a secretary who has dystonic hand cramps while typing, and a violinist who has finger spasms only while playing.

2.Hemidystonia involves one side of the body and almost always results from a focal lesion (vascular, neoplastic, or traumatic).

3.Generalized dystonia. Spasms occur in two or more limbs, and usually also in the trunk and neck. Symptoms usually begin in the legs and progressively involve other parts of the body.

Tremor

Tremor consists of rhythmic, oscillating movements of agonist and antagonist muscles. The movements are equal in amplitude and frequency. Symptoms are made worse by anxiety and disappear with sleep.

Classification

1.Physiologic tremor is a low-amplitude (8 to 12 Hz) tremor that is most prominent in outstretched hands and that under certain circumstances is present in all persons.

2.Essential tremor is a postural or action-involved tremor that

rarely is present at rest. The frequency usually is 4 to 12 Hz but may decrease with age. Data suggest that different anatomic locations (arm tremor only versus head and arm tremor versus isolated head tremor) may have clinically different presentations. Most subjects progressed slowly, but a small subgroup had a more rapid progression. The more rapid progression appeared to be associated with a higher age at onset and the presence of concomitant head and arm tremor.

3.Cerebellar tremor is most prominent in voluntary movements and has a frequency of 3 to 4 Hz. Patients perform poorly on finger-to-nose and heel-to-shin testing. The tremor may involve only the trunk in some patients.

4.Rest tremor (parkinsonian tremor) occurs at 3 to 7 Hz and is most obvious when the limb is fully supported and at rest. Rest tremor is reduced by action and intention.

Etiology

1.Physiologic tremor is exacerbated by excited mental states, metabolic endocrine derangements, fever, drugs (thyroid, lithium, (3-agonists, phynilline, and sodium valproate), alcohol withdrawal, and caffeine use.

2.Essential tremor

The cause of essential tremor is unclear. Most patients have strong family histories. The diagnosis can be confirmed by means of suppression of the through ingestion of a small amount of alcohol. Lack of response does exclude the diagnosis.

3. Cerebellar tremor is typically observed with a lack of feed-back of the bellum to the motor cortex. The cause of a Cerebellar trem-or include demyelinating disease (such as multiple sclerosis), a space-occupying lesion, or an ischemic, toxic, or infectious disorder.

4. Rest tremor is part of the clinical features of Parkinson's disease. However, some patients come to medical attention with this type of tremor and may not have other symptoms of Parkinson's disease, such as bradykinesia, postural instability, and cogwheel rigidity. The cause of a resting tremor is generally considered to be in the central nervous sys-tem (CNS), but the exact anatomic lesion is unknown.

Chorea

Chorea is hyperactive, fast, arrhythmic, often semipurposeful move-ment. It can affect the limbs, face, or trunk.

Huntington's disease is a progressive neurodegenerative disease

with autosomal dominant inheritance localized to chromosome 4. Patients with positive family histories often come to medical attention with chorea. The mean age at onset is 40 years, but the onset can be any time from childhood to old age. Some patients may notice problems with control of fine movement, dropping of objects, or incoordination before the onset of chorea.

Cognitive deficits manifest as problems of concentration, attention, and coordination of spatial motor acts rather than problems of memory. Subtle findings, such as changes in job performance or interests, may be revealed when the patient visits the physician. Memory problems often are short-term problems, and unlike patients with Alzheimer's disease, patients with end-stage Huntington's disease may retain recognition of family and familiar surroundings.

Other causes of chorea

- Pregnancy. Chorea gravidarum or chorea caused by hormone replacement in a pregnant patient should be investigated.
- Encephalitis
- Drugs. Levodopa, oral contraceptives, anticonvulsants, lithium
- Metabolic and autoimmune disorders. Consider systemic lupus erythematosus, antiphospholipid antibody syndrome, or lupus anticoagulant, thyrotoxicosis Sydenham's chorea, polycythemia rubra vera, and hypoparathyroidism.
- Infection. Lesions due to toxoplasmosis have been reported.

Hemiballismus

Definition. Hemiballismus is a rare disorder involving violent flinging of arm or leg on one side of the body.

Etiology. Caused by destruction of part of the contralateral subthalamic nucleus that results in disinhibition of the output of the globus pallidus. The cause I ally is hemorrhagic or ischemic infarction but also can be previous surgery or undiagnosed tumor.

Tics

Types of tic disorders

1.Motor tics are abrupt, simple motor tasks, such as rapid head jerks.

2.Vocal tics are repetitive vocalizations, such as grunting and throat cle

3.Complicated motor and vocal tics

a. Complicated motor tics are semipurposeful movements.

b. Complicated vocal tics are repeated words or sentences. They can made-up or obscene words.

4. Tourette's syndrome

Patients with Tourette's syndrome have multiple motor tics with one or more vocal tic. Onset of symptoms occurs before 21 years of age. The estimated prevalence of Tourette's syndrome is 1 to 10 cases per 10,000 persons. However, because the diagnosis of Tourette's syndrome can be missed in the mildest form, this may be a gross underestimation. Symptoms persist for at least 12 months.

Behavioral disturbances. Obsessive-compulsive disorder is characterized by repetitive, stereotyped behaviors or thoughts. Attention-deficit hyperactivity disorder (with or without hyperactivity) manifests as poor attention span, restlessness, poor concentration, and low impulse control. In addition to obsessive-compulsive disorder and attention-deficit hyperactivity disorder, some patients with Tourette's syndrome have problems with classroom learning and academics. Sleep disturbances include somnambulism, nightmares, insomnia, and restlessness. These disturbances may be related to treatment, environment, or superimposed psychiatric disease.

Etiology

1.Tics involve inherited changes in synaptic transmission.

2.The dopamine hypothesis involves both presynaptic and postsynaptic function. It has been proposed that Tourette's syndrome is caused by supersensitivity of the postsynaptic dopamine receptors, dopamine hyperinnervation, abnormal presynaptic function, or excessive phasic release of dopamine.

3.Tourette's syndrome is generally hereditary. The gene and the biochemical defect are unknown. There is strong evidence that monozygotic twins have a 86% concordance rate of Tourette's syndrome compared with a 20% rate among dizygotic twins. A gene has not been identified.

4.Other causes

a. Tics may be observed after head trauma, toxin exposure, and encephalitis.

b. Tics can occur in association with other primary neurologic disorders, such as Huntington's disease, Parkinson's disease, dystonia, and side effects of medications such as methyLPhenidate.

Myoclonus

Myoclonus is defined as sudden, brief, involuntary jerks or contractions, either rhythmic or irregular, of single muscles or groups of muscles. It can occur at rest or in response to touch, auditory, or visual stimulation.

Types of myoclonic disorders. Myoclonus can be divided into epileptic, nonepileptic, and inherited types.

1. Epileptic myoclonus comprises generally progressive degenerative disorders affecting the nervous system. Myoclonus can occur in association with ataxia, dementia, or other seizure types.

a. Progressive myoclonic epilepsy. Myoclonic and tonic-clonic seizures occur among patients with progressive neurologic decline.

Progressive myoclonic epilepsy is associated with ataxia and dementia.

b. Infection of the CNS

Creutzfeldt-Jakob disease manifests a rapid onset of dementia and associated neurologic findings of myoclonic jerks,

Subacute sclerosing panencephalitis. Myoclonus is preceded by intellectual decline, personality changes, ataxia, and hyperactive reflexes.

c. Drug-related conditions. Myoclonus has been found among patients treated with levodopa, bromocriptine, tricyclic antidepressants, and narcotics.

d. Toxic and metabolic conditions. Myoclonus can occur in a confused patient as either large rhythmic movements or small irregular jerks and may be stimulus induced. Myoclonic movements can be confused with seizures, particularly if the patient is acutely ill, and ruling out seizures may necessitate an EEG. The most common cause is severe renal or hepatic disease. Systemic infection or drug intoxication also can cause myoclonic jerks. In rare instances, heavy metal intoxication can cause myoclonus.

2. Nonepileptic myoclonus. Nonepileptic myoclonic disorders are nonprogressive. EEG correlation is found in some cases of epilepsia partialis continua and juvenile myoclonic epilepsy.

a. Action myoclonus is induced by a voluntary movement or stimulus, such as a loud noise, and is common after hypoxic injury.

b. Palatal myoclonus consists of regular, rhythmic movement of the palate that can spread to the throat, face, and diaphragm. Movement persists during sleep. The neuropathologic lesion involves the

red nucleus, the inferior olive, and the dentate nucleus. This lesion often is ischemic, but it can be neoplastic, inflammatory, or degenerative.

c. Segmental myoclonus may arise in an arm or leg secondary to peripheral nervous system or CNS trauma, infection, or inflammation. It also can accompany renal failure, neuropathy, or acquired immunodeficiency syndrome.

d. Sleep myoclonus occurs soon after going to or arousing from sleep. It can be confused with seizure, particularly among infants. This benign form of myoclonus can be difficult to differentiate from infantile spasms EEG findings are normal.

e. Epilepsia partialis continua manifests as regular myoclonic jerking associated with a cortical discharge and no change in level of consciousness.

f. Juvenile myoclonic epilepsy. Among children, jerks can precede seizure. A family history and abnormal EEG findings establish the diagnosis.

g. Opsoclonus-myoclonus among children

Neural crest tumors may be seen on chest radiograph, or chest CT may be needed. Elevated levels catecholamine metabolites are present in the urine.

3. Inherited myoclonus-dystonia is a new term used to describe cases of myoclonus that begin in the first two decades of life, are autosomal dominant with variable penetrance and little or no progression, are responsive to alcohol ingestion, and are associated with dystonia. This disorder has been linked to the site of the dopamine D2 receptor gene on chromosome 11 in some families, but the gene has not been located. In addition, other families with this syndrome do not link to this region of chromosome 11. Eight families have been linked to a region on chromosome 7q. The likelihood is that inherited myoclonus-dystonia is a phenotype with several genotypes depending on the family studied.

Hypokinetic syndromes

Hypokinesia is defined as a decrease in the normal amount, amplitude, or speed of automatic or volitional movements. The term **bradykinesia** often is used when the predominant movement abnor-

mality is slowness. The term akinesia sometimes is used to imply a severe reduction in the amount or amplitude of movement. In truth, it is rare for any of the three parameters of movement to be affected in isolation. Thus, a patient with bradykinesia typically manifests a decreased amount and amplitude of movement. The movement of patients with hypokinesia often is referred to as parkinsonian because bradykinesia is so common in Parkinson's disease. However, bradykinesia is only one of four cardinal features of Parkinson's disease, the others being rigidity, tremor, and postural imbalance. Bradykinesia in the absence of the other features is not suficient to make a diagnosis of Parkinson's disease. The term **parkinsonism** is used to condition characterized by one or more of these cardinal signs that clinically resembles idiopathic Parkinson's disease (IPD) but is histologically different and often accompanied by additional neurologic signs and symptoms.

Hypokinesia can be used to describe both slowed volitional movements, such as reaching for object, and automatic movements, such as eye blinking or arm swing while walking. When hypokinesia develops over a period of several months or longer, the patient and family members may be relatively unaware of the problem. A striking and remarkable decrease blink frequency often goes unnoticed until brought to the attention of the patient or family members. When hypokinesia begins to result in functional disability, patients become aware of problem in motor function, but rather than attribute it to the speed or amplitude of their movement, they more commonly describe the difficulty as "weakness." Through careful questing, the clinician can discern a history of weakness from that of hypokinesia. It is important to determine whether slowness or lack of movement is due to extrapyramidal system disorder (e.g., Parkinson's disease) or to certain psychiatric disorders (catatonia or severe depression). One final differentiation must be made from neuronal disorders producing severe stiffness with associated slowness of movement. Hypokinesia related to abnormalities of the motor system is seldom life threatening except extreme form, in which severe immobilization can result in serious complications such as pneumonia or pulmonary embolism. Yet hypokinesia always merits serious attention because it often results in considerable functional and social disability.

Etiology

1.Degenerative disorders of the basal ganglia.
2.Pharmacologic agents.
3.Vascular disorders.
4.Trauma.
5.Toxins.
6.Central nervous system infection.

The clinical manifestations of hypokinesia result from different combinations of reduction in the speed, frequency, and amplitude of spontaneous or automatic movement.

A. Hypomimia is a decrease in facial expression. The diminished range of facial response to emotional stimuli gives rise to the term **expressionless faces.** The reduced eye blink rate results in an appearance resembling a constant stare.

B. Diminished automatic movement is noticeable as a decrease in gesticulation and head movement during conversation, a reduction in the automatic repositioning of limbs while sitting in a chair or reclining in bed, and as a decrease the amplitude of arm swing while walking. In severe hypokinesia, affected arms may not swing at all but be held in a hemiflexed posture across the front of the trunk. Among patients with asymmetric hypokinesia, as is often the case Parkinson's disease, reduction in arm swing is greatest on the more involved side.

C. Impairment of repetitive movements is particularly prominent among patients with hypokinesia. The patient may state that activities such as hand writing or buttoning a shirt are particularly difficult. Not only are repetitive movements performed slowly, but also the amplitude of each successive movement typically becomes progressively smaller. This may account for the progressively smaller letters (micrographia) seen when a hypokinetic patient is asked to write a long sentence or for increasing difficulty with the successive fine movements required to successfully place a button through a button hole.

D. Impaired initiation of movement manifests as difficulty in arising or hesitancy in taking the first step while attempting to walk. Many pat: with Parkinson's disease have difficulty initiating two motor acts simultaneously such as standing up and shaking hands.

E. Freezing is a sudden involuntary cessation of a motor act, usually walking. This phenomenon is confined to basal disorders. Freez-

ing can occur spontaneously or be provoked by external circumstances, such as attempting to turn in mid gait or pass through a narrow space such as a doorway. Emotional stimuli, including anger or fear, can provoke freezing, as can the prospect of entering a room filled with people. A variety of sensory or motor tricks such as marching to a cadence are effective in overcoming freezing.

F. Hypophonia is characterized by diminished amplitude and inflection of speech. In its most severe form, it results in a muffled pattern of articulation.

Clinical findings of parkinsonism

1.Gait and posture should be evaluated by having the patient walk a distance of at least 20 feet (6 m) in an area free of obstacles. Patients with parkinsonism have a reduced length of stride. The arms do not swing and may be held flexed across the front of the trunk. The upright posture is commonly flexed in IPD. There may be difficulty in initiating gait, and turns may be accomplished with several small steps or with the body moving as a single unit (en bloc turning). The gait in IPD occasionally is characterized by progressively more rapid, small steps as the body leans forward (festination). On the other hand, the patient's feet may "freeze" in mid gait.

2.Rising from a chair is tested by asking the patient to rise with armscrossed in front of the body to prevent pushing off. A patient with hypokinesia may need several attempts to succeed or may be totally unable to rise without using the arms. If the patient is unable to rise without assistance, a judgment must be made whether the cause is weakness (which can be tested independently) or bradykinesia.

3.Postural reflexes are evaluated by asking the patient to establish a comfortable base, with feet slightly apart. While standing behind the patient, the examiner applies a brisk backward sternal perturbation. A normal response is to take a corrective step backward to prevent falling. When postural reflexes are impaired, more than one step is needed before balance is reestablished. When postural reflexes are absent, the patient continues to reel backward and falls if not stopped by the examiner.

4.**Rigidity.** If rigidity is present, it must be determined whether it is predominant in axial muscles (neck or trunk), in the

limbs, or equally severe both. Increased resistance to passive movement of the involved body part is easily appreciated when rigidity is severe. When subtle, rigidity can be reinforced by asking the patient to alternately open and close the first of the hand on the side opposite of the arm or leg being tested. The presence of tremor in the limb demonstrating rigidity gives rise to a ratchet-like sensation referred to as **cogwheel rigidity.**

5.Tremor may appear in one or more forms among patients with parkinsonism.

a. Resting tremor, the hallmark of IPD and also present in some other forms of parkinsonism, is most common in the hands and occurs to a slightly lesser extent in the lower extremities and mandible. Rest tremor rarely involves the head and never affects the voice. It appears at a frequency of 4 to 5 Hz and often is at least temporarily extinguished by volitional movement. Because it is well known that rest tremor is enhanced by stress or anxiety, a subtle tremor can be uncovered by asking the patient to perform difficult mental arithmetic, a mildly stressful task. Rest tremor is the hallmark of IPD, and its absence casts doubt on the diagnosis but certainly does not rule it out.

b. Action tremor can be present in Parkinson's disease as well as in other parkinsonian syndromes, especially those associated with cerebellar dysfunction. It can be present as a **postural tremor** while the arms are out stretched in front of the patient or as a **kinetic tremor** while the patient is performing a task such as the finger-to-nose test. Postural tremor alone, in the absence of parkinsonian signs, suggests a diagnosis of essential tremor.

c. Positional tremor. Some tremors are particularly prominent when the involved body part is placed in a specific position. The **wingbeating tremor** of Wilson's disease is an example of this phenomenon. This tremor is noticed when the arms are abducted at the shoulders while flexed at the elbow.

6.Bradykinesia can be documented by simply observing the speed, amplitude, and amount of ordinary movements made by the patient, such as gestures or shifting of body position. Specific tasks such as the finger-to-nose test also provide an opportunity to observe the speed of movement. Repetitive motion tasks such as tapping the index finger against the thumb demonstrate slowness

of movement and a progressive loss of amplitude as the movement is repeated.

7.Facial expression. The diminished facial expression typical of IPD is characterized by a constant neutral countenance with infrequent eye blinking. A **fixed facial expression,** often present in progressive supranuclear palsy, consists of an unchanging expression, such as surprise in which the forehead may be furrowed, the eyelids retracted, and the nasolabial folds deepened. **Myerson's sign** is present in Parkinson's disease and a variety of other basal ganglia disorders. It consists of persistent reflex blinking to repetitive finger taps applied to the glabella just superior to the bridge of the nose. Among healthy persons, there is rapid habituation to this stimulus, so that no blinking occurs after the fourth or fifth tap.

COMA AND IMPAUIRED CONSCIOUS LEVEL

In hospital neurology, the clinical analysis of unresponsive and comatose patients becomes a practical necessity. There is always an urgency about such medical problems—a need to determine the underlying disease process and the direction in which it is evolving and to protect the brain against more serious or irreversible damage. When called upon, the physician must therefore be prepared to implement a rapid, systematic investigation of the comatose patient; the need for prompt therapeutic and diagnostic action allows no time for deliberate, leisurely investigation.

The **terms consciousness, confusion, stupor, unconsciousness, and coma** have been endowed with so many different meanings that it is almost impossible to avoid ambiguity in their usage. They are not strictly medical terms but literary, philosophic, and psychological ones as well. The word consciousness is the most difficult of all. William James once remarked that everyone knows what consciousness is until he attempts to define it. To the psychologist, consciousness denotes a state of continuous awareness of one's self and environment. Knowledge of self includes all “feelings, attitudes and emotions, impulses, volitions, and the active or striving aspects of conduct,” in short, an awareness of all one's own mental functioning, particularly of the cognitive processes, and their relation to past memories and experience. These can be judged only by the patient's verbal account of his introspections and, indirectly, by his actions. Physicians, being more practical and objective for the most part, give greater credence to the patient's behavior and reactions to overt stimuli than to what the patient says. For this reason they usually give the term consciousness its commonest and simplest operational meaning—namely, the state of the patient's awareness of self and environment and his responsiveness to external stimulation and inner need. This narrow definition has another advantage in that unconsciousness has the opposite meaning—a state of unawareness of self and environment or a suspension of those mental activities by which people are made aware of themselves and their environment, coupled with a diminished responsiveness to environmental stimuli. Some authors make a distinction between the level of consciousness—meaning the state of arousal or the degree of variation from normal alertness as judged by the appearance

of facial muscles, fixity of gaze, and body posture—and the content of consciousness, i.e., the quality and coherence of thought and behavior. For medical purposes, the loss of normal arousal is by far the more important and dramatic aspect of disordered consciousness and the one identified by laypersons and physicians as being the central issue in coma. To add to the ambiguity, psychoanalysts have given the word unconscious a still different meaning; for them it is a repository of impulses and memories of previous experiences that cannot immediately be recalled to the conscious mind.

Description of States of Normal and Impaired Consciousness

Normal Consciousness This is the condition of the normal person when awake. In this state the individual is fully responsive to stimuli and indicates by his behavior and speech the same awareness of self and environment as that of the examiner. This normal state may fluctuate during the day from one of keen alertness or deep concentration with a marked constriction of the field of attention to one of mild general inattentiveness. From this state, the normal individual can be brought immediately to a state of full alertness and function.

Confusion In this condition the patient does not take into account all elements of his immediate environment. This state always implies a degree of imperceptiveness and distractibility, referred to traditionally as “clouding of the sensorium.” The term confusion lacks precision, but in general it denotes an inability to think with customary speed and clarity, usually marked by some degree of inattentiveness and disorientation. Here the difficulty is to define thinking, a term that refers variably to problem solving or to coherence of ideas and formation of memories. Confusion results most often from a process that influences the brain globally, such as a toxic or metabolic disturbance or a dementia. Any condition that causes drowsiness or stupor, including the natural state that comes from sleep deprivation, results in some degradation of mental performance and inattentiveness. A confusional state can also accompany focal cerebral disease in various locations, particularly in the right hemisphere, or result from disorders that also disturb language, memory, or visuospatial orientation, but these are readily distinguished from the global confusional state.

The **mildest degree of confusion** may be so slight that it can be overlooked unless the examiner searches for deviations from the patient's normal behavior and liveliness of conversation. The patient may

even be roughly oriented as to time and place, with only occasional irrelevant remarks betraying an incoherence of thinking. **Moderately confused** persons can carry on a simple conversation for short periods of time, but their thinking is slow and incoherent, their responses are inconsistent, and they are unable to stay on one topic and to inhibit inappropriate responses. Usually they are disoriented in time and place. They are distractible and at the mercy of every stimulus. Periods of irritability and excitability may alternate with drowsiness and diminished vigilance. Movements are often tremulous, jerky, and ineffectual. **Severely confused** and inattentive persons are usually unable to do more than carry out the simplest commands, and these only inconsistently and in brief sequence. Few if any thought processes are in operation. Their speech is usually limited to a few words or phrases; infrequently these individuals are voluble. They are unaware of much that goes on around them, are often disoriented in time and place, do not grasp their immediate situation, and may misidentify people or objects. Illusions may lead to fear or agitation. Occasionally, hallucinatory or delusional experiences impart a psychotic cast to the clinical picture, obscuring the deficit in attention.

Drowsiness and Stupor In these states, mental, speech, and physical activity are reduced. Drowsiness denotes an inability to sustain a wakeful state without the application of external stimuli. Inattentiveness and mild confusion are the rule, both improving with arousal. The lids droop without closing completely; there may be snoring, the jaw and limb muscles are slack, and the limbs are relaxed. This state is indistinguishable from light sleep, with slow arousal elicited by speaking to the patient or applying a tactile stimulus.

Stupor describes a patient who can be roused only by vigorous and repeated stimuli, at which time he opens his eyes, looks at the examiner, and does not appear to be unconscious; response to spoken commands is either absent or slow and inadequate. Restless or stereotyped motor activity is common in stuporous patients and they do not shift position as frequently as patients who are only drowsy. When left unstipulated, they quickly drift back into a sleep-like state. The eyes move outward and upward, a feature that is shared with sleep (see further on). Tendon and plantar reflexes and breathing pattern may or may not be altered, depending on how the underlying disease has affected the nervous system.

Coma The patient who appears to be asleep and is at the same time incapable of being aroused by external stimuli or inner need is in a state of coma. There are variations in the degree of coma; in its deepest stages, no reaction of any kind is obtainable: corneal, pupillary, pharyngeal, tendon, and plantar reflexes are in abeyance, and tone in the limb muscles is diminished. With lesser degrees of coma, pupillary reactions, reflex ocular movements, and corneal and other brainstem reflexes are preserved in varying degree, and muscle tone in the limbs may be increased. Respiration may be slow or rapid, periodic, or deranged in other ways (see further on). In still lighter stages, sometimes referred to by the ambiguous term semicoma, most of the above reflexes can be elicited, and the plantar reflexes may be either flexor or extensor (Babinski sign). Moreover, vigorous stimulation of the patient or distention of the bladder may cause a stirring or moaning and a quickening of respiration. These physical signs vary somewhat depending on the cause of coma. For example, patients with alcoholic coma may be areflexic and unresponsive to noxious stimuli, even when respiration and other vital functions are not threatened. The depth of coma and stupor, when compared in serial examinations, is most useful in assessing the direction in which the disease is evolving.

Relationship of Sleep to Coma Persons in sleep give little evidence of being aware of themselves or their environment; in this respect they are unconscious. Sleep shares a number of other features with the pathologic states of drowsiness, stupor, and coma. These include yawning, closure of the eyelids, cessation of blinking and swallowing, upward deviation or divergence or roving movements of the eyes, loss of muscular tone, decrease or loss of tendon reflexes, and even the presence of Babinski signs and irregular respirations, sometimes Cheyne-Stokes in type. Upon being awakened from deep sleep, a normal person may be confused for a few moments, as every physician knows. Nevertheless, sleeping persons may still respond to unaccustomed stimuli and at times are capable of some mental activity in the form of dreams that leave traces of memory, thus differing from persons in stupor or coma. The most important difference, of course, is that persons in sleep, when stimulated, can be roused to normal consciousness. There are important physiologic differences as well. Cerebral oxygen uptake does not decrease during sleep, as it usually does in coma. Recordable electrical activity—electroencephalographic

(EEG) and cerebral evoked responses—and spontaneous motor activity differ in the two states. The anatomic basis for these differences is not clear.

The Persistent Vegetative State, Locked-in Syndrome, and Akinetic Mutism

With increasing refinements in the treatment of severe systemic diseases and cerebral injury, more and more patients who formerly would have died have survived for indefinite periods without regaining any meaningful mental function. For the first week or two after the cerebral injury, these patients are in a state of deep coma. Then they begin to open their eyes, at first in response to painful stimuli and later spontaneously and for increasingly prolonged periods. The patient may blink in response to threat or to light and intermittently the eyes move from side to side, seemingly following objects or fixating momentarily on the physician or a family member and giving the erroneous impression of recognition. Respiration may quicken in response to stimulation and certain automatisms—such as swallowing, bruxism, grimacing, grunting, and moaning—may be observed. However, the patient remains totally inattentive, does not speak, and shows no signs of awareness of the environment or inner need; responsiveness is limited to primitive postural and reflex movements of the limbs. In brief, there is arousal or wakefulness and alternating arousal-nonarousal cycles may be established, but the patient regains neither awareness nor purposeful behavior of any kind. This state is characterized by a number of EEG abnormalities. After global anoxic injury, the EEG tends to display the most profound abnormalities, even to the point of being isoelectric. However, predominantly low-amplitude delta-frequency background activity, burst suppression, widespread alpha and theta activity, an alpha coma pattern, and sleep spindles have all been described in this syndrome. Moreover, the transition from coma to a state of partial awakening is generally not marked by a change in the EEG pattern.

If lasting, the above described syndrome is most appropriately referred to as the persistent vegetative state or PVS. This term has gained wide acceptance and applies to the clinical situation whatever the underlying cause. The most common pathologic bases of this state are diffuse cerebral injury due to closed head trauma, widespread laminar necrosis of the cortex after cardiac arrest, and thalamic necrosis

from a number of causes. Occasionally, the most prominent changes are in the thalamic and subthalamic nuclei. It is noteworthy that a persistent vegetative state may also be the terminal phase of progressive degenerative processes such as Alzheimer disease and of Creutzfeldt-Jakob disease. The profound and widespread dysfunction of the cerebrum is reflected by extreme reductions in cerebral blood flow and metabolism, measured with positron emission tomography (PET) and other techniques.

Additional terms that have been used to describe this syndrome of preserved autonomic and respiratory function without cognition include apallic syndrome and neocortical death. A recent position paper has codified the features of the persistent vegetative state and suggests dropping a number of related ambiguous terms, although some, such as akinetic mutism, have a more specific neurologic meaning and still find use.

The term akinetic mutism has been applied to yet another group of patients who are silent and inert as a result of bilateral lesions of the anterior parts of the frontal lobes, leaving intact the motor and sensory pathways; the patient is profoundly apathetic, lacking to an extreme degree the psychic drive or impulse to action (abulia). The abulic patient registers most of what is happening about him and forms memories.

The psychiatric patient with catatonia appears unresponsive, simulating stupor, light coma, or the akinetic mute state. There are no signs of structural brain disease such as pupillary or reflex abnormalities. Oculocephalic responses are preserved as in the awake state, i.e. the eyes move concurrently as the head is turned. There is usually resistance to eye opening, and some patients display a waxy flexibility of passive limb movement that gives the examiner a feeling of bending a wax rod; there is also the retention for a long period of seemingly uncomfortable limb postures. Peculiar motor mannerisms or repetitive motions, seen in a number of these patients, may give the impression of seizures; choreiform jerking has been reported.

Brain Death, In the late 1950s European neurologists called attention to a state of coma in which the brain was irreversibly damaged and had ceased to function but pulmonary and cardiac function could still be maintained by artificial means. A Harvard Medical School committee, in 1968, called it brain death and established a set of clini-

cal criteria by which it could be recognized. The concept that a person is dead if the brain is dead and that death of the brain may precede the cessation of cardiac function has posed a number of important ethical, legal, and social problems as well as medical ones. The various aspects of brain death have been the subject of close study by several professional committees, which have for the most part confirmed the 1968 guidelines for determining that the brain is dead.

The central considerations in the diagnosis of brain death are (1) absence of cerebral functions (unreceptivity and unresponsivity); (2) absence of brainstem functions, including spontaneous respiration; and (3) irreversibility of the state. To these is usually added evidence of catastrophic brain damage (trauma, cardiac arrest, cerebral hemorrhage, etc.).

Classification of Coma and Differential Diagnosis

The demonstration of focal brain disease or of meningeal irritation with abnormalities of the CSF is of particular help in the differential diagnosis of coma and serves to divide the diseases that cause coma into three classes, as follows:

I. Diseases that cause no focal or lateralizing neurologic signs, usually with normal brainstem functions. CT scan and cellular content of the CSF are normal.

A. Intoxications: alcohol, barbiturates and other sedative drugs, opiates, etc.

B. Metabolic disturbances: anoxia, diabetic acidosis, uremia, hepatic failure, nonketotic hyperosmolar hyperglycemia, hypo- and hypernatremia, hypoglycemia, addisonian crisis, profound nutritional deficiency, thyroid states.

C. Severe systemic infections: pneumonia, peritonitis, typhoid fever, malaria, septicemia, Waterhouse-Friderichsen syndrome.

D. Circulatory collapse (shock) from any cause.

E. Postseizure states and convulsive and non-convulsive status epilepticus

F. Hypertensive encephalopathy and eclampsia

G. Hyperthermia and hypothermia.

H. Concussion

I. Acute hydrocephalus

J. Late stages of certain degenerative diseases and Creutzfeldt-Jakob disease.

II. Diseases that cause meningeal irritation with or without fever, and with an excess of WBCs or RBCs in the CSF, usually without focal or lateralizing cerebral or brainstem signs. CT scanning or MRI, (which preferably should precede lumbar puncture) may be normal or abnormal.

A. Subarachnoid hemorrhage from ruptured aneurysm, arteriovenous malformation, occasionally trauma

B. Acute bacterial meningitis

C. Some forms of viral encephalitis

D. Neoplastic and parasitic meningitides.

III. Diseases that cause focal brainstem or lateralizing cerebral signs, with or without changes in the CSF. CT scanning and MRI are usually abnormal.

A. Hemispheric hemorrhage or massive infarction.

B. Brainstem infarction due to basilar artery thrombosis or embolism.

C .Brain abscess, subdural empyema.

D. Epidural and subdural hemorrhage and brain contusion.

E. Brain tumor

F. Cerebellar and pontine hemorrhage.

G. Miscellaneous: cortical vein thrombosis, some forms of viral encephalitis (herpes), focal embolic infarction due to bacterial endocarditis, acute hemorrhagic leukoencephalitis, disseminated (postinfectious) encephalomyelitis, intravascular lymphoma, thrombotic thrombocytopenic purpura, diffuse fat embolism, and others.

Using the clinical criteria outlined above, one can usually ascertain whether a given case of coma falls into one of these three categories. Concerning the group without focal or lateralizing or meningeal signs (which includes most of the metabolic encephalopathies, intoxications, concussion, and postseizure states), it must be kept in mind that residua from previous neurologic disease may confuse the clinical picture. Thus, an earlier hemiparesis from vascular disease or trauma may reassert itself in the course of uremic or hepatic coma with hypotension, hypoglycemia, diabetic acidosis, or following a seizure. In hypertensive encephalopathy, focal signs may also be present. Occasionally, for no understandable reason, one leg may seem to move less, one plantar reflex may be extensor, or seizures may be predominantly or entirely unilateral in a metabolic coma, particularly in the hyper-

glycemic–hyperosmolar state. Babinski signs and extensor rigidity, conventionally considered to be indicators of structural disease, do occur in profound intoxications with a number of agents.

With respect to the second group in the above classification, the signs of meningeal irritation (head retraction, stiffness of neck on forward bending, Kernig and Brudzinski signs) can usually be elicited in both bacterial meningitis and subarachnoid hemorrhage. However, in infants and in adults, if the coma is profound, stiff neck may be absent. In such cases the spinal fluid has to be examined in order to establish the diagnosis. In most cases of bacterial meningitis, the CSF pressure is not exceptionally high (usually less than 400 mmH2O). However, in cases associated with brain swelling, the CSF pressure is greatly elevated; the pupils become fixed and dilated, and there may be signs of compression of the brainstem with arrest of respiration. Patients in coma from ruptured aneurysms also have high CSF pressure; the CSF is overtly bloody and the blood is invariably visible in the CT scan throughout the basal cisterns and ventricles if the bleeding has been severe enough to cause coma.

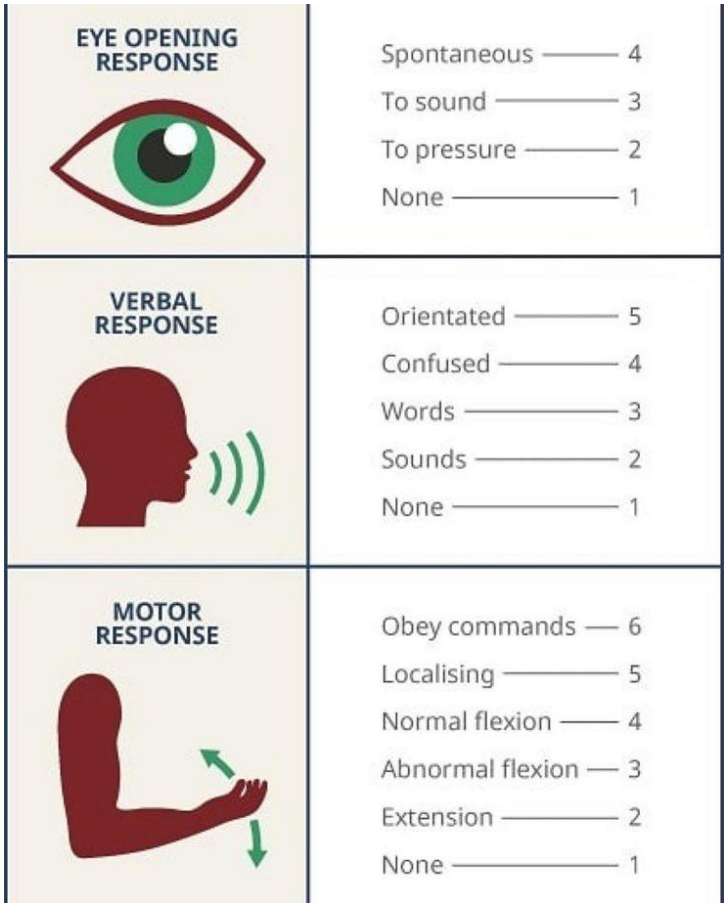
In the third group of patients, it is the focality of sensorimotor signs and the abnormal pupillary and ocular reflexes, postural states, and breathing patterns that provide the clues to serious structural lesions in the cerebral hemispheres and their effects upon segmental brainstem functions. As the brainstem effects become prominent, they may obscure earlier signs of cerebral disease.

It is worth emphasizing once more that hepatic, hypoglycemic, hyperglycemic, and hypoxic coma sometimes resemble coma due to brainstem lesions by causing asymmetrical motor signs, focal seizures, and decerebrate postures and that deep coma from drug intoxication may obliterate reflex eye movements. Also, certain structural lesions of the cerebral hemispheres are so diffuse as to produce a picture that simulates a metabolic disturbance; thrombotic thrombocytopenic purpura (TTP), fat embolism, and the late effects of global ischemia-anoxia are examples of such states. At other times they cause a diffuse encephalopathy with superimposed focal signs. Unilateral infarction due to anterior, middle, or posterior cerebral artery occlusion produces no more than drowsiness, as a rule; however, with massive unilateral infarction due to carotid artery occlusion, coma can occur if extensive brain edema and secondary tissue shift are associated. Edema of this

degree seldom develops before 12 or 24 h. Rapidly evolving hydrocephalus causes smallness of the pupils, rapid respiration, extensor rigidity of the legs, Babinski signs, and sometimes a loss of eye movements.

Finally, it should be restated that diagnosis has as its prime purpose the direction of therapy. The treatable causes of coma are: drug and alcohol intoxications, shock due to infection, cardiac failure, or systemic bleeding, epidural and subdural hematomas, brain abscess, bacterial and fungal meningitis, diabetic acidosis or hyperosmolar state, hypoglycemia, hypo- or hypernatremia, hepatic coma, uremia, status epilepticus, and hypertensive encephalopathy. Also treatable to a varying degree are uremia; putaminal and cerebellar hemorrhages, which can sometimes be evacuated successfully; edema from massive stroke, which may be ameliorated by hemicraniectomy; and hydrocephalus from any cause, which may respond to ventricular drainage.

Glasgow Coma Scale (GCS)



Picture 6

MENINGISM. RAISED INTRACRANIAL PRESSURE. CEREBROSPINAL FLUID EXAMINATION

Lumbar puncture and cerebrospinal fluid examination

Cerebrospinal fluid was first examined in the 19th century using primitive techniques. Cerebrospinal fluid analysis reached a peak in the 1950s and early 1960s, when almost no workup of a significant central nervous system problem was performed without a lumbar puncture.

With the advent of sophisticated imaging techniques, particularly computerized tomography and magnetic resonance imaging, lumbar puncture is no longer an important test in the diagnosis of most intracranial mass lesions. This is especially true with the potential risk of brain herniation if intracranial pressure is increased markedly. Lumbar puncture remains a critical procedure in the diagnosis of central nervous system infections and inflammatory diseases.

Indications for cerebrospinal fluid examination and lumbar puncture

A. Cerebrospinal fluid examination is key to the diagnosis and management of various central nervous system infections, including acute and chronic meningitis and encephalitis. In many patients with fever of unknown origin, even in the absence of meningeal signs, early lumbar puncture is commonly of value, especially because meningeal signs may be minimal or absent in very young or elderly patients. Meningeal infection especially should be sought in patients with fever and impaired sensorium or an immunocompromised state. If a patient has unexplained acute confusion, stupor, or coma, even if afebrile, Cerebrospinal fluid examination is necessary for evaluation for meningoencephalitis. In most clinical settings, computerized tomography of the brain or other neuroimaging should be performed before lumbar puncture to rule out a possible intracranial mass (e.g., hemorrhage or abscess), which would make lumbar puncture a potentially lethal procedure. However, if the patient is extremely ill and acute meningitis, such as meningococcal meningitis, is suspected, lumbar puncture should be performed without delay to avoid losingvaluable time in beginning appropriate therapy.

B. In patients with suspected subarachnoid hemorrhage, urgent computerized tomography is indicated to evaluate for the presence of

blood. However, in approximately 10% of patients with subarachnoid hemorrhage, computerized tomography does not show blood, and a spinal tap is indicated. If the diagnostic lumbar puncture shows subarachnoid blood or xanthochromia, cerebral angiography is needed to determine the source of the hemorrhage.

C. In patients with unexplained dementia, cerebrospinal fluid examination may be necessary to evaluate for central nervous system vacuities, infection, or granulomatous disease. The cerebrospinal fluid should always be examined if the patient has dementia and a positive result of a fluorescent treponemal antibody absorption test. Patients with radiographic hydrocephalus also may need a cerebrospinal fluid study to exclude chronic meningitis as a cause of symptomatic hydrocephalus. In patients with suspected Creutzfeldt-Jakob disease, a positive result of radioimmunoassay of the cerebrospinal fluid for "prion protein" (14-3-3 protein) is a sensitive and relatively specific marker for prion disease, although false-positive results have been described with encephalitis and with recent stroke.

D. Cerebrospinal fluid examination usually is not warranted for and can be dangerous for most patients with stroke. However, cerebrospinal fluid analysis can assist in the etiologic diagnosis of unexplained stroke in young or middle-aged patients who lack atherosclerotic risk factors. Causes such as central nervous system vasculitis, meningovascular syphilis, and AIDS may be diagnosed.

E. Cerebrospinal fluid studies can aid in the diagnosis of multiple sclerosis, although there is no specific cerebrospinal fluid marker for this disease. Elevated cerebrospinal fluid immunoglobulin G levels with normal serum IgG levels and the presence of oligoclonal bands in the cerebrospinal fluid are characteristic but not specific for multiple sclerosis. An elevated cerebrospinal fluid gamma globulin level can occur in neurosyphilis, viral meningoencephalitis, and subacute sclerosing panencephalitis.

F. Cerebrospinal fluid analysis often is necessary, after appropriate neuroimaging, in the evaluation of patients admitted with an initial tonic-clonic seizure or status epilepticus to exclude active central nervous system infection or hemorrhage.

G. Lumbar puncture is necessary to confirm the clinical suspicion of carcinomatous or leukemic meningitis. Numerous lumbar puncture sometimes are needed. The typical cerebrospinal fluid pattern is pleocytosis with elevated protein, low glucose, and a positive cytologic results

for a malignant tumor.

H. Cerebrospinal fluid studies can aid in the diagnosis of certain inflammatory or demyelinating forms of neuropathy, such as Guillain—Barre syndrome or chronic idiopathic demyelinating polyradiculoneuropathy. The cerebrospinal fluid protein level often is elevated without an abnormal cellular response.

I. Although lumbar puncture is generally contraindicated in the evaluation of patients with papilledema, lumbar puncture is indicated to document increased intracranial pressure in a patient with suspected idiopathic intracranial hypertension after results of neuroimaging studies have proved normal. The spinal fluid is under increased pressure but is otherwise normal in this entity, except for occasional decreases in cerebrospinal fluid protein level. lumbar puncture is needed to document low cerebrospinal fluid pressure in rare low-pressure syndromes in a patient with headaches that occur on standing and are relieved by lying down.

J. Lumbar puncture can be used to deliver intrathecal antibiotics and chemotherapy in the management of certain central nervous system infections and meningeal malignant tumors. Lumbar puncture also is needed in certain diagnostic procedures, such as myelography and cisternography.

Contraindications to lumbar puncture

A. Lumbar puncture is contraindicated in evaluation of any patient with increased intracranial pressure, except idiopathic intracranial hypertension, because of the real danger of cerebral herniation and death.

B. Lumbar puncture is contraindicated if there is suppuration in the skin or deeper tissues overlying the spinal canal because of the danger of inducing purulent meningitis.

C. Lumbar puncture is dangerous in the presence of anticoagulation therapy or a bleeding diathesis. Heparin administration should not be reinstituted for a minimum of 2 hours after lumbar puncture is performed. In general, lumbar puncture is hazardous if the platelet count is less than 50,000/mL, or especially if it is less than 20,000/mL. In such cases, platelet transfusions should be initiated if possible before lumbar puncture.

D. Lumbar puncture should not be performed when a spinal mass is suspected unless the procedure is part of a myelogram with neurosurgical assistance readily available. A dramatic deterioration in spinal cord or

cauda equina function can occur after lumbar puncture.

Complications of lumbar puncture

A. Brain herniation and death can occur if an lumbar puncture is performed on a patient with increased intracranial pressure from a cerebral mass lesion. Lumbar puncture is contraindicated in the evaluation of any patient believed to have an intracranial mass.

B. Headache of low-pressure type occurs in as many as 10% of patients after lumbar puncture (spinal headache). This type of headache occurs only on standing and is relieved by lying down. It usually is self-limiting, but it can require an epidural autologous blood patch for relief. Post- lumbar puncture headache is most common among young women with lower body mass. Use of a higher-gauge (smaller diameter) needle, needle insertion parallel to dural fibers (bevel up with patient on side), and replacing the stylet before needle removal may he lumbar puncture prevent post- lumbar puncture headache. The occurrence of post- lumbar puncture headache is unrelated to cerebrospinal fluid opening pressure, cells, and protein; patient position during lumbar puncture; duration of recumbence after lumbar puncture; amount of cerebrospinal fluid removed; or hydration after lumbar puncture.

C. Diplopia, which usually results from unilateral or bilateral cranial nerve VI palsy, occurs rarely and usually is self-limiting.

D. Aseptic meningitis occurs rarely and is characterized by posterior neck pain, headache, and neck stiffness. This process usually is self-limiting.

E. Spinal epidural, subdural, and subarachnoid hematoma can occur, especially in patients taking anticoagulants or with bleeding diatheses. Such hematomas usually are self-limiting and can cause local pain and meningeal irritation. However, in rare instances, epidural hematoma causes flaccid and potentially irreversible paraplegia that necessitates emergency surgical evacuation.

General comments on evaluation of lumbar puncture results

A. Normal cerebrospinal fluid pressure is 70 to 180 mm of water in the lateral recumbent position. Pressure should be greater than 200 mm of water to be considered elevated. In an obese patient with possible idiopathic intracranial hypertension, the pressure should be greater than 250 mm of water to establish this diagnosis.

B. Normal cerebrospinal fluid glucose level is approximately two-thirds the serum glucose level, which must be measured at the time of

the lumbar puncture. Hypoglycorrhachia (low cerebrospinal fluid glucose) with few white blood cells suggests fungal infection. Many white cells suggest bacterial infection. Abnormal (malignant) cells suggest a malignant meningeal tumor.

C. The cerebrospinal fluid protein level may be increased (>100 mg/dL) in many central nervous system infectious and malignant processes. Causes of elevation of cerebrospinal fluid protein level with normal findings at neuroimaging include myxedema, inflammatory demyelinating polyneuropathy, diabetic polyneuropathy, neurofibroma within the cerebrospinal fluid pathways, resolving subarachnoid hemorrhage, gliomatosis cerebri, central nervous system vasculitis, and any process that causes spinal compression or obstruction of cerebrospinal fluid flow.

D. Normally, the cerebrospinal fluid can contain up to five lymphocytes or mononuclear cells per milliliter. Pleocytosis causes cerebrospinal fluid clouding when there are at least 200 cells/mL. White blood cell count increases with subarachnoid infection, hemorrhage, chemical meningitis, or meningeal neoplasms. Pleocytosis also can occur for approxi mately 24 hours after a generalized seizure.

E. If initial spinal fluid appears bloody, one must attempt to determine whether the source of the blood is a traumatic tap or subarachnoid hemorrhage. If the initial tube of fluid is bloody and subsequent tubes are progressively clear, it is most likely that the tap was traumatic. One should then immediately centrifuge the fluid to see whether the supernatant is clear, which suggests a traumatic tap. If the supernatant fluid is xanthochromic (yellow-tinged), it is likely that the blood has been present in the cerebrospinal fluid for a few hours. Xanthochromia occurs several hours after subarachnoid hemorrhage, reaches its greatest intensity at the end of 1 week, and clears in approximately 2 to 4 weeks. Xanthochromia also can be observed in jaundice and hypercarotenemia.

F. Polymerase chain reaction testing of the cerebrospinal fluid has been found to have great utility in the diagnosis of several CNS infections.

Intracranial pressure

Whenever the circulation of cerebrospinal fluid becomes impeded, intracranial pressure rises, producing headaches, dizziness, or nausea and vomiting. Headaches are probably caused by irritation of dural nerve endings responding to pressure and tension. Nausea and vomiting are probably caused by irritation of the vagus nerve. Fast-developing intracranial pressure may produce the objective signs of beginning or fully developed hemorrhagic papilledema. Longer-lasting papilledema leads to secondary atrophy of the optic nerve head, characterized by marked paleness of the papilla. This atrophy is often associated with deterioration of vision. If the third ventricle is part of a hypertensive internal hydrocephalus, its distended suprachiasmatic recess may press on the center of the chiasm, causing it to deform in the shape of an inverted horseshoe. The pressure may result in atrophy of crossing fibers and therefore in bitemporal hemianopic field defects. This may occur even if the tumor responsible for the internal hydrocephalus is far away in the cerebellum.

Once the fontanelles are closed the skull is a rigid box which can only accommodate a limited volume. At an early age some separation of the sutures may occur.

The normal structures within the skull have more or less stable volume. There are some variations in volume depending on the person's activities ,cardiovascular as well as pulmonary status. These variations are temporary and the intracranial pressure goes back quickly to its normal level.

The skull is not a completely closed sphere, there are several openings mainly in the base of the skull. It is believed that intracranial pressure is a reflection of the atmospheric pressure which is conducted through the large neck vessels.

Normal intracranial pressure

The normal pressure for is 15 mm/Hg or 150 mm - 200 mm of water. Accurate measurement of pressure in new born and infants is difficult to obtain but it is believed that in the first few months of life the intracranial pressure is lower . Values up to 8 mm/Hg has been considered to be normal in the first few months of life.

The intracranial pressure is closely related to brain perfusion. The cerebral blood flow is dependent on the intracranial pressure. In a simplified statement cerebral perfusion pressure is the difference be-

tween the systemic blood pressure and the intracranial pressure. For this reason the intracranial pressure needs to be maintained at a steady state. This is accomplished by dynamic equilibrium of intracranial components.

As the skull is a rigid box any extra volume added to the intracranial cavity needs to be at the expense of normal intracranial components in order to maintain the intracranial pressure at a normal level.

The relationship between the intracranial volume and the intracranial pressure is in following. In the early stages adjustment is made to maintain an intracranial pressure within normal range. This is called autoregulation. If the process continues the increase in volume will be associated with gradual rise in intracranial pressure. This continues up to an intracranial pressure of about 50mm hg when the pattern changes and the intracranial cavity loses its compliance and behaves as a solid box. There will be steep rise and incremental rise of the intracranial pressure.

Dynamic of raised intracranial pressure

Raised intracranial pressure could be the result of the following:

1. Increase of the volume of the normal content of the intracranial cavity, as increase in the volume of cerebrospinal fluid as in hydrocephalus, increase in the volume of the brain tissue itself as in brain edema or increased cerebral blood volume.

2 Extravolume added to the intracranial cavity as in tumors or hematomas.

Where an extra volume is added to the intracranial cavity this has to be at the expense of the normal contents. To start with the cerebrospinal fluid is squeezed out, one finds that the ventricles become compressed and displaced. There will be paucity of subarachnoid spaces and the basal cisterns become less discernible. Once a state is reached where no more cerebrospinal fluid could be expulsed, a change in the cerebral blood flow occurs. Cerebral blood perfusion is related to intracranial pressure. With the rise of intracranial pressure there is diminished cerebral blood flow and more space is provided for the extra volume. However a state would be reached where any further decrease in cerebral blood flow would lead to cerebral ischemia. In this situation the brain itself starts to herniate through the hiatus of the tentorium and foramen magnum. At this stage the patient’s condition is precarious and needs urgent action to reduce the intracranial pressure.

Increase blood volume can occur as a result of a blockage to venous drainage from the cranial cavity or due to vasodilatation. It is very important to appreciate that cerebral blood vessels are very sensitive to changes in blood gases especially carbon dioxide. High PCO2 results in vasodilatation and increase in cerebral blood volume.

Clinical presentation

Raised intracranial pressure can produce specific as well as non-specific signs and symptoms. Cushing triad of headache, papilloedema and vomiting is considered the classical presentation of raised intracranial pressure.

Headache. Headache is caused by distortion of intracranial blood vessels as well as stretching of the intracranial dura. Typically the headache is worse in the morning. The morning headache is related to poor venous drainage during sleep and probably a rise of PCO2 due to diminished respiratory efforts, these factors lead to rise in intracranial pressure.

Papilloedema. Ophthalmoscopy would reveal various degrees of disc swelling depending on the severity of the raised intracranial pressure. The optic nerve is part of the brain and is surrounded by meningeal covering. With severe disc swelling hemorrhages could develop. Sustained raised intracranial pressure for long periods of time could lead to loss of vision. During this period intermittent loss of vision (Amaurosis fugax) is a grave sign and would necessitate an urgent action to reduce the pressure otherwise visual loss would be permanent. With prolonged periods of raised intracranial pressure visual loss might continue in spite of relief of intracranial pressure.

The mechanism of disc swelling is not fully understood but it is believed that it is due to several factors which include the interference with the axoplasmic transport in the retina, vascular congestion and may be transmission of raised cerebral spinal fluid pressure along the optic nerve leading to compression of the central retinal vein and edema of the optic disc.

Vomiting. This is particularly common in children especially with lesions involving the floor of the fourth ventricle and direct irritation of the vagus nucleus. In raised intracranial pressure vomiting is more common in the morning.

Measurement of Intracranial Pressure:

Intracranial pressure is not a constant value but is variable. It can rises sharply with coughing and sneezing, up to 50 or 60 mm/Hg to settle down to normal values in a short time. It also varies according to the activity the person is involved with. For these reasons single measurement of intracranial pressure is not a true representation. Intracranial pressure needs to be measured over 24 to 48 hours to get a true representation. During this period close observation should be done to monitor patient activity at the time of change in intracranial pressure.

Measurement of intracranial pressure using closed fluid system has been replaced by more accurate means.

In the closed fluid system ventricular catheter is introduced into the ventricle which will be attached to a transducer.

The most accurate method of measuring the intracranial pressure is by the use of electrodes with a sensor attached to its tip. There are several versions of these catheters manufactured by different companies.

The first step is to find the cause of the raised intracranial pressure and remove it if possible. If there is excessive cerebrospinal fluid as in hydrocephalus then shunt procedure or external drainage should be instituted. If there is a respectable tumor then this should be removed. In cases where there are no surgically treatable cause efforts should be directed at reducing intracranial pressure by one of the following means:

Osmotic Diuretic: This acts by dehydrating the brain. This is achieved by removing extracellular fluid by creating an osmotic gradient across the capillary wall. The most commonly used agent is Mannitol. This is a carbohydrate which is not metabolized in the blood and remains entirely in the extracellular space. The dose is 0.25- 1 gram per kilogram body weight over 10 - 15 minutes. 20% of the solution is used. The effect of Mannitol lasts about 4 to 6 hours.

Mannitol has some side effects, its action is reduced with repeated doses and can cause systemic acidosis and renal failure due to increase plasma osmolality. After Mannitol is stopped there is rebound of intracranial pressure.

Other diuretics used is Furosemide which is a renal diuretic.

Diuretics could be used as a temporary measure while the patient is prepared for definitive surgical treatment.

Steroids. These are mainly used to reduce brain swelling around brain tumors. They are very effective in these conditions.

Cerebrospinal fluid drainage. This is an effective and rapid way of reducing intracranial pressure in cases where ventricles are visible and can be cannulated. However in severe brain edema with collapsed ventricles it is difficult to get into the ventricle. The ventricular catheter could be used to monitor intracranial pressure at the same time.

Hyperventilation. As mentioned before cerebral blood vessels are sensitive to changes in blood gases. The aim of hyperventilation is to reduce the PCO2 to a level around 30 mm/Hg. Low PCO2 will cause vasoconstriction and reduced intracranial blood volume. Levels below 30 mm hg should be avoided as it can cause cerebral ischemia.

Barbiturate is used to induce deep coma, where there is a reduction in metabolic rate, oxygen consumption and CO2 production. This method is used only when all other means of treatment failed.

Meningeal syndrome

Pathophysiology

When the protecting barriers of the brain, including the skull, meninges, and blood-brain barrier, are broached by a pathogen, meningitis can result. Predisposing factors include preexisting diabetes mellitus, immunosuppression otitis media, pneumonia, sinusitis, and alcohol abuse. Meningeal inflammation and irritation elicit a protective reflex to prevent stretching of the inflamed and hypersensitive nerve roots, which is detectable clinically as neck stiffness or meningeal signs. Meningeal irritation due to inflammation also may cause headache and cranial nerve palsies. When cerebral edema and elevated intracranial pressure occur, alterations in mental status, headache, vomiting, seizures, and cranial nerve palsies may ensue.

Most acute viral meningitides produce symptoms with variations depending on the particular virus. Some individuals may experience a biphasic type of illness with nonspecific constitutional symptoms followed by meningitis.

In some cases meningeal syndrome appears without infection and there is aseptic meningitis.

Headache, fever, stiff neck, photophobia, drowsiness, myalgia, malaise, chills, sore throat, abdominal pain, nausea, and vomiting usually

characterize acute meningeal syndrome. Focal signs, seizures, and profound lethargy are rarely a part of this syndrome.

Meningeal signs

Neck stiffness in meningitis is tested by gentle forward flexion of the neck with the patient lying in the supine position. Meningeal irritation also can be tested by the jolt accentuation of headache. This is tested by asking the patient to turn his or her head horizontally at a frequency of 2-3 rotations per second. Worsening of a baseline headache represents a positive sign.

Severe meningeal irritation may result in the patient assuming the tripod position with the knees and hips flexed, the back arched lordotically, the neck extended, and the arms brought back to support the thorax.

When passive neck flexion in a supine patient results in flexion of the knees and hips, a positive Brudzinski sign is entertained. Yet another sign, the contralateral reflex, is present if passive flexion of one hip and knee causes flexion of the contralateral leg.

Kernig sign is elicited with the patient lying supine and the hip flexed at 90°. A positive sign is present when extension of the knee from this position elicits resistance or pain in the lower back or posterior thigh.

QUESTIONS FOR ASSESSMENT

Motor disorders

1. Structures where the central (upper) motoneurons are localized.
2. Structures where the peripheral (law) motoneurons are localized.
3. Somatotopic order of motoneurons in the motor cortical brain area.
4. Somatotopic order of the motor conductors in the internal capsule.
5. Somatotopic order of the motor (pyramidal) conductors in the spinal cord.
6. Somatotopic order of the peripheral motoneurons in the anterior horn of the spinal cord.
7. Segmental order of movement distribution on the cervical level of the spinal cord.
8. Segmental order of movement distribution on the lumbar level of the spinal cord.
9. Segments of the spinal cord corresponding to the bicipital, tricipital and carporadial reflexes.
10. Segments of the spinal cord corresponding to the knee and Achill's reflexes.
11. General classification of movement disorders associated with nervous system lesions.
12. Clinical characteristic of the paresis.
13. Classification of the paresis according to the localization of damage in the motor pathway.
14. Classification of the paresis according to the localization on the body.
15. Classification of the paresis according to the muscular tone.
16. Classification of the paresis according to its expressiveness.
17. Classification of paresis according to the stages of its development.
18. Classification of the pathological symptoms.
19. Clinical signs of the central paresis.
20. Clinical signs of the peripheral paresis.
21. Clinical characterization of the paresis in case of brain cortex lesion.
22. Clinical characterization of the paresis in case of subcortical brain lesion (white matter).

23. Clinical characterization of the paresis in case of internal capsule lesion.

24. Clinical characterization of the paresis in case of one-side brainstem lesion.

25. Clinical characterization of the paresis in case of lesion of two brainstem sides.

26. Clinical characterization of the paresis in case of one-side spinal cord lesion above the C4-segment level.

27. Clinical characterization of the paresis in case of lesion of two spinal cord sides above the C4-segment level.

28. Clinical characterization of the paresis in case of one-side spinal cord lesion below the T1-segment level.

30. Clinical characterization of the paresis in case of lesion of two spinal cord sides below the T1-segment level.

31. Clinical characterization of the movement disorders in case of one-side spinal cord lesion on the level of C5-T1 segments.

32. Clinical characterization of the movement disorders in case of lesion of two spinal cord sides on the level of C5-T1 segments.

33. Clinical characterization of the movement disorders in case of one-side spinal cord lesion on level of L2-S2 segments.

34. Clinical characterization of the movement disorders in case of lesion of two spinal cord sides on level of L2-S2 segments.

35. Clinical characterization of the movement disorder in case of lesion of the spinal cord root.

36. Clinical characterization of the movement disorder in case of cervical plexus lesion.

37. Clinical characterization of the movement disorder in case of lumbar plexus lesion.

38. Clinical characterization of the movement disorder in case of median nerve lesion.

39. Clinical characterization of the movement disorder in case of sciatic nerve lesion.

40. Structure of the gamma-circuit.

41. Functional significance of the gamma-circuit

42. Clinical characterization and types of epileptical seizures.

Sensory disorders

1. Structures where the first neuron of the superficial sensory conductors is localized.
2. Structures where the second neuron of the superficial sensory conductors is localized.
3. Structures where the third neuron of the superficial sensory conductors is localized.
4. Structures where the first neuron of the deep sensory conduc-tors is localized.
5. Structures where the second neuron of the deep sensory conductors is localized.
6. Structures where the third neuron of the deep sensory conductors is localized.
7. Somatotopic order of the sensory neurons in the cortex.
8. The primary and secondary cortical sensory fields.
9. Somatotopic order of sensitivity in the superficial sensory conductors.
10. Somatotopic order of sensitivity in the deep sensory conduc-tors.
11. Classification of sensitivity.
12. Types of sensitivity disorders.
13. Clinical characterization of pain.
14. Characterization of the phantom pain.
15. Characterization of the causalgia.
16. Characterization of the sympatalgia.
17. Types (syndromes) of sensitivity disorders.
18. Clinical characterization of the neural sensitivity disorder.
19. Clinical characterization of the polyneural sensitivity disorder.
20. Clinical characterization of the sensitivity disorder in case of the plexus lesion.
21. Clinical characterization of the root sensitivity disorder.
22. Clinical characterization of the conductor sensitivity disorder.
23. Clinical characterization of the segmental sensitivity disorder.
24. Clinical characterization of the sensitivity disorder in case of one-half spinal cord lesion.

25. Clinical characterization of the sensitivity disorder in case of lesion of the posterior column of the spinal cord.
26. Clinical characterization of the sensitivity disorder in case of lesion of the lateral column of the spinal cord.
27. Clinical characterization of the cortical sensitivity disorders.
28. Clinical features of the psychogenetic sensitivity disorders.
29. Clinical characterization of the sensitivity disorder in case of thalamus lesion.
30. Clinical characterization of the sensitivity disorder in case of one-side brainstem lesion.

Cranial nerve disorders.

1. Anatomical structures of the olfactory system.
2. Clinical characterization of the peripheral olfactory disorders.
3. Clinical characterization of the central olfactory disorders.
4. Topical diagnostics of the olfactory disorders.
5. Anatomical structures of the optic system.
6. Clinical characterization of the visual acuity disorders in case of the refraction anomalies.
7. Clinical characterization of the visual acuity disorders in case of optic system lesion.
8. Clinical characterization of disorders of the vision fields.
9. Characterization and clinical meaning of the eyes ground changes in diagnostics of the visual disorders.
10. Characterization of the normal pupil reactions.
11. Characterization and clinical meaning of disorders of the pupil reactions.
12. Characterization of the optic gnosis in case of optic system lesion.
13. Characterization of the color perception disorders in case of optic system changes.
14. Clinical syndrome of the optic nerve lesion.
15. Clinical syndrome of lesion of the external regions of the chiasm.
16. Clinical syndrome of lesion of the internal regions of the chiasm.
17. Clinical syndrome of the optic tract lesion.

18. Clinical syndrome of lesion of the lateral geniculate body.
19. Pupil disorders in case of lesion of the superior colliculi of the midbrain.
20. Clinical syndrome of lesion of the posterior region of the internal capsule.
21. Clinical syndrome of lesion of the optic radiation.
22. Clinical syndrome of lesion of the calcarine cortex.
23. Anatomical structures organizing oculomotion.
24. Clinical syndrome of lesion of the trunk of the oculomotor nerve.
25. Clinical syndrome of lesion of the nucleus of the oculomotor nerve.
26. Clinical syndrome of the brainstem lesion on the level of the nucleus of the oculomotor nerve.
27. Clinical syndrome of lesion of the Edinger-Westphal nucleus.
28. Clinical syndrome of lesion of the trochlear nerve.
29. Clinical syndrome of lesion of the abducens nerve.
30. Clinical syndrome of internuclear ophtalmoplegia.
31. Clinical syndrome of disorders of the conjugate movements of the eyes in case of hemisphere lesion.
32. Clinical syndrome of disorders of the conjugate movements of the eyes in case of brainstem lesion.
33. Clinical syndrome of disorders of the conjugate movements of the eyes in case of lesion of the midbrain colliculi.
34. Trigeminal nerve nuclei.
35. Motor disorders in case of the trigeminal nerve lesion.
36. Peripheral sensory disorders in case of the trigeminal nerve lesion.
37. Sensitivity disorders of the trigeminal nerve system in case of the brainstem lesion.
38. Sensitivity disorders of the trigeminal nerve system in case of the hemisphere lesion.
39. Syndrome of trigeminal neuralgia.
40. Clinical syndrome of the facial nerve lesion in the pontocerebellar angle.
41. Clinical syndrome of the facial nerve lesion in the internal acoustic meatus.

42. Clinical syndrome of the facial nerve lesion above the origin of the major petrosal nerve.
43. Clinical syndrome of the facial nerve lesion above the origin of the stapedius nerve.
44. Clinical syndrome of the facial nerve lesion above the origin of the chorda tympani.
45. Clinical syndrome of the facial nerve lesion on the level of the outlet from the cranium.
46. Clinical syndrome of the lesion of the motor nucleus of the facial nerve.
47. Clinical syndrome of the one-side lesion of the brainstem on the level of the inner knee of the facial nerve.
48. Clinical syndrome of the one-side lesion of the brainstem near the facial nerve nucleus.
49. Clinical syndrome of the central lesion of the mimic muscles.
50. Clinical syndrome of the lesion of the cochlear portion of the vestibule-cochlear nerve.
51. Clinical syndrome of the lesion of the vestibular portion of the vestibule-cochlear nerve.
52. Characterization of the systemic vertigo.
53. Differential diagnostics of the conduction deafness and nerve or inner ear deafness.
54. Clinical syndrome of the glossopharyngeal nerve lesion.
55. Clinical syndrome of the glossopharyngeal neuralgia.
56. Clinical syndrome of the one-side lesion of the vagus nerve.
57. Clinical syndrome of the two-side lesion of the vagus nerve.
58. Clinical syndrome of the one-side lesion of the accessory nerve.
59. Clinical syndrome of the two-side lesion of the accessory nerve.
60. Clinical syndrome of the lesion of the trunk of the hypoglossal nerve.
61. Clinical syndrome of the lesion of the nucleus of the hypoglossal nerve.

62. Clinical syndrome of the one-side lesion of the brainstem on the level of the hypoglossal nerve nucleus.
63. Progressive bulbar paralysis.
64. Pseudobulbar paralysis.
65. Reflexes of the oral automatism.
66. Mechanism of the formation and the features of the brainstem lesion syndromes.

Syndromes of the brainstem lesion.

1. Clinical anatomy of the midbrain.
2. Clinical signs of transverse lesion of the midbrain.
3. Clinical signs of the unilateral lesion of the midbrain.
4. Weber's syndrome.
5. Clinical signs of lesion of the tegmentum of the midbrain.
6. Clinical anatomy of the pons of the brainstem.
7. Clinical signs of transverse lesion of the pons.
8. Clinical signs of the unilateral lesion of the pons.
9. Clinical anatomy of the medulla oblongata.
10. Clinical signs of transverse lesion of the medulla oblongata.
11. Clinical signs of the unilateral lesion of the medulla oblongata.
12. Jackson's syndrome.
13. Avellis syndrome.
14. Wallenberg's syndrome.

Syndromes of the cerebral cortex lesion

1. Anatomy of the cerebral cortex (external and internal structure).
2. Anatomy of the white matter: projection, association and commissural fibers.
3. Functional organization of the cortex.
4. Types of the speech disorders.
5. Clinical characterization of the motor aphasia.
6. Topico-diagnostic meaning of the motor aphasia.
7. Clinical characterization of the sensory aphasia.
8. Topico-diagnostic meaning of the sensory aphasia.
9. Clinical characterization of the amnesic aphasia.
10. Topico-diagnostic meaning of the amnesic aphasia.

11. Clinical characterization and diagnostic meaning of the optic agnosia.
12. Clinical characterization and diagnostic meaning of the acoustic agnosia.
13. Types and diagnostic meaning of the sensory agnosia.
14. Clinical characterization and diagnostic meaning of the ideo-motor apraxia.
15. Clinical characterization and diagnostic meaning of the limb-kinetic apraxia.
16. Clinical characterization and diagnostic meaning of the con-structive apraxia.
17. Clinical syndrome of the frontal lobe lesion.
18. Clinical characterization of the psychiatric disorders in case of the orbital lobe lesion.
19. Clinical syndrome of the temporal lobe lesion.
20. Clinical syndrome of the parietal lobe lesion.
21. Clinical syndrome of the occipital lobe lesion.

Disorders of coordination. Extrapyramidal disorders

1. Clinical anatomy of the cerebellum.
2. Afferent ascendant pathway of the cerebellum.
3. Afferent descendant pathway of the cerebellum.
4. Efferent pathway of the cerebellum.
5. Symptoms and signs of disorders of coordination in axial muscles.
6. Symptoms and signs of disorders of coordination in the head.
7. Symptoms and signs of disorders of coordination in arms.
8. Symptoms and signs of disorders of coordination in legs.
9. Signs of the paleo- and archicerebellum lesion.
10. Signs of the neocerebellum lesion.
11. Clinical features of the cerebellar ataxia.
12. Clinical features of the sensory ataxia.
13. Clinical features of the sensory ataxia in case of the posteri-or funiculi lesion.
14. Clinical features of the sensory polyneural ataxia.
15. Clinical features of the vestibular ataxia.

16. Clinical features of the ataxia in case of the internal capsule lesion.
17. Clinical features of the frontal lobe ataxia.
18. Clinical features of the psychogenic ataxia.
19. Anatomy of the basal ganglia.
20. Functions of the extrapyramidal system.
21. Classification of the syndromes of the extrapyramidal system lesion.
22. Hypokinesia-hypertonia syndrome.
23. Hyperkinesia-hypotonia syndrome.
24. Clinical characterization of the chorea.
25. Clinical characterization of the athetosis.
26. Clinical characterization of the spasmodic torticollis and torsion dystonia
27. Clinical characterization of the ballistic syndrome.
28. Other types of the hyperkinesias.

Impaired conscious level

1. Conscious level examination in the neurology.
2. The Glasgow coma scale.
3. Classification of the conscious level impairment.
4. Clinical characterization of the clear conscious.
5. Clinical characterization of the confusion.
6. Clinical characterization of the sopor.
7. Clinical characterization of the moderate coma.
8. Clinical characterization of the deep coma.
9. Clinical characterization of the atonic coma.
10. Pathophysiology of coma.
11. Causes of coma.
12. Clinical characterization of the apallic syndrome.
13. Clinical characterization of the akinetic mutism.
14. Clinical characterization of the “locked in” syndrome.
15. Clinical characterization of the delirium.

Meningism. Raised intracranial pressure. CSF examination

1. Causes of raised intracranial pressure (RIP).
2. Mechanisms of RIP.
3. Clinical characterization of RIP.

4. Ophtalmoscopic signs of RIP.
5. Changes of the scull X-ray in RIP.
6. Diagnostic measures in primary diagnostic of RIP.
7. Causes of meningism.
8. Mechanisms of meningism.
9. Clinical signs of meningism.
10. Paraclinical signs of meningism.
11. Diagnostic measures in meningism.
12. Mechanisms of nonocclusive hydrocephalus.
13. Clinical characterization of nonocclusive hydrocephalus.
14. Mechanisms of occlusion of CSF flow.
15. Clinical characterization of occlusion of the Monro foramen.
16. Clinical characterization of occlusion of the brain aqueduct.
17. Clinical characterization of occlusion at the level of the 4th ventricle.
18. Investigations in hydrocephalo-occlusive syndrome.
19. Treatment of the hydrocephalo-occlusive syndrome.
20. Normal parameters of the CSF.
21. Changes of SCF in hydrocephalo-occlusive syndrome.
22. Characterization of the hemorrhagic syndrome of CSF.
23. Characterization of the inflammatory syndrome of CSF.
24. Indications for the LP.
25. Contraindications for the LP.
26. Technique of the LP.
27. Complications of the LP.

PLAN OF CLINICAL RESEARCH OF THE PATIENT

Passport part

Surname, name, patronymic. Age. Trade. The marital status. Residence. Date of hospitalization in clinic.

Complaints

Complaints with their detailed characteristic are marked. 1. At pains their character, localization, duration, intensity, dynamics, the reasons of occurrence and intensifying, the factors of simplification accompanying frustration is underlined. 2. At weakening of movements in extremities localization, an expressiveness of these frustration, dynamics, accompanied frustration is described. 3. At complaints to attacks their character, duration, condition of consciousness, before- and after attack periods, the reasons of occurrence and discontinuance, impellent, sensitive, vegetative frustration from words of the patient and witnesses is in detail described. 4. At sensitive frustration describe character, localization, intensity, dynamics accompanying infringements.

History of disease

The date started of disease, provisional time when there were the factors previous or accompanying disease is marked. It is possible to note, to what the patient connects occurrence of disease: trauma of head, body, disease - flu, angina, etc., physical and mental stress, mental traumas, intoxications, etc. Describe signs of illness. In detail describe development and current of disease, duration and the reasons of the periods of deterioration. Mark sequence of change of infringements. The factors worsening course of illnesses. Change of serviceability during illness. Represent results of researches before clinic investigation in the chronological statement, before fixed diagnoses. Describe sequence and efficiency of medical actions. The case history is described investigating till the moment of examining.

History of life

The birthplace. Development in infancy. The transferred children's illnesses. Formation. Job conditions, conditions of life. Intoxications (smoking, alcohol, narcotics). Time of the beginning menstruation, pregnancy, childbirth, abortions. Health of members of family, relations in family.

Experienced diseases. Character of trauma and duration of post-traumatic frustration. The transferred operations, anesthetic aid. Mental, physical and emotional stresses. An intolerance of medicines, foodstuff, production factors, the description of reactions to them. Chronic and hereditary diseases at close relatives. A family tree.

The general condition

Position of the patient at the moment of examination. A general view, a body build, a feed, weight and body height. A skin: coloring, cicatrices, intradermal and hypodermic formations. A condition of hair, nails, mycoses. A condition of oral cavity. Lymph nodes. Deformations of a backbone (kyphosis, lordosis, scoliosis). Mobility of various departments of a backbone, morbidity at a load, morbidity of acanthus at a percussion. The form of a skull, a percussion of a skull. Deformation of joints, muscle contraction, ankyloses. Development of muscular system. An endocrine condition. Development of secondary sexual attributes.

Mild - data percussions and auscultation.

Heart - data percussions and auscultation. Pulse, his characteristic. Arterial pressure.

Organs of digestion, liver and lien.

Pelvic organs -delay, incontinence of urine and feces, imperative desires, etc.

Neurological condition

Consciousness: orientation in place and time, ability to contact. Mood. Behavior at investigation, attitude to the disease. Memory. Crazy ideas. Deceits of perception. A condition of intelligence.

Meningeal signs

Muscle tension of nape, Kernig's sign.

Cranial nerves

Olfactory nerve. An olfaction: it is kept, reduced, lost. Presence of olfactory hallucinations. To investigate an olfaction with the heLPof not irritating odorous substances, separately on the right and at the left.

Optic nerve. Visual acuity with correction and without correction. A color perception. A field of vision. A condition of an eye ground.

Oculomotor, trochlear, abducence nerves. Width and evenness paLPebral fissures. Volume of movements of eye globes in the par-

ties, upwards, downwards. Presence of a converging strabismus. diplopia. Paresis and paralyses of look.

Pupils - the form, size (mydriasis, miosis); uniformity. Reaction of pupils to light (straight, concomitant), on convergence and accommodation.

Trigeminal nerve. Morbidity of pressure upon points of an output of branches triple nerve (supraorbital, infraorbital and mental). Pains and paresthesia in face. Pain, temperature, tactile sensitivity of skin of face, mucous mouth, nose, tongue (kept, reduced, lost, perverted - hyperalgia). To describe borders of frustration of sensitivity on the person (zones 1, 2, 3 branches, segmentary zones Zelder's). Taste on forward two thirds of tongue. A chewing musculation (expressiveness, strain, atrophies). Volume of movement of a mandible. Corneal, conjunctive, nasal, mandibular reflexes (kept, reduced, no, uniformity).

Facial nerve. A condition of mimic musculation - an expressiveness and uniformity frontal and nasolabial fold, locating angle mouth in rest and at the set movements (wrinkle up forehead, close eye, knit brows, puff up cheeks, show teeth). Watering, xerophthalmus.

Acoustical nerve. Sensory acuity on colloquial and whisper speech SEPARATELY on each ear. A sonitus, a giddiness. A nystagmus.

Glossopharyngeal and wandering nerves. A swallowing - choke at meal, hit of liquid nutrition in a nose). Sonority of a voice - normal, is weakened, an aphonia. Mobility of a soft palate - sufficient, is weakened, from what party. Taste on a back third of tongue - normal, is weakened, no. A sialosis. Palatine, pharyngeal reflexes, their vivacity, uniformity.

Accessories nerve. Appearance sternocleidomastoid and trapezoid muscles - normal, atrophies, from what party, a degree. Volume of active movements at turns of a head, at lifting shoulders, at rapprochement of scapulas.

Hypoglossal nerve. A kind of tongue: atrophies, fibrillar twitchings (from what party). A position of tongue at protrusion - on an average line, a deviation aside. Volume of active movements tongue in sides, upwards, downwards. A sharpness and clearness of pronunciation - normal, dysarthtria, anarthtria.

Impellent sphere

Survey of a musculation of extremities and trunks. Presence of an atrophy or a hypertrophy - to specify, what muscles, a degree of an atrophy, to lead the data of gaugings centimetric - a tape of a circle of extremities (a brachium, a forearm, a femur, an anticnemion). Fibrillar and fascicular twitchings (is whether or not, their localization).

Active movements. Volume of active movements in articulation of extremities. At restriction of movements to specify, in what joints and a degree of restriction. An animal force. Test Barre (top and bottom).

Passive movements are possible in full or circumscribed. To specify a degree of restriction (in degrees). Presence of a rigidity in joints. Contraction. A condition of a muscle tone (it is defined during passive movements and at a paLPation of muscles): normal, it is reduced, raised. Character of rising of a muscle tone (pyramidal or to extrapyramidal type). Presence of a phenomenon of "cogwheel". Synkineses. A mechanical excitability of muscles.

Consensual movements (hyperkinesias): a tremor, a chorea, an athetosis, choreoathetosis, cramps, habit spasms, torsion spastic stricture, myoclonias. At the description hyperkinesias mark their amplitude, rate, rhythm, stereotype or variety, a constancy (constants, only in rest or at movements). Hypokinesia.

Coordination of movements: finger - nasal and calcaneo-knee assays - exact, with miss, with intention tremor. Hypermetria, adiadochokinesia. Fastness in Romberg's position. Test combined flexions of trunk and a hip (Babinski's test). A scanning speech.

Gait of the patient: normal, spastic, paretic, hemiplegics, ataxic (with the open and closed eyes), spastic-ataxic, "cock gait", "waddling gait", pretentious - elaborate, etc.

Reflex sphere

Deep (tensions and periostal reflexes) - radiocarpal, tendons of a biceps, triceps, knee, Achilles tendon reflex. To specify a degree of an expressiveness (high, normal amplitude, are reduced, are absent), uniformity. Dermal reflexes - belly, plantar - their degree (alive, are reduced, are absent), uniformity. Pathological foot reflexes Babinski's, Oppengeim's, Gordon's, Sheffer's, Chaddok's, Rossolimo's, Bekhterev's: no, are available, from what party, their expressiveness. Clone feet and knee cups. Pathological reflexes on arms: Yakobson - Bekhterev's, Bekhterev's, Tremner's. Grasping reflex. Protective bending reflexes, automatisms if are available, on what party, their

character, a level from which they are caused. Reflexes of oral automatism.

Sensitive sphere

Pains (local, projective, irradiating, their character). Paresthesias, describe their character, degree, localization. Morbidity at pressing on nervous trunks, if is present to specify in what points, and its degree). Signs of a tension of a sciatic nerve (Lasseg's, Nery's, landings), femoral nerve (Wasserman's, Matskevich's). Antalgic posture of the patient. Antalgic scoliosis.

Superficial sensitivity (tactile, pain, temperature). At infringement of superficial sensitivity to specify, on what sites of a skin changes, what character (hypoesthesia, anesthesia) are marked. Deep sensitivity - muscular - articulate feeling, vibratory sensitivity. At the description of muscular - articulate feeling to specify, in what joints and in what degree it is upset. two-dimensional spatial feeling, discrimination, feeling of localization. Types of frustration of sensitivity: periphery, radicular, segmentary, conduction, central (hemi type).

Frustration cortical functions (aphasia, apraxia, agnosia).

At presence of an aphasia more detailed research is made for revealing such as speech infringement: comprehension inverted to researched to oral speech, an estimation of correctness of speech of the patient (freedom of speech, a vocabulary, paraphasias, comprehension written), the letter active, under dictation, copying, denomination for memory object (amnesic aphasia). Apraxia, agnosia.

Vegetative nervous system

Local changes of a skin, pigmentation, trophicity, temperature of a skin. Acrocyanosis, mottled skin. Dermographism, expressiveness, stability. The diaphoresis - normal, is raised, lowered. Oculocardiac Aschner's reflex. Ortoclinostatic test. Gorner's syndrome. Exophthalmus.

The formulation of clinical syndromes

The topical diagnosis

Substantiation of the preliminary clinical diagnosis. On the basis of the reason of disease, the mechanism of development of the disease, clinically fixed description of localization and character of pathological process - the topical diagnosis the clinical form of disease - the preliminary clinical diagnosis is proved.

The differential diagnosis

The diseases similar by criteria of diagnostics are listed. The plan of clinical research is resulted.

The final clinical diagnosis

Etiology and pathogenesis of disease. Treatment planning.

The prognosis.

Epicrisis.

CLINICAL TASKS

1. The patient has reduced strength of the right hand with atonia and atrophy of its muscles. Fibrillar jerks are observed in weakened muscles. The bicipital, tricipital and carporadial reflexes are missing on the right side. Name the disorder. Make the topical diagnosis.

2. The patient has reduced strength of the left hand and leg, together with atrophy of the deltoid and bicipital muscles, the bicipital reflex is missing on the left side. The tricipital, carporadial, knee and Achill's reflexes are increased and dermal abdominal reflexes are reduced on the left. Babinski's and Oppengeim's pathological reflexes are present on the left. Name the disorders. Make the topical diagnosis.

3. The patient has sharply reduced strength of the distal parts of legs; both hypotonia and atrophy of the muscles of shins and feet are present. Achill's and the plantar reflexes are missing. The patient cannot stand on heels. Name the disorder. Where is the lesion localized?

4. Active movements are absent in the legs, the tone of muscles is high. The knee and Achill's reflexes are increased. There are patella and feet clonuses. Babinski's and Rossolimo's pathological reflexes are positive in both sides. All sorts of sensitivities are lost from the inguinal level. Ischuria is present. Describe the character of paralysis and make the topical diagnosis.

5. The patient has weakness and atrophy of the small muscles of the left hand. The carporadial reflex is reduced. All sorts of sensitivities on the internal surface of the left brachium and forearm are lost. Make the syndromes and the topical diagnosis.

6. The patient has legs weakness. The tone of hip extensors is increased. The knee and Achill's reflexes are high. There are clonuses of patella and feet. The middle and lower abdomen reflexes are missing. The pathological feet reflexes are positive in both sides. Pain and temperature sensations are lost from the navel downwards in both sides. Ischuria and constipation are observed. Name the syndromes. Make the topical diagnosis.

7. Name paralyses according to the location on the body: paralysis of one, two, three, four extremities, half of the body. Explain differences between paralysis and paresis.

8. Name motor and sensory disorders in case of lesion of the cervical and lumbar thickenings of the spinal cord as well as of its thoracic part.

9. Which paralysis is typical of the right internal capsule lesion?

10. What is the wrist view in case of the ulnar, radial and median nerves lesion?

11. The patient has pains on the anterior surface of the right leg. Strength in the extensors of the leg is reduced. The right quadriceps femur atonia and atrophy are observed. The right knee reflex is absent. There is hypoesthesia on the right anterior surface of the femur and anterior internal surface of the leg. Which nerve is affected? Which stretching signs are typical for this nerve damage?

12. The patient has severe pains on the back and lateral surfaces of the left leg and in the foot. The gait is "cock-like". The left foot drops and is turned slightly inside. Achill's left reflex is not present. There is hypoesthesia on the back and lateral surfaces of the leg and on the foot on the left. Laseg's sign is positive on the left side. Make the topical diagnosis.

13. Describe the clinical pattern of the spinal cone lesion. Which diseases is it necessary to differentiate between in case of such a disorder?

14. The left pyramidal path is damaged on the level of 6-th thoracic segment. Describe the clinical syndrome.

15. The patient has tetraplegia: peripheral paralysis of the hands and central paralysis of the legs. Make the topical diagnosis.

16. The patient has limited movements in the right humeral and ulnar joints. However movements of the wrist are not limited. The patient cannot brush the hair; bring a spoon to the mouth because of movement limitation. Atonia and atrophy of the right shoulder muscles, deltoid and biceps are observed. The right biceps reflex is absent. The sensitivity is reduced on the right shoulder field, and on the lateral surface of the brachium and forearm. Make the topical diagnosis. What is the name of this paralysis?

17. There are limited motions of the left leg. The leg extensor muscular tone is increased. The left knee and Achill's reflexes are higher than on the right. The left abdomen reflexes are absent. Babinski's reflex on the left side is positive. There are analgesia and thermoesthesia at the level of the left papilla in the form of a narrow strip. Pain and temperature sensitivities are lost below the level of the right papilla. Tactile, vibratory, muscle sensitivity is absent below the level of the left rib arc. Make the topical diagnosis. What is the name of the described syndrome?

18. The left leg active movements are absent. Atrophy and atonia together with fascicular jerks are observed. The left cremasteric, knee, Achill's and plantar reflexes are missing. Make the topical diagnosis.

19. What clinical pattern is observed in case of lesion of the upper part of the left front central gyrus? Which diseases is it necessary to differentiate between in this case?

20. The patient has severe arms and legs weakness. The muscle tone is increased in the arms flexors and in the legs extensors. The arms and legs tendon and periosteal reflexes are high, the abdominal reflexes are missing. Babinski's reflexes on both sides are positive. Anesthesia below the neck is observed. The delay of urination and constipation are detected. Name syndromes and make the topical diagnosis.

21. Describe the cauda equina lesion.

22. What reflexes and how are they changed in case of lesion of anterior horns of the lumbar thickening?

23. Which paralyses are typical of damage of the precentral gyrus of the frontal lobe?

24. What do Babinski's and Rossolimo’s reflexes indicate?

25. Horner’s syndrome has been detected in both sides. Active arms and legs movements are missing. Atonia and atrophy of the shoulder and arms muscles are revealed. The legs muscle tone is increased. The bicipital, tricipital and carporadial reflexes are reduced. The knee and Achill’s reflexes are high. There are patella and feet clonuses. The dermal abdomen reflexes are absent. Babinski's and Rossolimo’s reflexes are positive in both sides. All sorts of sensitivities are lost below the area of the shoulders. There are ischuria and constipation. Make the topical diagnosis.

26. The legs force is sharply reduced. There is atrophy and atonia of both gluteus muscles and muscles of the back surface of the femur, shank and feet. The anal reflex is positive, the knee reflexes are reduced, Achill’s and plantar reflexes are absent. “Saddle-like” anesthesia on the back surface of the femurs, legs and feet is observed. The patient has delay of urination and defecation. Make the topical diagnosis.

27. The active arms and legs movements are lost. The muscular tone is increased in the arms flexors and in the legs extensors. The arms and legs tendon and periosteal reflexes are high. There are patella and feet clones. The dermal abdomen reflexes are absent. Babinski's and Rossolimo’s plantar reflexes are present in both sides. The protective reflexes are observed. All sorts of sensitivities are lost in the area of the nape, neck, trunk and all extremities. There is delay of urination and constipation. There are severe respiration disorders (dyspnea) and hiccup. The chest X-raying reveals sharp limitation of the diaphragm mobility. Make the topical diagnosis. Describe the conductory type of the sensory disorder.

28. The patient has pains in the distant departments of the arms and legs, loss of all sorts of sensitivities in the arms in the form of «gloves» and «stockings» in the legs. Carporadial, Achill's and plantar reflexes are lost. While standing and walking with closed eyes there is instability and falling to different sides. Name syndromes and make the topical diagnosis. What is the name of the described disorder?

29. The patient has loss of all sorts of sensitivity in the perineum area, absence of the anal reflex, as well as urine and feces incontinence. Name syndromes and make the topical diagnosis.

30. The patient complains of severe shooting pains in the legs and perineum's area which are sharply intensifying when coughing and sneezing. The active legs movements are lost. The legs muscles atonia and atrophy are detected. Frank hypoesthesia of all sorts of sensitivity is observed in the lower extremities and in the perineum. The cremasteric, knee, Achill's and anal reflexes are absent. Pathological reflexes are not present. Incontinence of urine and feces is observed. Make and explain the topical diagnosis.

31. The patient has pains, loss of all sorts of sensitivity in the form of a wide half-belt on the trunk from the umbilicus to the inguinal level. The right medial and inferior dermal abdomen reflexes are lost. Make and explain the topical diagnosis.

32. Right arm analgesia and thermoesthesia are detected. The right bicipital, tricipital and carporadial reflexes are absent. Make and explain the topical diagnosis.

33. The tactile, muscle-joint, vibratory sensitivities loss is detected on the right side from the umbilicus level downwards. The right medial dermal abdominal reflex is missing. The lower abdominal, knee and Achill's reflexes are present. Sensory ataxia in the right leg is observed: it is not possible to make the heel-to-knee test by this leg with closed eyes. Make and explain the topical diagnosis.

34. Left-side hemianaesthesia, sensory hemiataxia are detected. Make and explain the topical diagnosis.

35. Left-side hemianaesthesia, sensory hemiataxia, hemianopia and hemialgia are observed. The pain in the left-hand half of the body is excruciating, poorly localized, not stopped by analgesic drugs. It becomes severer at rest and decreases when the patient is distracted. Name the syndrome. Make the topical diagnosis.

36. The patient has left-side hemianaesthesia, sensory hemiataxia and hemianopia. Make the topical diagnosis.

37. Left-side hemiplegia, hemianaesthesia and hemianopia are revealed. Make the topical diagnosis.

38. The patient has severe smarting pain and vesicular rash on the right half of the face. Make the topical diagnosis.

39. Right side anosmia has been detected. The nasal mucosa is not damaged. Make the topical diagnosis.

40. Describe the clinical features of Argyle-Robertson syndrome.

41. What is necessary to examine to survey the optic nerves function?

42. The patient has right side ptosis, whose appearance was preceded by diplopia when looking left and straight. The sharply mydriatic pupil and absence of its response to light and accommodation are detected after passive eyelid raising. The right eyeglobe is diverted outside (divergent strabismus). The motions of the eye globe to the inside and upwards are limited downwards. Make the topical diagnosis. Explain the described signs.

43. The right eye globe is turned to the nose (convergent strabismus), double vision of objects is seen when looking right. Make the topical diagnosis.

44. The patient has doubling of objects when looking down. The right eye movements are limited downwards. Make the topical diagnosis.

45. Which muscles are innervated by the trigeminal nerve? Which reflexes are reduced in case of lesion of the trigeminal nerve?

46. The patient has right mimic paralysis: the angle of mouth is sharply dropped, the nasolabial fold is flattened, the mouth is twisted to the left, the palpebral fissure is extended, the right eye cannot be closed (lagophthalmia), Bell's sign. The fluid food streams from the right angle of the mouth. The forehead folds are not formed on the right side. There is right eye lacrimacia, right ear hyperacousia and taste loss on the anterior 2/3 of the right half of the tongue. Describe the neurological disorders. Make the topical diagnosis.

47. The patient has right mimic paralysis: the mouth angle is dropped, the nasolabial fold is flattened, the mouth is twisted to the left, the palpebral fissure is extended (lagophthalmia), Bell's sign. The forehead folds are not formed on the right side. There is right eye lacrimacia. Hearing and taste are preserved. Describe the neurologic disorders. Make the topical diagnosis.

48. The right mimic paralysis is detected - the angle of mouth is dropped, the nasolabial fold is flattened, the palpebral fissure is extended (lagophthalmia), Bell's sign. The forehead folds are not formed. There is right eye lacrimacia. Hearing is preserved. The taste on the front 2/3 right half of the tongue is lost. Describe the neurologic disorders. Make the topical diagnosis.

49. The right mimic paralysis is detected – the mouth is twisted to the left, the angle of the mouth is dropped, the nasolabial fold is flattened, the palpebral fissure is extended, lagophthalmia, Bell's sign. The forehead fold is not formed. There is right eye dryness and right ear hyperacousia. The taste on the front 2/3 right half of the tongue is lost. Describe the neurological disorders. Make the topical diagnosis.

50. The right mimic paralysis is detected - the mouth angle is dropped, the nasolabial fold is flattened, the palpebral fissure is extended, lagophthalmia, Bell's sign. The forehead folds are not formed on the right. There is right eye dryness, taste loss on the anterior 2/3 of the tongue and right ear hearing loss. Describe the neurological disorders. Make the topical diagnosis.

51. The right mimic paralysis is detected - the angle of mouth is dropped, the nasolabial fold is flattened, the palpebral fissure is extended, lagophthalmia, Bell's sign. The forehead folds are not formed. There is hyperlacrimation and dysacusis on the right side. The strength of the left arm and legs is reduced. The tone of the arm flexors and the leg extensors is increased. The left tendinous and periosteal reflexes are increased. The left abdomen skin reflexes are absent. Babinski's reflex is present on the left leg. Describe the neurological disorders. Make the topical diagnosis.

52. The patient complains of tinnitus and ringing in the left ear, hearing impairment on the left, dizziness in the form of equilibrium loss and subjects rocking. The bone conduction on the left is decreased. Describe the neurological disorders. Make the topical diagnosis.

53. The patient has tongue immobility, muscles atrophy and fibrillar jerks. Though speech is absent, however written contact is possible. Swallowing is preserved. Describe the neurologic disorders. Make the topical diagnosis. What are the differences between the central and peripheral tongue paralysis?

54. The tongue deviation to the left is detected. There is atrophy of the left tongue half and fibrillar jerks. Describe neurologic disorders. Make the topical diagnosis.

55. There is narrowing of the left palpebral fissure and the pupil, as well as left eye globe retraction. Describe the neurological disorders. Make the topical diagnosis. What is the name of the described syndrome?

56. The patient's head drops down onto the chest. Head rotations are impossible. The shoulders are dropped, arms rising above the horizontal level is sharply reduced. «Scapula alary». Atrophy of the sterno-cleido-mastoidal and trapezoidal muscles is revealed. Describe the neurological disorders. Make the topical diagnosis.

57. The patient has a smarting pain in the right half of face, hyperemia and its sweating. Horner's syndrome in the right side, hyperpathia on this face half and neck, asymmetry of pulse and arterial pressure are observed. Describe the neurological disorders. Make the topical diagnosis.

58. The patient complains of the excruciating paroxysmal pains lasting for some seconds, and high sensitivity of the tongue root, soft palate, tonsil and pharynx on the right side. Attacks of pains are provoked by speaking, laughing, coughing, yawning, swallowing. The reflexes of the soft palate and the pharynx back wall are preserved. Describe the neurological disorders. Make the topical diagnosis.

59. The patient's speech is muffled, obscure, slurred by rhinophonia. He uses only soft gruel-like food, as fluid food causes excruciating coughing and choking. The tongue movements are sharply limited. The tongue atrophy and fibrillar jerks of its muscles are observed. The soft palate is immobile. The pharyngeal reflexes and soft palate reflexes are reduced. Describe the neurological disorders. Make the topical diagnosis. Name the described disorder.

60. The patient has right hemianopia with extra field of vision. The eye ground is normal. The pupils photoreaction from the blind halves of the retina is present. The optic discs are pink, the borders are clear. Describe the neurological disorder. Make the topical diagnosis. Which types of hemianopia do you know?

61. What is the diagnostic meaning of the optic papilloedema? What diseases does it accompany?

62. The patient has right hemianopia. The pupils photoreaction from the blind halves of the retina is absent. The optic discs are pale,

the borders are clear. Describe the neurological disorders. Make the topical diagnosis.

63. The general epileptic seizure starts with rotation of the head and eyes to the right. Describe the neurological disorders. Make the topical diagnosis.

64. The patient feels short-term sensation of objectionable odors before a general epileptic seizure: burnt meat, rotten eggs. Describe the neurological disorders. Make the topical diagnosis.

65. The patient has periodical jerks of the right arm and muscles of the right half of the face, which are not accompanied by consciousness loss. Describe the neurological disorder. Make the topical diagnosis.

66. The patient has diplopia, right partial ptosis and mydriasis. The right eye globe is deviated outward. Eye movements inwards, upwards and downwards are limited. Voluntary movements of the left arm and leg are absent; the muscle tone of the arm flexors and the leg extensors is increased. The left tendon and periosteal reflexes are higher than on the right. Babinski's reflex on the left side is positive. Describe the neurological disorder. Make the topical diagnosis.

67. What is the name of the syndrome appearing in case of lesion of the brainstem half? What are its clinical signs?

68. The patient’s tongue is diverted to the left; its left half demonstrates muscle atrophy. Central paralysis of the right arm and leg is observed. Describe the neurological disorder. Make the topical diagnosis.

69. The patient has euphoria. He does not evaluate his state critically. He is silly and makes scabrous jokes. His memory is impaired. He is slovenly and displays no self-control. The oral automatism reflexes and grasp reflexes are observed. Make the topical diagnosis.

70. The patient has astereognosis, apraxia, acalculia, alexia. He is a right-handed person. Make the topical diagnosis.

71. Pain and temperature sensations are lost on the left half of the face. All sorts of sensitivities are absent on the right side from the occipital level downwards (on the neck, trunk, extremities). Make the topical diagnosis.

72. The patient has bitemporal hemianopia. Make the topical diagnosis.

73. The patient walks by small steps, his trunk is deviated forwards, his hands and legs are half-bended. The face is mask-like. Speech is monotonic, low and fading. Stereotypic fingers tremor like «money counting» is observed. The extremities muscle tone is increased diffusely, the sign "of «cogwheel" is positive. Make the topical diagnosis. What is the name of the described syndrome?

74. The child has fast, arrhythmic, involuntary, jerks of the extremities and the trunk. He grimaces, smacks, frequently puts out the tongue. The muscular tone of the extremities is reduced. Make the topical diagnosis. What is the name of the described syndrome?

75. Characterize hyperkynesias. When do they appear?

76. List the forms of the extrapyramidal hyperkynesias.

77. The patient has forgotten how to dress, cannot use a cup, a spoon. He is dressed and fed. Where is the lesion? What is the name of the described syndrome?

78. Which signs are typical of the occipital lobe lesion?

79. Characterize aphasia. When does it appear? List types of aphasias.

80. The patient has horizontal nystagmus when abducting the eye globes sideways. The gait is shaky («drunk»). The patient walks placing his legs widely; unsteadiness is increasing in lateral turning, especially to the right. Romberg’s test is positive on the right side.

Discoordination, intention tremors in the course of finger-to-nose and pointer tests, adiadochokinesis are detected on the right side. He can't make knee-to-heel test by the right leg. There is a change in the handwriting (megalography). The muscular tone is reduced in the right hand and leg. The joint-muscle sensation is preserved. Paresis of the extremities is absent. Describe the disorders. Make the topical diagnosis.

81. The patient has horizontal nystagmus when abducting the eye globes sideways. The strength of the arms and legs is preserved. He can't walk and stand independently. He falls to different sides. The tendon and periostal reflexes are low. The muscular tone of all extremities is reduced. Sensitivity is not impaired. Describe the disorders. Make the topical diagnosis. What diseases can be accompanied by the described symptoms?

82. The patient has scanning speech. Horizontal nystagmus is detected. He has shaky ("«drunk") gait and instability when taking Romberg's test. Discoordination and intention tremor in the course of finger-to-nose, pointer and heel-to-knee tests, adiadochokinesis in both sides, megalography are observed. Associated movements are missing (asynergy). There is extremities muscle hypotonia. The tendon and periosteal reflexes are reduced. The arms and legs paresis is absent. Sensitivity is preserved. Describe the disorders. Make the topical diagnosis.

ANSWERS FOR CLINICAL CASES

1. Peripheral paralysis of the right arm. Lesion of the anterior horns of the spinal cord at the level C5-TH1 on the right side.

2. Peripheral paresis of the deltoid and bicipital muscles, central paresis of the hand and leg left side. Combined lesion of the anterior horns and pyramidal tract on the left side at the level C5-C6.

3. Peripheral law paraparesis. Lesion of the lumbar plexus.

4. Central law paraplegia, total conductory anesthesia from the Th12 segment, central neurogenic bladder. Complete transversive lesion of the spinal cord at the level Th12.

5. Peripheral paresis of the left hand, total anesthesia in the dermatomes C8-Th1. Lesion of the law part of the left brachial plexus (C8-Th1).

6. Central law paraparesis, conductor superficial anesthesia from the level Th10, central neurogenic bladder. Transversive lesion of the anterior half of the spinal cord at the level Th10.

7. Monoparesis, paraparesis, threparesis, tetraparesis, hemiparesis. The paresis is decrease of muscle force and restriction of volume kinesias. Paralysis is absence of any voluntary movements.

8. Cervical thickening: peripheral paresis of the arms and central law paraparesis, total conductory anesthesia from the C5 segment, central neurogenic bladder. Thoracic part: central law paraparesis, total conductory anesthesia from the affected segments, central neurogenic bladder. Lumbar thickening: peripheral law paraparesis, total conductory anesthesia from the lumbar segments, central neurogenic bladder.

9. Left central hemiparesis.

10. Ulnar nerve – «claw hand», median nerve – «monkey hand», radial nerve – «wrist dropped».

11. The femoral nerve. The Wassermann symptom is typical for the femoral nerve lesion.

12. Lesion of the left sciatic nerve.

13. Upper cervical segments: central tetraplegia, total conductory anesthesia from the neck, central neurogenic bladder. Cervical thickening: peripheral paresis of the arms and central law paraparesis, total conductory anesthesia from the C5 segment, central neurogenic bladder. Thoracic part: central law paraparesis, total conductory anesthesia from the affected segments, central neurogenic bladder. Lumbar thickening: peripheral law paraparesis, total conductory anesthesia from the lumbar segments, central neurogenic bladder. Conus: peripheral neurogenic bladder? Anesthesia in the anogenital region.

14. Central paralysis of the left leg.

15. Combined lesion of the anterior horns and pyramidal tract at the level C5-Th1 on the both sides.

16. Peripheral proximal paresis of the right arm, radicular sensory disorder in the dermatomes C8-Th1. Lesion of the upper part of the brachial plexus. The paralysis named after Dejerine-Klumpke.

17. Central paresis of the left leg, segmentary disorder of pain and temperature sensitivities at the level Th5 left side, conductory disorder superficial sensitivity right side from the level Th7. Lesion of the posterior horns and lateral funiculus of the spinal cord at the level Th5 left side. The disorder is named after Brown-Secqard.

18. Peripheral paralysis of the left leg. Lesion of the anterior horns of the spinal cord at the level L1-S2 on the left side.

19. Central paresis of the right leg. Differential diagnosis is between a tumor, stroke, head injury.

20. Central tetraplegia, total conductory anesthesia below C2-C3, central neurogenic bladder. Complete transection of the spinal cord at the level C2-C3.

21. Severe shooting pains in the legs and perineum's area which are sharply intensifying when coughing and sneezing, peripheral law paraparesis, hypoesthesia of all sorts of sensitivity in the lower extremities and in the perineum, incontinence of urine and feces.

22. Reflexes of legs are lost ore decreased.

23. Contralateral central hemiparesis.

24. Babinski's and Rossolimo's reflexes indicate lesion of the pyramidal tract.

25. Complete transection of the spinal cord at the level C5-Th1.

26. Complete transection of the spinal cord at the level L5-S2 (epiconus).

27. Complete transection of the spinal cord at the level C2-C3. Conductory sensory disorder is characterized by disturbance of some jre all sensitivities below the level of affected segment.

28. Polyneural total hypoesthesia of the arms and legs, sensory ataxia. Diffuse lesion of the peripheral nerves of the arms and legs.

29. Peripheral neurogenic bladder, total anesthesia in the anogenital region. Lesion of the conus of the spinal cord (S3-S5).

30. Lesion of the cauda equine.

31. Lesion of the posterior roots Th10-Th12 right side.

32. Lesion of the posterior horns of the spinal cord right side at the level C5-Th1.

33. Lesion of the posterior funiculus left side at the level Th10.

34. Lesion of the sensory pathways in the posterior part of the right internal capsule.

35. Lesion of the right thalamus.

36. Lesion of the posterior part of the right internal capsule.

37. Total lesion of the right internal capsule.

38. Herpetic lesion of the right trigeminal ganglion.

39. Lesion of the olfactory bulb ore olfactory tract right side.

40. Absence of the direct and concomitant reactions of pupil to light, presence of reactions to convergence and accommodation.

41. Examination of the optic nerve functions include: visual acuity, visual fields, eyeground.

42. Lesion of the right oculomotor nerve. Paresis of the levator palpebre, rectus superior, inferior, medial muscles, inferior oblique muscle, sphincter of the pupil and ciliare muscle.

43. Lesion of the right abducens nerve.

44. Lesion of the right trochlear nerve.

45. Masticatory muscles are innervated by the trigeminal nerve. Mandibular reflex is reduced in case of lesion of the trigeminal nerve.

46. Peripheral mimic paresis, eye lacrimacia, hyperacousia right side, ageusia on the anterior 2/3 of the right half of the tongue. Lesion of the right facial nerve above the stapedius nerve.

47. Peripheral mimic paresis, eye lacrimacia right side. Lesion of the right facial nerve below the chorda tympani.

48. Peripheral mimic paresis, eye lacrimacia right side, ageusia on the anterior 2/3 of the right half of the tongue. Lesion of the right facial nerve below the stapedius nerve.

49. Peripheral mimic paresis, xerophthalmia, hypoacousia right side, ageusia on the anterior 2/3 of the right half of the tongue. Lesion of the right facial nerve above the nerve petrosus mayor.

50. Peripheral mimic paresis, xerophthalmia, hypoacousia right side, ageusia on the anterior 2/3 of the right half of the tongue. Combined lesion of the right facial and cochleovestibular nerves in the porus acousticus internus.

51. Peripheral mimic paresis right side, left central biparesis. Lesion of the ventrocaudal part of the pons right side (nucleus of the facial nerve and the pyramidal tract).

52. Hypoacousia left side, vestibular ataxia. Lesion of the left cochleovestibular nerve.

53. Peripheral bilateral glossoplegia. Bilateral lesion of the nuclei of the hypoglossal nerve. Peripheral tongue paresis produces atrophies and fascicular jerks of the muscles, central paresis does not.

54. Peripheral paresis of the left half of the tongue. Lesion of the nucleus of the left hypoglossal nerve.

55. Ptosis, myosis, endophthalmus. Lesion of the sympathetic innervation of the left eye. The syndrome is named after Horner.

56. Peripheral bilateral paresis of the sternoclaidomastoideal and trapezoid muscles. Bilateral lesion of the accessorial nerve.

57. Lesion of the superior cervical ganglion right side.

58. Glossopharyngeal neuralgia. Irritation of the right glossopharyngeal nerve.

59. Bilateral peripheral paresis of the pharynx, larynx. Soft palate, tongue. Bilateral lesion of the nuclei IX, X, XII cranial nerves. Bilateral bulbar syndrome.

60. Central right hemianopia. Lesion of the optic radiation ore visual cortex in the occipital lobe left side.

61. Papilloedema indicates raised intracranial pressure.

62. Tractus right hemianopia. Lesion of the left optic tract.

63. The partial motor adversive seizure with secondary generalization. Irritation of the back portion of inferior frontal gyrus (field 8) left side.

64. The partial sensory olphactory seizure. Irritation of the parahyppocampal gyrus.

65. The partial simple motor clonic seizure. Irritation of the law and middle portions of the precentral gyrus of the frontal lobe left side.

66. Signs of the oculomotor nerve lesion right side, left central hemiparesis. Lesion of the brain peduncle right side (Veber syndrome).

67. The syndrome appearing in case of lesion of the brainstem half is named alternating syndrome. Clinical signs of the alternating syndrome include symptoms of the one several cranial nerves palsy same side and central hemiparesis ore hemihypoesthesia opposite side.

68. Peripheral paresis of the left half of the tongue, central right biparesis. Lesion of the left medial law part of the medulla (nucleus of the hypoglossal nerve and pyramidal tract).

69. Lesion of the left frontal lobe.

70. Lesion of the left parietal lobe.

71. Lesion of the left half of the tegmentum of the pons (spinal nucleus of the trigeminal nerve and the spinnothalamic tract).

72. Lesion of the central part of the optic chiasm.

73. Bilateral parkinsonic syndrome. Bilateral lesion of the substantia nigra of the midbrain.

74. Bilateral lesion of the striate body. Chorea hyperkinesia.

75. Hyperkinesias are a forced involuntary movements that destructs voluntary intended movements. This syndrome develops in the neostriatum is damaged.

76. Extrapyramidal hyperkynesias are of different kinds: athetosis, chorea, spasmodic torticollis, torsion dystonia, ballism, myoclonia.

77. The name of the syndrome is apatic-abulic. The lesion of the frontal lobe.

78. The lesion of the occipital lobe produces central contralateral hemianopia ore loss of upper ore law quadrants of the visual fields; visual agnosia and alexia (dominant hemisphere lesion). Irritation of the occipital lobe produces simple ore complex visual hallucinations ore illusions.

79. Aphasia is a disorder of speech due to the focal lesion of the cerebral cortical fields. Aphasia appear because a stroke, brain tumor, head injury, encephalitis, brain abscess. Types of aphasias are motor, sensory, amnestic, semantic and total (global).

80. Cerebellar ataxia (static-locomotor and dynamic right side). Lesion of the vermis and right hemisphere of the cerebellum.

81. Cerebellar static-locomotor ataxia. Lesion of the vermis of the cerebellum.

82. Cerebellar ataxia (static-locomotor and dynamic bilateral). Lesion of the vermis and both hemispheres of the cerebellum.

REFERENCE

1. Голубев В.Л. Неврологические синдромы [Текст] : рук. для врачей / В. Л. Голубев, А. М. Вейн. - 4-е изд. - М. : МЕД-пресс-информ, 2012. - 734 с.
2. Методология клинического диагноза в неврологии и нейрохирургии [Текст] : учеб. пособие для самост. работы студентов лечеб. фак. / В. А. Жаднов [и др.] ; Ряз. гос. мед. ун-т. - Рязань: РИО РязГМУ, 2015. - 135 с. - имеется электрон. док.
3. Гусев Е.И. Неврология и нейрохирургия. В 2 т. Т. 1. Неврология [Электронный ресурс]: учебник под ред. А.Н. Коновалова, А.В. Козлова. - 4-е изд., доп. - М. : ГЭОТАР-Медиа, 2015. - ISBN 978-5-9704-2901-3. - Текст (визуальный): электронный.
4. Гусев Е.И. Неврология и нейрохирургия [Текст] : в 2 т.: учебник для студентов учреждений высш. проф. образования. Т. 2. Нейрохирургия / Е. И. Гусев, А. Н. Коновалов, В. И. Скворцова. - 4-е изд., доп. - М.: Изд. группа "ГЭОТАР-Медиа", 2015. - 403 с.
5. Скоромец А.А. Нервные болезни [Текст] : учеб. пособие для системы послевуз. проф. образования врачей / А. А. Скоромец, А. П. Скоромец, Т. А. Скоромец. - 8-е изд. - М. : МЕДпресс-информ, 2014. - 564 с.
6. Триумфов А.В. Топическая диагностика заболеваний нервной системы [Текст] : крат. рук. / А. В. Триумфов; авт. вступ. ст. Е.И. Гусев. - 17-е изд. - М. : МЕДпресс-информ, 2012. - 262 с.
7. Adams Victor's Principles of Neurology / eds.: Victor Maurice, Allan H. Ropper, Raymond D. Adams. – 7th ed. – New York: McGraw-Hill Professional, 2000.
8. Biller J. Practical Neurology / J. Biller. – 2002. – P. I.
9. Biller J. Practical Neurology / J. Biller. – 2002. – P. II.
10. Duus Peter. Topical Diagnosis in Neurology / Peter Duus. – Stuttgart;New York : Thieme,1998.
11. Lindsay K.W. Neurology and neurosurgery illustrated. Part.1 / K. W. Lindsay, L. Bone, R. Callander. - 3rd ed. - Edinburgh;London;Philadelphia : Churchill Livingstone, 1997. - 212p.
12. Lindsay K.W. Neurology and neurosurgery illustrated. Part.2 / K. W. Lindsay, L. Bone, R. Callander. - 3rd ed. - Edinburgh;London;Philadelphia : Churchill Livingstone, 1997.

13. Zhadnov V.A. Clinical syndromes and topical diagnosis in neurology and neurosurgery / V.A. Zhadnov, O.V. Evdokimova; RyazanSMU. – Ryazan: RSMU, 2018. – 112 p.