**PATHOPHYSIOLOGY OF NERVOUS SYSTEM**

According to the theory of nervism (nervosism) physiological as well as pathological processes in the organism occur with participation of the nervous system. The nervous system reacts to any changes in the internal environment of the organism as well as in the external environment and the first responds to the action of the pathogenic factors. As the main control system of the organism, the nervous system in various forms participates in the pathogenesis of each disease. Mobilization of the defensive - compensatory resources of the organism form the basis of activity of the nervous system in pathology. But in the course of numerous diseases the nervous system is affected, its defensive - compensatory abilities weaken, and the nervous system itself becomes the source of the pathological reflexes which are injurious for the organism. Disturbances in the nervous system functions affect activity of different organs and systems. So, one of the important tasks of the pathophysiology is to differentiate the defensive - compensatoty reactions from the pathological reactions in the pathology.

In disorders of the central nervous system activity, correlation between the processes of excitation and inhibition are disturbed. Metabolic changes in the cerebral tissue form the basis of these disturbances.

Nervous system activity disturbances may be of organic or functional character. In organic disturbances the structural changes in nerve centers or lesions of nerve fibers are available, whereas in functional disturbances an attempt to find any morphological changes in the nervous system fails, as if they are absent. But from the dialectical point of view this is impossible. Because from the standpoint of principle of causation every change in any function is conditioned by the morphological change in the corresponding organ. So, the term "functional disease" reflects imperfection of research methods which cannot reveal the slight morphological changes that form the basis of the functional disturbances. In due course, technical improvement makes it possible to detect these changes.

Really, some quarter of a century ago the late pathoanatomist, academician J. Y. Huseynov demonstrated on the screen two microphotographies: the nerve of the practically healthy person and that of neurotic under the electron microscope. The first nerve was as smooth as ivory, whereas on the second nerve one could see tiny thorns. Thus, actually the neurosis ceased to be the functional disease.

It is impossible categorically separate the functional and organic changes. Outcome of the influence of the pathogenic factor on the nervous system depends on the power of the factor, duration of its action and the functional state of the nervous system. For example, acute cerebral ischemia connected with brief vascular spasm results in temporary functional changes, whereas prolonged spasm causes irreversible organic changes in the brain.

Disturbances in the nervous system functions arise under the influence of various pathogenic factors which affect the nervous system directly or by reflex way, or cause changes in the higher nervous activity through the second signaling system.

Specific and non - specific injurious factors may affect neurons:

1) specific factors affect only the nerve cells (neurons): neurotoxins, pharmacological substances of neurotropic character, specific autoantibodies, etc.;

2) non - specific factors may affect cells of all tissues of the organism: hypoxia, disturbances in microcirculation, factors accelerating peroxide oxidation of lipides, etc.

The pathogenic factors affecting the nervous system may be divided into the following groups:

I. Exogenous factors:

1) physical factors - mechanical and electric trauma, ionizing radiation;

2) chemical factors - compouds of mercury and manganese, carbon monoxide, cyanides, chloroform, methyl and ethyl alcohols, some drugs;

3) biological factors;

a) microorganisims and viruses (lepra, rabies);

b) microbial toxins (botulism, tetanus);

c) poisons of animals and insects;

d) plant toxins.

4) alimentary factors - starvation, vitamin deficiency (especially that of B group);

5) conditioned pathogenic stimuli;

6) verbal stimuli and negative emotions influencing the organism thorough the second signalling system.

Some pathogenic factors affect mainly certain parts of the nervous system. For instance, in mercury poisoning lesion is localized in the area of celiac plexus, in manganese poisoning-in striate body; in carboxyhemoglobinemia-pale globe and black substance are damaged first. Such selectivity in the action of the pathogenic agents is connected with special features of metabolism, membrane permeability and character of blood circulation in different neurons.

The virus of poliomyelitis reproduces and moves along motor nerves and reaches the central nervous system, the virus rabies (lyssin) moves along axons of motor and sensory nerves into spinal cord and brain. Tetanotoxin moves along axons of motor nerves. Many organic (methyl alcohol in optic tract) and inorganic (ascending arsenous neuritis) poisons also spread in nerve trunks.

A number of pathogenic agents may affect the organism under special conditions. For example, viruses of grippe can penetrate the central nervous system only when permeability of the hematoencephalic barrier is increased, and viruses of rabies - when the nerve fibers are damaged.

II. Endogenous factors:

1) hereditary diseases (Down’s syndrome, phenylketonuria) and hereditary disposition to neuropsychical diseases (epilepsy, schizophrenia);

2) disturbances in blood circulation (hyperemia, spasm, sclerosis, thrombosis and embolism in cerebral vessels);

3) tumors, exudates and cicatrices resulted from inflammatory processes, edema;

4) metabolites accumulated in the organism as a result of disturbances in the endocrine system and metabolism (diabetes mellitus, hyperthyrosis, hypothyrosis);

5) age and sexual peculiarities of the organism.

III. Secondary pathogenic factors - come into being in the nervous system itself under the influence of exogenous and endogenous factors (metabolites, mediators, etc.that are formed in the nervous tissue under pathological conditions).

In the end, hypoxia forms the basis of many disturbances in the nervous system activity.

Lesions of neurons, nerve fibers and synapses cause disturbances in their main functions:

1) excitability of the neurons;

2) conduction of impulses along nerve fibers;

3) transmission of nerve impulses from one neuron to another one and to working organ;

4) processes of inhibition in the nerve cells and synapses;

Lesion of nerve cell or its axon causes lengthening of chronaxy, whereas lability is decreased. Therefore, to excite the damaged nerve cell, more protracted action of the electric current (or more powerful current) is required. The following factors decrease excitability of neurons:

1) decreased osmotic pressure in the extracellular medium;

2) ionzing radiation;

3) poisons which weaken the processes of oxidation (cyanides, sulfides);

4) some pharmacological preparations which weaken activity of the sodium - potassium pump (luminal, aminasine);

5) hypoxia;

6) excessive increase of the oxygen content in the medium;

7) cold;

8) severe hyperthermia.

Factors accelerating peroxide oxidation of lipides in the cell membrane ( hypoxia, ionizing radiation, excessive functional strain) damage the membrane and increase its permeability, ions and biologically active substances (enzymes, mediators, peptides) go out of the neuron. This disturbs excitability of the damaged neuron and causes joining of healthy cells into the pathological process.

Excitability of neurons may be increased by moderate rise of oxygen content in the medium and slight hyperthermia.

The main manifestation of the lesion of nerve fibers is disturbed conduction of impulses which may be caused by the following factors:

1) dissection of nerve fibers;

2) lesion of myelinic sheath of nerve fibers (disseminated sclerosis);

3) compression of nerve fibers by tumors or cicatrix;

4) ultraviolet and radioactive rays;

5) bacterial toxins (diphtheria, abdominal typhoid);

6) some viruses (grippe, poliomyelitis);

7) some specific neurotropic poisons (tetradotoxin);

8) hypoxia;

9) cold.

Transmission of nerve impulses from one neuron to another one or to working organ (synaptic transmission) is disturbed under the influence of hypoxia, bacterial toxins, neurotropic poisons, etc. Action mechanism of these factors is different:

1) disturbances in formation of mediator in presynaptic terminal and its secretion into synaptic cleft (hypoxia, fatigue);

2) changes in activity of enzymes breaking down the mediators (eserine and galantamine inibit cholinesterase);

3) blockade of receptors in the postsynaptic membrane (curare and d - tubocurarine combine with cholinoreceptor more firmly than acetylcholine).

Numerous infections and intoxications (grippe, diphtheria) disturb activity of the adrenergic synapses, causing decreased muscular tone and acute vascular insufficiency (collapse).

Mechanism of toxic action of some insecticides and poison gases containing phosphoric organic substances is based on the blockade of breakdown of acetylcholine that is formed in cholinergic synapses. These substances destroy cholinesterase, and acetylcholine accumulates in synaptic cleft causing muscular paralysis and death from paralysis of respiratory center.

Some bacterial toxins, viruses, poisons (tetanotoxin, strychnine, etc.) affect inhibitory synapses. Under their influence the processes of inhilition in motoneurons disappear, and any weak stimulation causes seizure of convulsions. Their action mechanism is different.

1) tetanotoxin blocks up release of mediator from the presynaptic terminal and its secretion into synaptic cleft;

2) strychnine combines with receptor in postsynaptic membrane and takes the place of glycine.

The main functional manifestations of lesions of neurons are: denervation syndrome, pathological lability, pathological parabiosis.

The changes in organs and tissues after their denervation are called denervation syndrome. Two types of denervation are distinguished:

1. Anatomic denervation - is caused by dissection of all nerves of organ or tissue. But actually it is impossible to deprive the tissue of nerves completely, because nerve fibers exist even in the walls of blood vessels supplying the tissue.

2. Chemical denervation - results from action of some pharmacological substances (d - tubocurarine, atropine, nicotine, novocain) and physical methods sharply decreasing metabolic rate in nerve fibers and neurons (hypothermia) which block up conduction of impulses.

Clinical manifestations of denervation are:

1) motor disorders (paralysis, contracture);

2) disorders of sensitivity;

3) trophic disorders (trophic ulcer, dystrophy).

The most severe functional and morpological changes after denervation are observed in skin, mucous membranes, skeletal muscles. In the skin trophic ulcera come into being, epiderm loses its ability to regenerate, development of hairs and blood circulatoin are disturbed. In skeletal muscles metabolism and excitability are disturbed; gradually the muscles are atrophied. So, if the innervation of tissues is not recovered, the changes become irreversible.

Activity of the internal organs is recovered several weeks after denervation, but their reaction to the influence of the control mechanisms is weakened. For instance, heart loses the ability to change its activity according to the needs of the organism, that is, during physical strain heart rate is not increased.

In the organs after anatomic denervation sensibility to the mediator of the dissected nerve is increased. This may be connected with increased number of free receptors in the denervated tissues.

Lability of the excitable tissues may change under the influence of different stimuli. Two types of disturbances of lability are distinguished:

1) pathological inertness;

2) pathological lability.

Pathological inertness is characterized by decreased lability of tissues which after all leads to the inhibition. Reduced functional mobility is connected with inability of tissue to restore its power and plastic reserves. Therefore, excitation and inhibition replace one another at a low rate, and the number of stimuli which can cause excitation in the tissue per unit of time, is decreased.

Pathological lability is characterized by increased functional mobility of tissue, that is, excitation and inhibition replace one another rapidly. Therefore, tissue can respond to a greater number of stimuli per unit of time than normally. But this state cannot be regarded as optimal. Because soon this reaction is weakened.

Parabiosis also may be physiological and pathological. In the development of both physiological and pathological parabiosis the same stages are observed:

I. Primum (the initial stage) - excitability decreases, lability increases.

II. Optimum - is characterized by increased activity; excitation reaches the maximum level, lability descreases.

III. Pessimum - is characterized by decreased activity; excitablity and lability change in parallel:

1) provisory (equalizing) phase - the ability of nerve to respond to rhythmic impusles is reduced with stimulation of any strength, but the reduction has a greater influence on the effect of frequent (strong) than that of infrequent (weak) stimuli, so that the effect of both is almost equal;

2) paradoxical phase - frequent (strong) stimuli are blocked up and cause weak effect, in comparison with which the effect, caused by infrequent (weak) stimuli is stronger.

3) inhibitory phase - both frequent (strong) and infrequent (weak) stimuli do not cause effect.

However, the pathological parabiosis differ from the physiological parabiosis in principle. On the level of any part of the nervous system (nerve cell, nerve trunk, nerve ending) the physiological parabiosis is a reversible process whereas the pathological parabiosis is an irreversible process, and if the factor that causes it, becomes exceedingly powerful, the death of the tissue may occur.

On the level of the whole organism any reaction of parabiosis that limits the adaptative activity of the nervous system, is regarded as the pathological parabiosis, even though it is reversible process. For example, itch caused by eczema is manifestation of the pathological parabiosis, because it results in insomnia and neurosis, decreases ability to work. It is regarded as paradoxical phase of parabiosis, because when scratching violently, the pain is not felt, whereas light touch causes pain.

Duration of phases of the pathological parabiosis is longer than that of the physiological parabiosis.

In order to eliminate the pathological parabiosis, antiparabiotic preparations (for instance, novocain) are used.

A number of disorders of the nervous system are caused by disturbances in interneuronal connections (deafferentation syndrome, disorders in connections between excitation and inhibition).

Cessation of impulses coming to the central nervous system by sensory nerves is called deafferentation. Deafferentation syndrome includes, besides loss of sensitivity, a number of other disturbances. Because deafferentation deprives the central nervous system of feedback mechanism without which precise activity of the central nervous system is impossible.

Experimental model of deafferentation is reproduced by the dissection of posterior roots of the spinal cord. This causes temporary paralysis in the corresponding extremity. Then its movements are recovered owing to the influences from the intact extremities and higher parts of the central nervous system. But muscular tone is weak, coordination of movements is disturbuted. Thanks to the increased excitability, the motoneurons in the corresponding spinal cord segments respond to stimuli of different character (resiratory, exteroceptive, proprioceptive), and the extremity begins to move, for instance, in the rhythm of the respiratoty movements of the chest. The slight movement of the head or trunk causes sharp extension of the extremity. It may move also under the influence of stimuli acting on other extremites. Animals treat this extremity as if it is of strange body; rats may even bite of and eat it.

As a result of deafferentation blood circulation and trophicity of tissues are also affected, and disturbances in microcirculation, edema, hemorrhage, dystrophy and even trophic ulcer occur.

Deafferentation is one of the main symptoms of tabes dorsalis (caused by syphilis) when the posterior columns of the spinal cord are affected. Such a person maintains his vertical position only thankes to the control of eyes, and when closing the eyes, he falls at once.

Structural changes in the nervous tissue, dissection of conduction tracts and nerve trunks (trauma, insult) are the main causes of the disturbances in the interneuronal connections. They may cause central and peripheral paralysis.

All types of the nervous system pathology result from disturbances of the main nervous processes, that is, excitation and inhibition, and connections between them.

Usually in lesions of different parts of the central nervous system the processes of inhibition are changed the first; in different nervous structures weakening of inhibition and hyperreactivity are observed.

Disturbances in the synaptic inhibition are of great significance in the pathology of the interneuronal connections.

Sometimes in certain neurons of the central nervous system the inhibitory processes are sharply weakened, and powerful stream of impulses are formed in those neurons. This may be connected with the influence of substances that selectively weaken the inhibitory processes or strengthen processes of excitation. Such group of neurons which generate stream of impulses that do not correspond to the conditions, was called by Kryzhanovsky “generator of pathologically strenghened excitation”. 2 stages are distinguished in the formation and activity of this generator:

I - power of the inhibitory processes in the neurons of the generator is changed slightly, and the generator is activated only under the influence of stimuli that are characteristic of itself;

II - excitation threshold of neurons sharply falls, inhibitory processes are weakened to still greater extent, and therefore, generator is activated under the influence of various (frequently - occasional) stimuli; high - frequency impulses may be generated without any stimulation (spontaneous paroxysms).

Generator of pathologically strengthened excitation is basis of many nervous discorders.

In rest and in active state neurons always experience inhibitory influences. When neurons are excited, the inhibitory processes become weaker. Such an disinhibition is of the physiological character, because it is controlled and corresponds to the necessary level of the neuron’s activity.

In disinhibition of the pathological character neuron becomes hyperactive and goes out of the control. This occurs as a result of direct damage to inhibitory mechanisms, selective action of some toxins (tetanic toxin, strychnine, etc).

Inhibition deficiency and disinhibition take place practically in all forms of pathology of the nervous system. So, they refer to the typical pathological processes. Inhibition deficiency is of great importance in formation and activity of generator of pathologically strengthened excitation.

The typical experimental syndrome of disinhibition is decerebrate rigidity.

Formation of the generator of pathologically strengthened excitation not always causes pathological reactions. If spreading of the exctitation that is generated by it, is blocked up by the mechanisms of the inhibitory control, the generator becomes functionally isolated and does not cause systemic pathological effects. Pathology comes into being when the section of the central nervous system that is hyperactivated by the action of generator, actively influences other structures of the centrel nervous system, draws them into the pathological reaction and unites them in the new, pathodynamic organization - pathological system. Such a section of the central nervous system is called the pathological determinant, because it determines the character of activity of the pathological system. So, the pathological determinant in the nervous system is the changed structure of the central nervous system which forms the pathological system and determines the character of its activity (Kryzhanovsky). Any structure of the central nervous system (section, nucleus, the aggregate of nuclei, the nervous center) may become the pathological determinant.

Pathological phenomena developing on the level of the central nervous system include pathological system, pathological dominant, hysteriosis, protective inhibition, pathological reflexes. These are connected with disturbances in interrelations between the nerve centers, and are called the pathological system phenomena.

Lesion of certain part of the central nervous system causes changes in connections between this part and intact nervous structures. The pathodynamic complex that is formed as a result of interconnections between affected and intact nervous structures was called pathological system by Kryzhanovsky. Lesion plays a part of cause and conditions for formation of the pathological changes in the nervous system, but development of the pathological process depends on activity of the pathological system.

Unlike the physiological systems, activity of the pathological system is not of adaptive character; it disturbs activity of organs controlled by the nervous structure where this pathological system is formed. The pathological system weakens function of physiological systems and disturbs associative activity of the brain.

So, the pathological system causes deadaptation in the organism or exerts direct pathogenic influence. It may spread over other parts of the central nervous system. In such cases its resistance to the influence of remedies is increased.

The main clinical manifestations of the pathological system are syndromes of the hyperactive character (pain syndromes of the central origin, hyperkinesis, pathological reflexes, etc).

Lesions and diseases of the nervous system result in formation of the pathological dominant phenomena of different character.

Usually the nerve centers controlling the activities that are important for concrete life situations, become dominant. So the physiological dominant promotes performance of the organism’s finctions and its adaptations to the environment. But the pathological dominant weakens the organism’s adaptability, that is, limits its compensatory possibilities, diminishes ability to work. It causes prolongation of the disease and frequents recidivations.

An example of the pathological sensory dominant is causalgia (burning pain) which appears as a result of injury to nerve trunk. Continuous painful impulses form focus of the stagnant excitation in corresponding areas of the cerebral cortex and subcortex. Violent pain sensation is still more strengthened by additional outside irritants (tactile, light, sound).

Pathological motor dominant is observed in striopallidal system as a result of grippe, contusion, etc. This stagnant focus of excitation spreads over brain stem and spinal cord, and causes continuous tremor in muscles of neck, extremities, trunk. Trembling increases during rapid breathing and voluntary movement, thanks to impulses coming to dominant center from respiratory center and cerebral cortex.

In order to eliminate pathological dominant, another (opposite) focus may be formed.

Hysteriosis of nerve centers is a type of changed reactivity and lability. It is caused by prolonged stimulation of a sensory nerve which results in inhibition in neurons of corresponding reflex arc, and increased excitability in other reflex arcs (increased reactivity and lability of the nerve centers). Therefore, weak subliminal stimulation of another sensory nerve causes stronger reflex reaction. Hysteriosis is observed in tetanus, rabies, strychinine poisoning, some types of electric trauma, etc.

Protective inhibition is particularly important in disorders of the higher nervous activity: exceedingly strong stimuli cause inhibition. This protects neurons from the injurious action of extraordinarily strong and prolonged irritants. Origination of the protective inhibition depends not only on the power of the stimulus, but also on the state of the cerebral cortex; it comes into being more easily in individuals with weak nervous system.

All the reflex reactions which restrict adaptation of the organism and its equilibration in the external environment, are called pathological reflexes. Pathological reflexes result from functional as well as organic (tumor, scar, hemorrhage) changes in the central nervous system. Most of the pathological reflexes are connected with elimination of the inhibitory influences of the reticular formation on the motor centers of the spinal cord and strengthening of the activating influences. The following types og the pathological unconditioned reflexes are distinguished:

1. Inverted reflexes - are based on the formation of dominant focus in the nerve center (as a result of injury or compression of nerves, poisoning with tetanotoxin). For instance, in the case of dominant focus in the center of the extensor of the hand, reflex excitation of the motoneurons of the flexor (caused by its strained tendon) supports the dominant center. So, strained tendon of flexor causes extension instead of flexion.

2. Reflexes with extraordinary projection - are connected with removal of the inhibitory influences of the higher parts of the central nervous system: Babinski’s reflex (Babinski sign), pathological sucking reflex, grasping reflex, etc.

Babinski’s reflex (toe phenomenon) is demonstrated when a firm tactile stimulus is applied to the laleral sole of the foot: the great toe extends upward, and the other toes fan outward. This is in contradistinction to the normal effect in which all the toes bend downward. Bakinski’s phenomenon may be caused by transection of the foot portion of the corticospinal tract, and is not observed when damage occurs in the noncorticospinal portions of the motor control system without involving the corticospinal tract.

3. Reflex palsy - develops in the muscles with intact motor innervation as a result of inhibition of their motoneurons (in the spinal cord) by impulses from damaged sensory nerves. For instance, lesion or formation of scar in the area of the sensory branches of the ulnar nerve cause reflex palsy in the muscles that are innervated by this nerve.

4. Pathological reflex contracture - arises on the background of stagnant dominant focus of extitation in the center of the flexors (in the spinal cord) caused by painful impulses from inflamed joint or area of fracture. This is defence reflex creating the most comfortable position for the sore extremity. Protracted flexion develops into contracture.

Disorders of motor functions of the nervous system are based on lesions of cortical motor centers, corticspinal tracts, spinal cord motoneurons, motor nerve fibers, as well as sensory nerves and so on. The causes of motor disorders may be inflammatory changes (mainly of an infectious origin), traumata, hemorrhages, thrombosis, embolism, or functional changes (hysteria, psychical trauma). The main forms of the motor disorders connected with the nervous system pathology are the following:

1) loss of motor functions (paralysis, paresis);

2) hyperkinesia (hyperkinesis);

3) disturbances in coordination of movements.

Total loss of motor functions is called paralysis. It results from complete interruption of the spread of motor impulses and is manifested in total inability to perform voluntary movements. Incomplete loss of motor functions, that is, weakening of voluntary movements due to incomplete interruption of the spread of motor impulses is called paresis. Both paralysis and paresis may be central or peripheral.

Central paralyses and pareses arise as a result of lesion of cortical (central) motoneurons or their axons passing in pyramidal tract. Peripheral paralyses and pareses result from loss or weakening of functions of spinal (peripheral) motoneurons as a result of lesion of anterior horns, anterior roots of spinal cord or peripheral motor nerve fibers.

Several forms of central paralysis and paresis are distinguished:

1) monoplegia or monoparesis - affection of one extremity;

2) hemiplegia or hemiparesis - affection of half of the body opposite to the site of affection in the central nervous system (thanks to decussation of most pyramidal fibers on the borderline between the brain and spinal cord);

3) paraplegia or paraparesis - simultaneous affection of either both upper or both lower extremities;

4) tetraplegia or tetraparesis - simultaneous affection of all four extremites;

5) crossed paralysis - affection of upper extremity on one side, and that of lower extremity on other side.

Central paralyses and pareses may be caused by hemorrhages into brain and between meninges, thrombosis and embolism of cerebral vessels, craniocerebral injury, tumours of the nerve tissue, etc.

Lesion of the central motoneurons causes at first complete loss of movements of the corresponding muscles, but after several hours muscular tension as well as resistance of the muscles against the passive movement, are increased. Although voluntary movements are lost, the reflex reactions are retained and even intensified; their reflexogenic zones are extended (for instance, the patellar reflex can be evoked by a tap not only against the patellar tendon, but also against the thigh). Because the inhibitory influences on the reflexes coupling in the spinal cord are weakened as a result of affection of the central apparatus.

Disinhibition manifests itself in intensification of the extension reflex (Babinski’s reflex) or the flextion reflex (Mendel - Bechterew reflex or dorsum pedis reflex - flexion of the II-V toes when the dorsum of the foot is tapped).

Atrophy is negligible or does not develop at all.

Transection of the brain stem affects, in addition to the pyramidal, also the extrapyramidal tracts, and decerebrate rigidity occurs: tetraplegia develops, the paralysed extremites are extended, the tone of the muscles at the neck and back increases which results in opisthotonos - the head is bent backward, the jaws are locked, and the trunk is arched forward.

Extrapyramidal motor disorders are characterized not so much by loss of motor functions as by phenomena of dissociation (disturbances in the realations between the cerebral cortex, basal ganglia, cerebellum and spinal cord) which are marked by loss of synkinetic movements. The automatism inherent in movements diminishes,they become difficult, slow and insufficiently coordinated. The person appears fixed in his posture and moves like an automation.

In extrapyramidial disorders not spastic (as in pyramidal paralyses), but plastic muscle tone increases, and rigidity develops owing to affection of the pallidal and thalamic regions (for instance, in carbon monoxide poisoning). General rigidity is observed. Muscular hypertonia displays excessive static tone with difficult transition to dynamic tone, that is, in attempts to change the position of an extremity the resistance is equal in the agonists and antagonists, the extremity remains in the position artificially imparted to it.

The peripheral paralyses and pareses are observed in injuries of nerve fibers, polyneuritis is frequently accompanied by simultaneous motor disorders and disorders of sensitivity.

Peripheral paralyses are characterized by loss of both voluntary and reflex movements; tendon reflexes disappear. Owing to complete absence of efferent impulses, the muscles lose their tone (hypotonia). In passive movements the muscles offer no resistance. Muscles are soft and flaccid (flabby). Hence the other name of the peripheral paralysis - “flaccid paralysis”.

Deprivation of innervation leads to decreased metabolic rate in muscles and therefore, as distinct from the central paralysis, peripheral paralysis results in dystrophy and atrophy in muscles.

Reaction of degeneration sets in. Stimulation of a nerve with electric current does not cause contraction of the muscle it innervates. Direct stimulation of the muscle by alternating current does not evoke its contraction. The muscle reacts to the influence of the direct current, but the stimulation threshold is elevated. In contrast to the normal response, the anodal closure produces stronger contractions than does cathodal closure.

Experimentally peripheral paralysis can be easily produced by transection of any peripheral nerve which contains motor fibers.

The anterior horns of the spinal cord (where the peripheral motoneurons are localized) may be affected in injuries to the cord. This phenomena are reproduced in experiment by transection of the spinal cord which is followed by spinal shock. The spinal shock is characterized by sharply decreased excitability and depression of reflex activity of the regions located below the site of transection, whereas the function of the regions above the transection is almost unaffected.

In spinal shock all motor reflexes disappear, the blood pressure drops, the urinary and defecation reflexes are absent. Consequences of the spinal shock are more severe in the individuals that are more developed from the evolutionary point of view (monkeys and especially human being). They depend also on the level of the injury (transection). For instance, quick death usually follows spinal cord transections above the level of C4 where the phrenic nerves originate.

The spinal shock results from the complete disconnection between the brain and spinal cord which eliminates the impulses arising in the higher parts of the central nervous system and influencing the excitability of the segmental apparatus of the spinal cord. After elimination of the signs of the spinal shock usually hyperreflexia occurs which is connected with cessation of inhibitory influences of the reticular formation on the spinal cord motoneurons.

Hyperkinesias (hyperkineses) are involuntary, excessive movements of strange form and configuration of skeletal muscles in different parts of the body. They may be spinal, pyramidal, extrapyramidal and may result from stimulation of motor regions of the central nervous system.

Hyperkinesias of spinal origin are characterized by fibrillary contractions, that is, isolated contractions of various muscle fibers (for instance, in spinal muscular atrophy) which are observed in any stimulation of a periheral motor neuron.

Hyperkinesias of pyramidal origin are most commonly manifested in convulsive states. Convulsions are sharp involuntary contractions of muscles with changes in their tone. Clonic and tonic convulsions are distinguished:

1. Clonic convulsions - are intermittent rhythmic involuntary muscular contractions rapidly alternating with relaxations. Clonic convulsions of the muscles of speech cause stammering, those of the group of muscles of the face - are called tic.

Tics (habit spasms) are sudden, irregular, stereotyped, repetitive movements of variable complexity that almost always involve similar groups or fragments of muscles. The abnormalities in mild form are behaviour, but more severe, chronic tics frequently are part of a wider illness called. Tourette’s syndrome (Gilles de la Tourette’s disorder), in which the various motor tics often are accompanied by random bizarre grunting or barking noises as well as shouting of scatologic or obscene expletives. Like chorei form movements, habit spasms are quick, most often involve muscles about eyes or mouth, but sometimes affect the distal extremities. Tics disappear with sleep and are aggravated by emotional tension. But most tics can be reduced by voluntary effort, while chorea cannot.

2. Tonic convulsions - are characterized by periodic protracted involuntary muscular contractions with increase in their tone. They are observed in tetanus.

Alternation of clonic and tonic convulsions is observed in epilepsy, hyperglycemic and hepatic coma, some forms of the craniocerebral injury; clonic convulsions are followed by tonic convulsions in guinea pigs during anaphylactic shock.

Convulsions may be of a reflex character, due to strong pain stimulation (stimulation of the sciatic nerve or posterior roots by scar or neuroma, inflammation of meninges) and may be produced by conditioned reflexes (a convulsive seizure under the influence of a situation in which it had occured previously).

Convulsive seizures grow more frequent under the influence of emotions and various stimuli.

In experiment convulsive seizures can be produced by passing electric current through the animal’s brain or using pharmacological preparations (strychnine) directly influencing the cerebral cortex.

Since extrapyramidal system (striate body, red nucleus, black subtance, etc.) controls coordination of movements and exercises inhibitory influences on the tonic reflexes, disturbances in their activity are most frequent causes of all types of motor disorders.

Exterapyramidal hyperkinesias include tremor, chorea and athetosis:

1. Tremor (trembling) - is weak involuntary contractions of skeletal muscles owing to alternating changes in tone of antagonist muscles. It is observed in chronic alcoholism, mercury poisoning, some organic diseases of the nervous system (Parkinson’s disease, epidemic encephalitis, disseminated sclerosis). During voluntary movements trembling may be increased (in disseminated sclerosis) or, quite the reverse, it may cease (in epidemic encephalitis, Parkinson’s disease).

2. Chorea (Gr. choreia - dance) - is characterized by involuntary, non - rhythmic and irregularly distributed, coarse, quick twitching movements of groups of muscles in the face, tongue, extremities. Patients find it difficult or impossible to maintain a fixed posture or an uninterrupted muscular contreaction (to maintain protrusion of the tongue or fixation of the grip). Muscular effort accentuates the abnormal movements and commonly induces “overflow activity” spreading initialy into the ipsilateral, then the contralateral extremities, the trunk or the face. Emotions also intensify the involuntary movements, whereas during the sleep hyperkinesis is not observed.

3. Athetosis (Gr. athetos - without position) - stands between chorea and dystonia in its pathopysiology and expression. It is characterized by convulsive, but slower movements primarily in the distal parts of the extremities, the movements involing simultaneously the agonists and antagonists. Twisting, writhing, snake-like movements and postures sometimes involve the entire muscular apparatus (trunk, neck, face). The movements can appear at rest, but are greatly intensified by efforts to initiate voluntary activity.

Disturbances in coordination of movements without paralysis are called ataxia (Gr. ataxia - disorder). Static ataxia (lack of muscular coordination in standing still) and dynamic ataxia (lack of muscular coordination in movement) are distinguished. In dynamic ataxia the movements lack smoothness, precision and balance.

Ataxia may be caused by affection of cerebellum, cerebral hemispheres, brain stem, vestibular apparatus, posterior columns of the spinal cord:

1. Cerebral ataxia - results from lesions of the temporal and frontal lobes of the cerebral hemispheres, which are associated with cerebellum. As distinct from cerebellar ataxia, in cerebral ataxia the individual falls to the side, opposite to lesion.

2. Thalamic ataxia –is observed in the thalamic syndrome because of loss of position and kinesthetic signals normally relayed through thalamus to the cerebral cortex.

3.Cerebellar ataxia - is characterized by more marked symptoms. To increase the area of support, when walking, the person (animal) puts the feet too apart, has a rolling (tottering) gait and (unlike the cerebral ataxia) falls to the side of lesion (because on that side the muscular tone is decreased). Animals deprived of cerebellum move with difficulty; they throw out their legs, which are struck against the floor and stagger as the result of impaired tone of the abductors and adductors of the extremities.

4. Vestibular ataxia - the features of disturbances in coordination of movements remind those in cerebellar ataxia. Tottering gait is observed.

5. Cerebrospinal ataxia – is connected with lesion of the posterior columns, arises as a result of loss of proprioceptive sensation on the same side. In this type of ataxia Romberg’s syndrome is observed: if the person closes his eyes in the standing position with feet together he falls backwards.

Disorders of sensitivity arise as a result of impaired transmission of excitation along sensory nerves from the peripheral receptors and along afferent tracts to the cerebral cortex. Lesions may occur on various levels of the central nervous system (grey matter of the spinal cord and medulla oblongata, optic thalamus, ascending parietal gyrus, upper temporal region of the cerebral cortex). The following forms of sensitivity disorders are distinguished:

1. hyperesthesia – increased sensitivity;
2. paresthesia (dysesthesia)-abnormal (perverted) sensitivity;
3. anesthesia-loss of sensitivity;
4. hypesthesia- diminished sensitivity;
5. dissociation of sensitivity.

Intense hyperesthesia frequently results from injury to large nerves (trigeminal nerve, sciatic nerve) or formation of neuroma which stimulates the proximal end of the transacted nerve; sensitivity is greatly intensified and is characterized by causalgia (burning pain).

Hyperesthesia may be provoked in animals by removal of the cerebral cortex. Decorticated animals display a violent defence reaction to weak stimuli. This is connected with liberation of the third sensory neurons localized in the optic thalamus from inhibitory influences of the cerebral cortex.

Paresthesia arises as a result of unusual stimulation (in circulatory disorders, intoxications and inflammatory processes) of peripheral nerves or central sensory structures (the nuclei of the optic thalamus or the assenting parietal gyrus). These ceses are marked by peculiar sensations of crawling, burning or numbness.

Hypesthesia and anesthesia may be of different character: tactile anesthesia, pain anesthesia (analgesia), thermoanesthesia, loss of propriocertive sensitivity(disturbance in the appreciation of the position of organs in the space). Dessection of peripheral nerve causes loss of all types of sensitivity in the corresponding area. Complete transection of the spinal cord causes blockade of all sensations distal to the segments of transection.

Hypestheseia is observed in some diseases (leprosy).

Dissosiative disorders of sensitivity may arise only in cases of injury to the spinal cord and the medulla oblongata since in them the pathways of pain and temperature sensitivity run apart. If only one half of the spinal cord is transected on a single side, the Brown- Sequard syndrome occurs. All motor functions are blocked on the side of the transection, in all segments below the level of the transection, paralyses come into being; whereas only some of the modalities of sensation (proprioceptive and tactile sensitivity) are lost on the transected side, and others (pain and temperature sensitivity) are lost on the opposite side (the conductors of pain and temperature sensitivity cross over as they enter the spinal cord).

Disturbances in the functions of the vegetative nervous system may result from infections, intoxications, injuries affecting different (central as well as peripheral) parts of this system. In experiment they are produced by transection of sympathetic (sympathicotomy) or parasympathetic (vagotomy) nerves, removal of sympathetic ganglia (sympathectomy or desympathization), action of the pharmacological preparations changing activity of the sympathetic (sympathicomimetic and sympathicolytic drugs) or parasympathetic (cholinomimetic and cholinolytic drugs) parts of the vegetative nervous system.

Exclusion of vegetative nerves (in cases of lesion) is soon manifested in increased sensitivity of the denervated structures. For instance, sympathectomy makes the denervated vessels excessively sensitive to adrenalin and transection of the parasympathetic nerves innervating the iris renders it excessively sensitive to parasympathomimetic substances.

In some persons hereditary- constitutional excess of synpathetic (sympathicotonia) or parasympathetic (parasympathicotonia or vagotonia) tone is observed (sympathicotonics and vagotonics). Constitutional vagotonia is one of the forms of the general vegetative neurosis.

Total sympathectomy results in dilatation of arterioles and decrease of arterial pressure, bradycardia, increased body temperature .

After total vagotomy rare and deep respiration (disturbed Hering –Breuer reflex), pulmonary hyperemia and edema (paralysis of the pulmonary vasoconstrictors), sharp decrease in gastric and pancreatic secretion are observed. Since contraction of the muscles closing the larynx is connected with vagus nerve activity, it remains open during swallowing, and aspiration pneumonia develops.

So, bilateral vagotomy causes severe changes in the organism which soon lead to death, whereas partial vagotomy is sometimes used in treatment of the gastric and duodenal ulcer.

Poisoning with parasympatholytic drugs that are used in the clinical medicine (atropine, platyphyllin, scopolamine) results in general insufficiency or loss of parasympathetic innervation. Toxins and antigens of some pathogenic bacteria (diphtheria, botulism) also cause disturbances in the parasympathetic innervation of the organism.

Poisoning with some phosphorized organic compounds and combinations of manganese results in sharp increase of the parasympathetic nervous system excitability (by the way of decreasing cholinesterase activity).

Some inhibitors of cholinesterase (eserine, neostigmine, galantamine) are used as drugs, though their large doses may cause severe poisoning.

Potassium ions , vitamin B1 , choline, some infectious agents (viruses of grippe, bacteria of abdominal typhoid, paratyphoid) and allergens increase tone of the parasympathetic nervous system by the way of increasing synthesis of acetylcholine and its influence on the cells. Cholemia in mechanical and parenchymatous jaundices is accompanied by increased excitability of the parasympathetic nervous system.

Diseases that are accompanied by increased intracranial pressure and compression of the medulla oblongata (cerebral tumors, meningitis, arachnoiditis) cause stimulation of the vagus nerve centers and result in vagotonia of central origin.

Since the vegetative centers are localized in the spinal cord, medulla oblongata and hypothalamus , lesions in these parts of the central nervous system cause disturbances in the vegetative functions. For instance in spinal shock together with motor disturbances, decreased arterial pressure , disorders in thermoregulation and perspiration are observed.

Diseases and lesions of the medulla oblongata cause dysfunction in the respiratory center as well as centers controlling heart activity, vascular tension, lacrimation, salvation, gastric and pancreatic secretion.

Activity of the hypothalamus as a higher center controlling all the vegetative functions, is closely connected with limbic system, basal ganglia, cerebellum, cerebral cortex. Therefore, affection of these parts of the central nervous system also results in disturbances in the vegetative functions.

Stimulation of the posteror hypothalamus results in activation of the sympathetic nervous system (tachycardia, increased respiration rate and blood pressure) and that of anterior hypothalamus – activation of the parasympathetic nervous system (bradycardia, decreased respiration rate and blood pressure).

Stimulation of the frontal lobe causes changes in circulation, respiration, digestive and sexual functions. Owing to the influence of the cerebral cortex on the vegetative functions, it participate closely in the pathogenesis of disease, stenocardia, gastric ulcer, metabolic diseases).

Vegetative neurosis is widespread disorder frequently extending to both parts of the vegetative nervous system and characterized by the state of increased excitability and lability of the vegetative nervous system. Its manifestations are disturbances in frequency and rhythm of heart activity, disorders in vascular tension (vascular dystonia, vascular crisis), increased sweating or xeroderma (dryness of the skin), white or red demography, disorders in digestion (dyspepsia, diarrhea, constipation), etc.

Dysfunction of the nervous system underlies many trophic disorders. Lesions in the nervous system are frequently accompanied by trophic disorders in the skin (changes in hair growth and epidermis regeneration, necrosis, trophic edema, disturbances in fat deposition), formation of trophic ulcera in the skin and mucous membranes, atrohy in skeletal muscles, osteoporosis.

After dissection of certain ramus of the trigeminal nerve, in the cornea of rabbit’s eye keratosis was observed. Injury to the tibial nerve frequently causes chronic ulcer of the foot.

Dissection of the posterior horns causes trophic disorders in the areas innervated by corresponding segments of the spinal cord. This experiment demonstrates that afferent impulses coming from the tissues also play a part in the nervous trophicity.

It is assumed that nerve cells influence the metabolism by the following ways:

1. by the help of mediators (acetylcholine in the parasympathetic nervous system, and norepinephrine – in the sympathetic nervous system); evidently a small amount of acetylcholine secreted in the parasympathetic synapses in resting state serves for regulation of the metabolism;
2. by the help of axoplasmatic current, that is, movement of proteins, electrolytes and enzymes along axons from neurons in distal direction to the working cells. Neuron and structure that is innervated by it – form the regional trophic.

Contour in trophic factors are called trophogens or trophins. Injury of this trophic contour leads to distrophical processes not only in the innervated organ (muscle, skin, other neurons), but also in the innervating neuron itself.

Equally with deficiency of the normalizing trophic factors, in the pathogenesis of affection of the nervous system pathotrophogens, that is, pathogenic trophic factors may play the leading part. They come into being in the pathologically changed tissues and induce pathological states.

Lesions in the spinal cord may cause trophic disorders (decubitus ulcers in paralyses due to diffuse transverse lesions in the spinal cord, chronic perforating ulcers of the feet in connection with tabes dorsalis, diseases of the joints.)

Trophic disorders caused by lesions in the brain are manifested in pathological deposition of fat in the organism vascular disturbances accompanied by tissue changes, affection of joints, decumbitus ulcers (in hemiplegia).

Results of many experiments demonstrate the reflex origin of trophic disorders. For instance, suturing a thread soaked in turpentine to the proximal end of a transected sciatic nerve causes reflex development of an ulcer on the opposite extremity.

Marked trophic disturbances on the skin and in internal organs frequently accompany chronic experimental neurosis.

The following factors play a part in the pathogenesis of neurogenic dystrophy resulted from deprivation of organs of the nervous system’s influence on the tissue trophicity:

1. neurohormones (including those moving by axoplasmatic current) do not come into working organs from damaged nerve;
2. the organ with disturbed innervation cannot inform the nervous system about its state, and therefore, even the intact nerves cannot influence its trophicity;
3. the pathological impulses (pain) coming to the central nervous system from the proximal part of the dissected nerve intensify disfunction of nerve centers and by this way influence negatively the metabolic processes in periphery;
4. dissected sensory nerve begins to conduct impulses in the opposite direction and this causes disturbances in metabolic processes;
5. in the organ with disturbed innervation the genetic apparatus of cells is changed, the substances of antigen nature are synthesized, to which the organism’s immune system responds by rejection of transplant type reaction;
6. the organ that gets out of the nervous system control , responds to the humoral influences(biologically active substances, drugs) by extraordinary reactions which are usually stronger than the power of the agent which causes them (Cannon’s law of denervation);
7. the organ which has lost its sensory innervation, is exposed to the influence of the injurious environmental agents to still greater extent; this accelerates distrophical changes in the denervated tissue.

Disturbances in the higher nervous activity are studied by the way of complete (decortication) or partial removal of the cerebral cortex, injury to the cerebral cortex, influence of different toxins and poisons on the cerebral cortex and reproduction of pathological states of the higher nervous activity in experiments (experimental neuroses), as well as conditioned, biophysical and neurophysiological (electroencephalography) methods.

The character of disorders in the higher nervous activity reproduced in experiment depends on the level of the evolutional development of the animal. For instance, disturbances in the higher nervous activity are more severe in rat than in rabbit, and in dog than in rat. They are the most distinctly manifested in monkeys, though not so severe as in man.

Decorticated animals lose orientation in the external environment and may perish from thirst and hunger, though with good care they may live for an indefinitely long time.

Loss of all conditioned reflexes that are produced throughout the life (both artificial - laboratory, as well as natural- to the appearance and smell of the food) underlies the changes in the behaviour of the animals after decortication. The conditioned reflexes that are lost, do not recover and new ones cannot be elaborated. Only unconditioned reflex activity is retained, but it is also considerably weakened. Therefore, functions of all systems of the organism are sharply changed.

After complete bilateral removal of the cerebral cortex dogs lapse into long sleep which is interrupted only for micturition and defecation. Subsequently sleep is frequently alternated with waking. The dogs seem blind and deaf, though they react to light and sound. They do not respond when called, do not recognize their master, do not go up to food and do not discriminate odours, but voraciously eat the food introduced into the oral cavity. Because owing to the loss of the inhibitory influences of the cortex instincts are somewhat strengthened. Animals become more aggressive, their skin grows more sensitive.

The dogs retain their motoricity, posture and forms of locomotion conditioned by the function of the diencephalon and the striopallidal system. But the agility and smoothness of movements are noticeably impaired. The unconditioned skeletal motor reflexes become crude and imperfect. Rapid fatigability is observed.

Decortication causes disturbances also in the vegetable functions. The salivary and gastric glands cannot properly adjust their secretory activity to the quantity and quality of the food.

Disturbances in the regulation of the cardiovascular and respiratory systems (a slight physical effort causes tachycardia and dyspnea), metabolism, heat regulation, are observed; trophicity of tissues is disturbed, the immune reactions diminish. Young animals fail to grow normally.

In monkeys extirpation of the cerebral cortex causes still greater disturbances which are manifested in a loss of skills, marked motor disorders and loss of minicry and gestures.

Decortication affects also the function of analysers, the ability of fine analysis and synthesis is lost. The retained subcortical unconditioned reflex activity and its highest manifestations (instincts) are incapable of ensuring a normal existence of animals under the constantly changing conditions of the external environment.

Consequences of the partial removal of the cerebral cortex (removal of the cortex of one hemisphere, the anterior and posterior parts of both hemispheres, the central part of one of the analysers- auditory, visual, motor, etc.) depend on the site and extent of injury.

Removal of the cortex of one hemisphere results in the following pathological phenomena:

1. Asymmetry of movements, that is, absence of coordinated movements, on both sides. Owing to the decussation of the pyramidal tracts, motor disorders are observed on the side opposite to that of removed cortex.
2. Higher threshold of stimulation on the affected side, lower cutaneous and pain sensitivity, and higher general excitability due to the impaired normal relations between the cortex and subcortical region.
3. Slight atrophy of the skeletomotor apparatus on the side opposite to the site of affection (due to disturbance in the nervous trophicity). Extirpation of various portions of the cerebral cortex causes a function of analysers, that is, of some particular part of the cortex. The functions may be to a certain extent restored by scattered regions of the analyser which suffice to effect the same reaction, but in an inperfect, elementary form.

The following stages are distinguished in the pathological changes of the higher nervous activity caused by surgical influences (partial removal of the cerebral cortex) and trauma to the brain:

1. diffuse protective inhibition in response to surgical trauma, temporary partial or complete disappearance of conditioned and certain unconditioned reflexes, and sometimes even of instincts. Impossibility to distinguish on this background the functional changes that are specific for the injured part of the brain;
2. weakening of the processes of active internal inhibition and increase of excitation, conditioned and unconditioned reflexes are recovered and at first exceed the initial norm, the local consequences of the extirpation (specific changes) are partially revealed;
3. abatement of the general reaction (effects of the trauma) and distinct manifestations of local disorders connected with injured analyser (disappearance of all conditioned connections and impossibility to elaborate the new ones connected with this analyser), development of process of restoration by peripheral part of analyser (beginning with restoration of functions of regions farthest removed from the site of injury and the last to be restored is the function of the injured part);
4. pathological phenona in cases of scar formation, which compresses the surrounding part of the cerebral cortex and result in weakened cortical activity alternating with convulsive seizures.

There are certain differences in changes of the higher nervous activity caused by various infections and toxic substances. However , the general regularities are also revealed in the development of these changes. As a result of intoxication, first of all the processes of active internal inhibition (as evolutionally younger and therefore, more vulnerable ones) are affected. Usually the cortical excitability is increased; but on the culmination of the intoxication protective inhibition develops which at first results in loss of artificial conditioned reflexes that were elaborated later, and then- that of natural conditioned reflexes. Inhibition extends to subcortex, and the normal interrelations between cortex and subcortex are disturbed.

Intoxication takes a favourable course in individuals with strong, equilibrated type of the nervous system, and its course is severe and protracted in those with neurosis.

Emotional disorders develop as a result of lesion of the hypothalamus, limbic system and neocortex. 3 types of the emotional disoders are distinguished:

1. hyperthymia-strengthening of emotions;
2. hypothymia (depression of spirits)-weakening of emotions;
3. perverted emotions.

Stimulation of not only different structures of the brain, but frequently that of different parts of the same cerebral structure (for instance, different hypothalamic or amygdaloid nuclei) may result in various emotional reactions. Besides, rections caused by stimulation of the same point of the brain may differ depending on the species of the experimental animal.

The emotions of the positive character are mostly resulted from stimulation of lateral hypothalamic nuclei, some parts of the mesencephalic reticular formation, septum, amygdula, hippocampus and some other structures of the limbic system. Stimulation of anterolateral hypothalamic nuclei, anterior parts of the reticular formation and septum, certain amygdaloid nuclei causes the negative emotions (rage, fear).

In Olds' experiments positive reactions (pleasure or enjoyment) causing self- stimulation in rats were observed when the electrodes have been implanted in the posterior hypothalamus or the midbrain reticular formation. Negative emotions in cats (ferocious reaction when it attacks other animals in vicinity) were caused by stimulation of the dorsal part of the diencephalon and the ventromedial nucleus of the hypothalamus.

Removal of the cerebral cortex or some hypothalamic nuclei results in reaction of the “sham rage”. For instance, such a dog loses the ability to evaluate the stimuli objectively, responds to any external stimulus by the reaction of rage, for no reason attacks its owner or the experimenter.

The phenomena of non – motivated (causeless) excitement, anger, rage or euphoria are united under the general name “emotional stress”. Cannon called such reactions “sham emotions”. For example , in persons with pathology of the anterior hypothalamus phenomena of excitation with euphoria are observed, which turn into urritability and anger for no reason.

Fascicles of fibers localized immediately in front of hypothalamus participate in the suppression of the rage reaction, when they are dissected, the violent fit of rage occurs, and the animal destroys everything on its way.

Exactly the opposite emotional behaviour (placidity and tameness) occur when the reward centers are stimulated.

Various changes in the emotional reactions are observed in experiments when different cerebral structures are distructed or stimulated as well as in man in cases of lesions or diseases of those structures.

Bilateral destructioin of the caudate nucleus in monkeys causes stupor, decreased sensitivity to painful stimulation, orientation reflex disappears, ”emotional dullness” develops.

Bilateral destruction of the pale globe in experiment causes hypokinesis, constrained movements, impoverishment of facial expression, sharply expressed plastic tone.

The pallidal patients (with lesion of the pale globe)have mimic immobility of the face (mask- like face),the symptom by which they are recognized at the first glance. Removal of the orbital cortex in cat and monkey causes increased irritability and aggressive behaviour. Lesions of frontal lobes also cause emotional disorders. In persons after operation on these lobes intensity of feelings (joy, sorrow, etc.) is lost , imagination and creative abilities are considerably decreased; the patients become careless.

In cases of tumors of the medial parts of frontal lobe flaccidity and apathy develop, frequently the memory for the current events is lost.

Destruction of the anterior portions of both temporal lobes in monkey results in removal of the amygdalas which lie deep in these part of the temporal lobes. This causes combination of changes in behaviour called Kluver- Bucy syndrome: excessive tendency to examine objects orally (even tries to eat solid objects), loss of fear, decreased aggressiveness, tameness, changes in dietary habits (even herbivorous animal often becomes carnivorous), sometimes psychical blindness and frequently excessive sex drive (attempts to copulate with immature animals, with those of the wrong sex or of different species).

Affected persons respond in a manner not too different from that of monkey. The most widespread form of the stable functional disorders of the higher nervous activity is neurosis. According to Pavlov’s theory disturbances in the strength of the processes of excitation and inhibition, their mobility and mutual balance in the cerebral cortex underlie the neuroses. As distinct from animals, in human beings neurosis may result also from dysbalance between the first and the second signaling systems, Though in psychical diseases also the balance between excitation and inhibition is disturbed, but they cannot be identified with neuroses. Because, unlike the neuroses the psychical diseases are accompanied also by psychical disorders and degradation of the personality.

Experimental neuroses are produced by the way of overstrain of the excitation and inhibition, their strength and mobility in the cerebral cortex. They also result from clash of the nervous processes.

Overstrain of the process of excitation in the cerebral cortex is achieved by the way of protracted intensive stimulation.

Overstrain of the process of inhibition in the cerebral cortex is achieved by the way of excessively difficult, very fine marginal differentiations, protracted influence of conditioned inhibitory reflexes, change in the habitual order in the system of positive and negative conditioned reflexes, etc. For instance, conditioned reflex to light circle flashed on screen was elaborated in a dog. Then the dog was supposed to differentiate the circle from an ellipse (of the same size and lighting having 1:2, 2:3 or even 4:5 semi- axis ratio) which was not reinforced. So, the ellipse became a conditioned inhibitory agent. After that attempt was made to elaborate a finer differentiation by gradually reducing the difference between the ellipse and the circle. When an ellipse closely resembling a circle (with 7:8 or 8:9 semi – axis ratio) was introduced into the experiment, it was no longer possible to obtain a complete differentiation. All the previously elaborated differentiations disappeared, and the dog’s behaviour sharply changed. It was continuously excited and restless, tore off the instruments attached to him, bit through the rubber tubes, refused food, etc. So, the dog suffered nervous breakdown.

Overstrain of the mobility of the excitation and inhibition in the cerebral cortex is achieved by the way of continuous and rapid alternation of positive and negative stimuli.

The following types of experimental neuroses are distinguished:

1. Neurosis with predominance of the process of excitation –is characterized by general motor excitement, persistent, inadequate nervousness (agitation), aggressivity, maliciousness, loss of previously acquired active internal inhibition. Frequently it turns into the neurosis of the inhibitory type (owing to the development of the protective inhibition).

2. Neurosis with predominance of the process of inhibition is characterized by sluggishness, passive defence reactions, protective inhibition, depression, somnolency (sleepiness), total disappearance of the previously eleborated conditioned reflexes and in the severe cases – loss of also unconditioned reflexes.

3. Neurosis with the pathological mobility of the nervous processes:

1. neurosis with the pathological inertness - is characterized by development of phobos (Gr. phobos - fear);
2. neurosis with the pathological lability when “fussiness”, incompleteness of actions, increased motor activity are noted.

4. Circular (cyclic) neurosis – is characterized by alternation of above- mentioned types of neuroses.

In dogs experimental neurosis was produced also by extraordinary and unusual influences, for example, by a strong noise or sudden explosion of powder. In one of the experimental dogs after a flood unusual motor excitement, almost total disappearance of all previously elaborated conditioned bonds and phenomena of inhibition of the cortex were observed. When the normal relations in the cerebral cortex were re-established (two months after the flood) reproductions of a situation resembling the flood (while the dog was in the stand water was allowed to trickle into the room and form a puddle on the floor) provoked a repeated nervous breakdown.

Experimental neuroses are characterized by phasic states which manifest themselves in disturbed relationship between the action of the stimulus and the response reaction.

Since the phasic phenomena may arise not only in pathology, but also (for a few minutes) during transition from waking to sleep,they are called also the hypnotic phases. These phases resemble those observed in parabiosis, but in neurosis one more (ultra- paradoxical) phase is observed:

1. equalizing phase- all conditioned stimuli regardless of their strength, produce an equal effect, because exhausted neurons can response to strong stimuli only during a short time, after which widespread internal inhibition develops;
2. paradoxical phase- weak stimuli produce a strong effect, and strong stimuli – the weakest effect (or no effect at all);
3. ultraparadoxical phase- reaction of the cerebral cortex to the stimuli is perverted, that is, positive stimuli act as negative ones and vice versa; because as a result of excessive weakness, positive stimuli cause overstrain of the excitation and result in widespread inhibition, whereas negative stimuli overstrain inhibitory process which results in predominance of excitation;
4. inhibitory phase- weakening or total disappearance of all conditioned reflex reactions.

Usually it is impossible to observe all the forms of the phasic state in one animal. Besides, between the equalizing and paradoxical phases two transition phases were revealed:

1. narcotic phase- is characteristic of normal transition from waking to sleep and narcotic sleep; cessation of responses to weak, average and at last, to strong stimuli is observed;
2. phase of stimuli of average strength – neurons can respond only to the stimuli of average strength; because strong stimuli cause protective inhibition (the neurons are exhausted), whereas weak stimuli cannot cause response (threshold of stimulation of neurons is increased).

Reaction of animals to experimentally reproduced neurosis vary depending on the type of nervous system. Neurosis is more easily reproduced in the weak, inhibitable type (melancholic) and the strong unbalanced type (choleric) than in the strong, balanced and active type (sanguine) and especially, in the strong, balanced and inert type (phlegmatic). The strong, unbalanced type frequently lapses into neurotic state in cases of overstrain of the inhibitory processes, whereas extraordinarily strong stimuli fail to affect it. The weak, inhibitable type usually reacts with a breakdown under overstrain of both the inhibitory and stimulatory processes.

1. experimental neurosis with predominance of excitation is characterized by general motor excitement, aggressiveness, loss of previously acquired active internal inhibition;
2. experimental neurosis with predominance of inhibition is characterized by sluggishness, sleepiness, protective inhibition, total disappearance of the previously reproduced conditioned reflexes, and in the severe cases - loss of also unconditioned reflexes.

In addition to the type of the nervous system, all the factors that affect efficiency of cortical neurons (age, nutrition, intoxication, infection) also participate in the development of experimental neuroses.

The experimental neurosis is reversible process. The cerebral cortex activity may be restored after some rest, moderation of the experimental conditions, decreased overstrain at the higher nervous activity, employment of drugs strengthening inhibition (bromides) or excitation (caffeine), combination of both, and hypnotics.

Experimental neuroses served as a model for studying pathogenesis of the disorders of higher nervous activity in man, helped to form conception of the mechanism of development of such neuroses as neurasthenia, psychasthenia, hysteria, to indicate the ways of investigation of pathogenesis of disturbances in higher nervous activity in schizophrenia and certain other diseases, to explain the phenomena of hypnosis, delirium and hallucinations.

Neuroses in human beings may be divided into two groups:

1. primary neuroses- result from direct influence of the external environmental agents on the nervous system;
2. secondary neuroses - are caused by somatic diseases (toxicosis, endocrinopathies).

However, this classification is conventional because neurosis may result from the somatic diseases, as well as it may result in the somatic diseases. Frequently neurosis is the initial stage in development of various phychosomatic diseases (hypertensive disease, stenocardia, atherosclerosis, gastric and duodenal ulcer, etc.), or it develops on the background of somatic diseases (especially those of endocrine system).

The etiologic factors of neuroses in men are divided into three groups:

1. Biological factors: hereditary predisposition, sex (neurosis is more frequent in women), age (neurosis develops more frequently in pubertal and climacteric periods), constitutional features (asthenics are more disposed to neuroses), pregnancy, the diseases that decrease organism’s resistance, etc.
2. Social factors: professional activity (information overload, monotony of labour operations), unfavourable family life, unsatisfactory domestic conditions, pecularities of the sexual upbringing, etc.
3. Psychogenic factors: pecularities of personality, psychical traumas in childhood, psychotraumatizing situations (serious illness, loss of relations or friends, official or “academic” troubles, etc.

Thus, the basis of human neuroses is the neurotic conflict, that is, such attitude of the personality to the complicated psychotraumatizing situation which impedes its rational solution.

Tree most spread forms of human neuroses are distinguished:

1. neurasthenia;
2. hysteria;
3. obsessional neurosis.