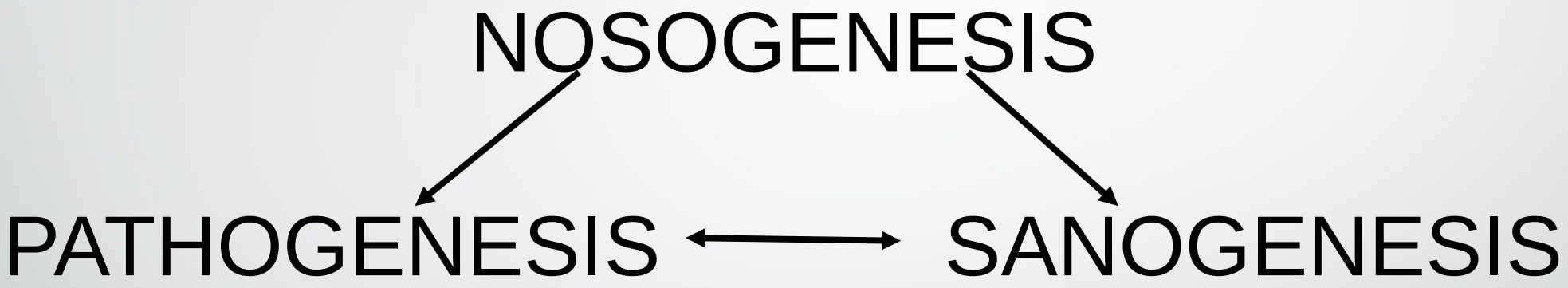
NOSOGENESIS

In the development of the DISEASE, the following elements are DISTINGUISHED:

- Damage
- protective and compensatory reactions
- compensation and decompensation

NOSOGENESIS-the study of the mechanisms of occurrence, development and outcomes of diseases.


NOSOGENESIS
PATHOGENESIS
SANOGENESIS

PATHOGENESIS-the study of the mechanisms of changes in the body that occur as a result of damage.

THE ROLE OF ETIOLOGICAL FACTOR IN DISEASE DEVELOPMENT

- TEMPORARY ROLE (THE FACTOR THAT STARTS THE PROCESS);
- PERMANENT ROLE (ACTS THROUGHOUT THE DISEASE, DETERMINES THE OUTCOME OF THE DISEASE);
- VARIABLE ROLE (THE ETIOLOGICAL FACTOR DETERMINES THE ONSET OF THE DISEASE, THEN ITS VALUE CHANGES).

PATHOGENETIC FACTORS are changes in the body that are the result of damage and determine the further course of the disease. This is the result of the interaction of an etiological factor with a susceptible organism.

There is a hierarchy among pathogenetic factors.

THE MAIN LINK OF PATHOGENESIS IS THE PATHOGENETIC FACTOR THAT IS NECESSARY FOR THE DEVELOPMENT OF ALL SUBSEQUENT LINKS OF PATHOGENESIS AND PRECEDES THEM.

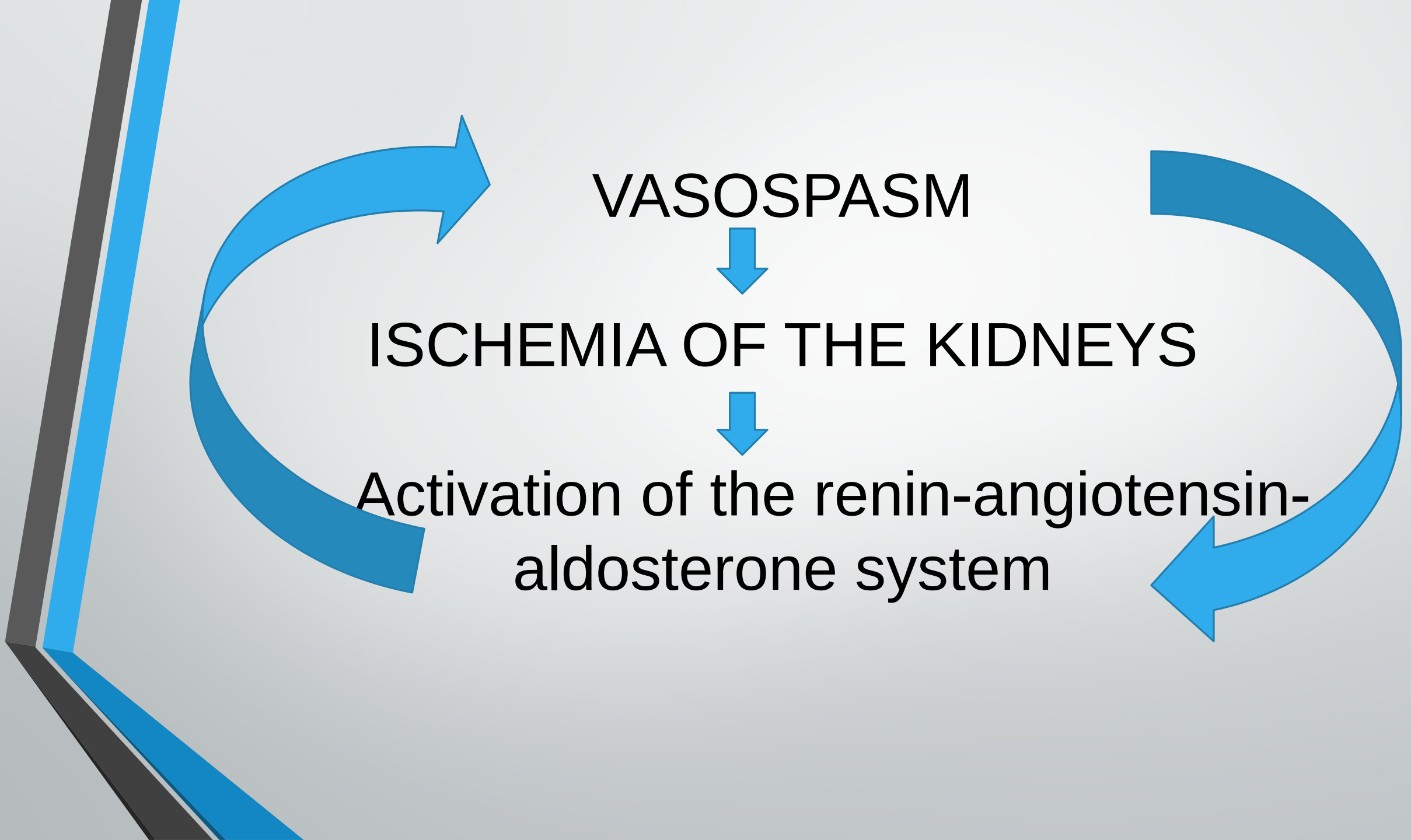
When it is eliminated, the disease stops.

There may be several pathogenetic factors. They don't exist in isolation.There is a relationship between them (cause-and-effect relationships).

Insulin deficiency-hyperglycemia-glucosuria-polyuria-polydipsia

This is a linear relationship.

VICIOUS CIRCLE – circuit the causal relationship between the pathogenetic factors in the development of the disease, when the final pathogenetic factor causes the initial gain factor.


VASOSPASM
ISCHEMIA OF THE KIDNEYS
Activation of the renin-angiotensin-aldosterone system

MAIN MECHANISMS OF PATHOGENESIS

I. NEURO-REFLEX;
II. NEUROHUMORAL;
III. CELLULAR.

I. NEURO-REFLEX MECHANISM-the disease occurs as a result of violation of any element of the reflex arc:

- the receptor,
- guides,
- the nerve center,
- working body.

The role of receptors and conductors can be proved:

- annoyance,
- by turning it off.

The role of nerve centers can be proved:

- by switching off,
- annoyance.

Pathological reflex

- this is an inadequate, inappropriate reaction of the body, limiting its adaptation to the environment.

MECHANISMS OF THE PATHOLOGICAL REFLEX

- violation of trophic function of the nervous system,
- trace reactions,
- pathological dominant,
- parabiosis,
- pathological disinhibition.

Violation of trophic function

Proved By A.D. Speransky.
When cutting the sciatic nerve exceptviolations of motor function belowcutting also causes corneal ulceration.
Mechanism – a violation of the circulation of the damaged nerves trophogenic and the formation of pathological trevorrow.

Trace reactions

Studied By A.D. Speransky.

He injected tetanus toxin into the rabbit's muscle – local convulsions occurred (the first blow to Speransky). Then the animal was subjected to cooling – a generalized convulsive reaction appeared (the second blow to Speransky).

This mechanism explains the occurrence of some relapses of the disease not as a result of repeated action of a specific factor, but under the influence of a non-specific factor.

Pathological dominant

The author of the doctrine

of the dominant is A. A. Ukhtomsky.

A pathological dominant is a focus

in the central nervous system that has

- excessive excitability,
- excessive inertia, stagnation,
- excessive ability to sum pulses,
- excessive ability to inhibit other nerve centers.

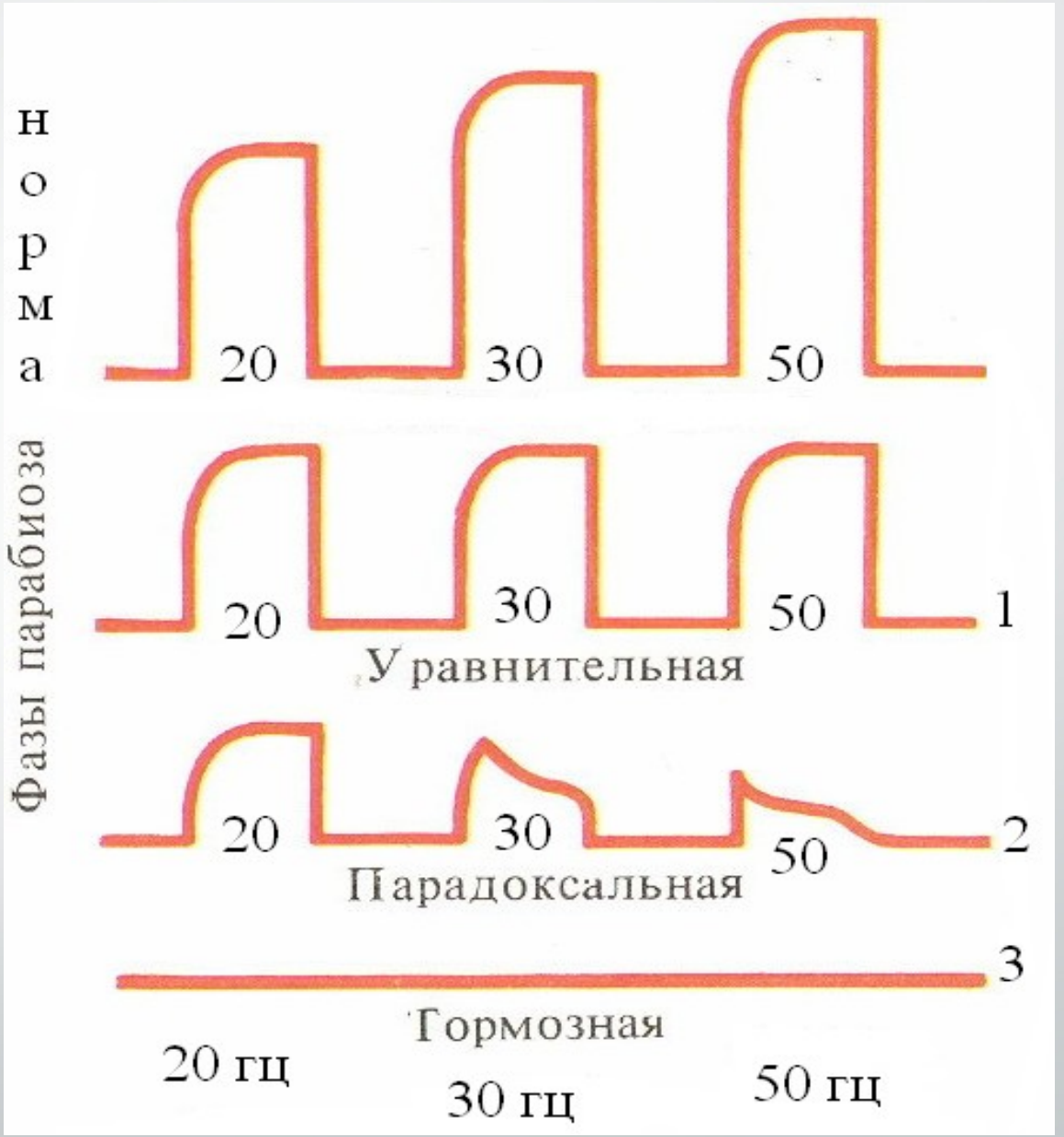
Parabiosis is a persistent non-oscillating excitation that has lost its ability to spread.

Founder of the doctrine-

N.E. Vvedensky.

N.E. Vvedensky conducted experiments on a neuromuscular preparation and established the presence of three phases of nerve fiber parabiosis:

1. Equalization
2. Paradoxical
3. Brake system



In the laboratory of I. p. Pavlov, when studying experimental neuroses on a nerve cell, the phases of parabiosis according to N. E. Vvedensky were confirmed, and three more phases of parabiosis were discovered:

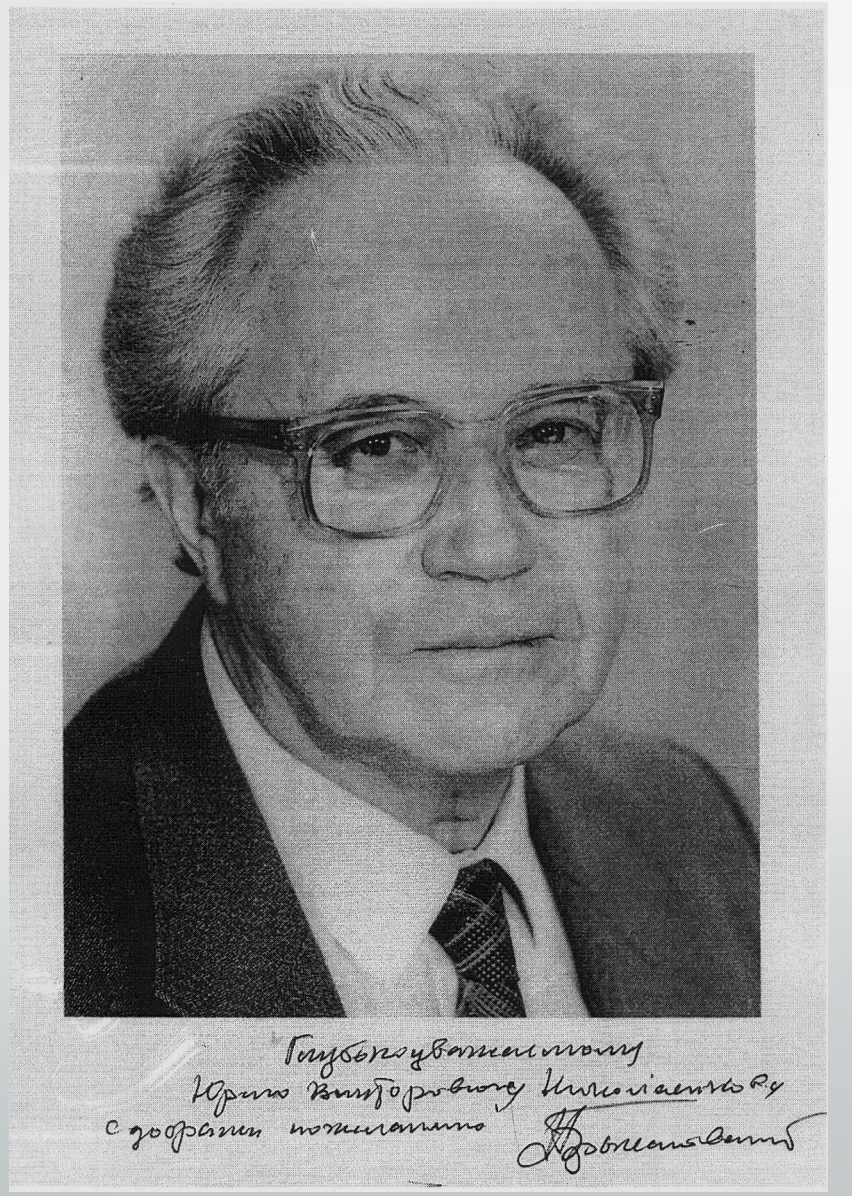
1. Medium-sized stimuli
2. Narcotic
3. Ultra paradoxical

Pathological disinhibition

Studied By G. N. Kryzhanovsky.

Occurs when there is a violation of the interaction between the centers of excitation and inhibition.

For example, when the inhibitory effects of the striatum on the pale ball decrease, the excitability of the latter increases and hyperkinesis occurs.



II. NEURO-HUMORAL MECHANISM-disorders in the course of the disease occur when the amount or activity of BAS (hormones and other humoral substances) changes.

Humoral factors affect the structures of the nervous system, changing their activity and including the neuro-reflex mechanism of pathogenesis.

Proof of the role of the neuro-humoral mechanism of pathogenesis:

- removal of endocrine glands,
- introduction of hormones and other substances into the body.

III. CELLULAR MECHANISM

1. Violation of the energy supply of the cell.
2. Damage to the membrane apparatus and enzyme systems of the cell.
3. An imbalance of ions and fluids in the cell.
4. Violation of the cell's genetic program and (or) mechanisms of its implementation.
5. Disorder of intracellular mechanisms of cell function regulation.

SANOGENESIS is the study of the mechanisms of protective and compensatory reactions that develop in response to damage and are aimed at restoring the disturbed vital activity of the body.

Classification of sanogenetic mechanisms:

- primary-perform a protective and adaptive role in a healthy body, and in a patient – a protective and compensatory role:
- protective (cough, vomiting);
- - compensatory (increased erythropoiesis in inhabitants of the mountains and in chronic heart failure);
- secondary-present only in the diseased body and perform a protective and compensatory role:
- protective (inflammation);
- - compensatory (vicarious compensation).

Compensation-compensation for the structure and function of the body that is disrupted during the course of the disease.

Forms of compensation:

- at the expense of reserves;
- the strengthening or weakening of the function;
- curating;
- hypertrophy;
- reparative regeneration;
- restructuring the structure and function;
- recombination.

Compensation mechanisms:

- neuro-reflex (blood output from the depot in case of blood loss);
- neuro-humoral (increased production of contrinsular hormones in diabetes mellitus);
- cellular (myocardial hypertrophy in heart failure).

The compensation stage:

1. Formations.
2. Fastenings.
3. Depletions.

DYNAMICS OF THE DISEASE

pathogenic stimulus

↓

activation of protective and adaptive reactions

↓

damage to the structure violation of function

↓

protective and compensatory reactions

↓

compensation (latent period)

↓

decompensation (prodromal period

and the period of the height of the disease)

↓ ↓

compensation (recovery) or **decompensation** (death)

QUESTIONS FOR SELF-MONITORING OF KNOWLEDGE

1. What's the difference in terms of nosogenes, pathogenesis and sanogenesis?
2. How is the importance of the nervous system in the development of the disease proved?
3. To define pathogenetic factors. What is their role? Provide examples.
4. What is a vicious circle? Provide examples.
5. What is the main pathogenetic factor? Provide examples.
6. To define the pathological reflex. What is the reason for the formation of a pathological reflex?
7. what are the features of inflammation in denervated tissue and when cutting sympathetic nerve fibers?
8. what are damage mediators?
9. Give examples of trace reactions. Who was the first scientist to study them?
10. how will inflammation occur with hypofunction of the thyroid gland?

11. Name the leading mechanisms of pathogenesis.

12. What properties does the pathological dominant have? Who among the scientists dealt with this problem?

13. Give an example of cause-and-effect relationships in the course of the disease.

14. Describe the vicious circle of blood loss.

15. What methods can be used to prove the importance of nerve centers in the pathogenesis of the disease?

16. what stages are characteristic of nerve fiber and nerve cell parabiosis?

17. What are the features of the course of inflammation in animals that are in a state of hibernation?

18. What methods can be used to register the presence of cellular damage in the course ofdiseases?

19. Define sanogenesis.

20. Classification of sanogenetic mechanisms.

21. Name the compensation phases.

22. What are the main forms of compensation?

INDEPENDENT WORK OF STUDENTS IN THE CLASSROOM

Goal: to make sure that the body is able to compensate for damage caused by a disease-causing factor.

Method: fix the frog with its back up. Make skin incisions on both sides along the middle axillary line. Grab the tissue with tweezers as close to the armpit as possible and open the chest cavity with a small incision. The lung should come out. Do the same on the other side. To observe the magnitude of the excursions of the lungs, the blood filling them. Then tie one lung at the root. Observe the second lung, note the change in the size of the lung, the frequency of respiratory movements. Tie the second lung at the root.

TASKS

1. it has been Observed that nerve damage can cause dystrophic changes in organs and tissues outside the zone of its innervation, sometimes in remote areas. For example, after damage to the sciatic nerve, corneal turbidity and ulceration may occur. What is the mechanism of this phenomenon? Who among the scientists worked on this problem?

2. with stenosis of the left atrioventricular opening, the following clinical manifestations develop: shortness of breath, cough, pain in the heart, cyanosis, edema of the lower extremities. What is the main link in the mechanism of development of these disorders?

3. two mice are placed in flasks of the same volume: one mouse is in a state of anesthesia (experimental), the second – control. The flasks are hermetically sealed and observed for the development of signs of oxygen starvation in mice (agitation, changes in the frequency and rhythm of breathing, impaired coordination of movements (ataxia), convulsions, respiratory arrest). Which mouse will have these disorders earlier and more pronounced? Give an explanation.

4. one of the manifestations of radiation sickness is leukopenia. If soon after the disappearance of the symptoms of the disease, the body is affected by an irritant of a different nature (for example, cold), then leukopenia may occur again, i.e. a relapse of the disease. Explain the mechanism of this phenomenon. Who among the scientists worked on this problem?

5. a Characteristic symptom of pertussis is a frequent paroxysmal cough, which occurs when the mucous membrane of the respiratory tract is irritated by a pertussis toxin. Loud sounds, injections, and conditioned stimuli can provoke a cough. it persists for a long time after recovery. However, during an exciting game, the whooping cough may weaken and even stop. What is the mechanism of this phenomenon?

6. Once during the performance of a Symphony, the conductor felt a sharp attack of pain behind the sternum and left the stage. The use of vasodilators eliminated the pain. During the second performance of the Symphony, the conductor again experienced an attack of chest pain on the same musical phrase. The conductor refused to perform this Symphony, and the attacks stopped. Explain the mechanism of recurrent pain. Who among the scientists worked on this problem?

7. in two mice, overheating is reproduced: the experimental mouse is pre-administered 10 UNITS of insulin, the second mouse is a control mouse. When animals are overheated, they consistently experience: agitation, changes in the frequency and rhythm of breathing, impaired coordination of movements (ataxia), convulsions, and respiratory arrest. Will there be differences in the course of overheating in the experimental and control mice? Explain the results.

8. there are cases When patients with mental disorders refuse normal food and at the same time feel a desire to eat garbage, substandard products that cause disgust in a healthy person. Explain the mechanism of this phenomenon.

9. it is Noted that in patients suffering from hypertension, in response to a heat stimulus, vasoconstriction may occur, and not the expansion characteristic of a healthy person. In patients with angina, in contrast to healthy people, physical activity often does not cause dilation of the coronary vessels, but rather narrowing. Explain the mechanism of these reflex reactions. Who among the scientists worked on this problem?

10. How will the hypofunction of the thyroid gland affect the course of the inflammatory process? Give an explanation.

11. the frog's chest cavity was opened and the lungs were exposed. At autopsy, the lungs had the same size, blood filling (color) and excursion. Then a ligature was applied to the root of the right lung. The right lung was sleeping, and at the same time, the size, blood filling, and excursion of the left lung increased. After some time, the root of the second lung was bandaged: respiratory movements stopped, but there were no signs of asphyxia. Explain the data received. Name the form of compensation.

12. Select the sanogenetic mechanisms that occur in humans when climbing mountains:

a. Tachycardia
b. rapid breathing
c. increased blood pressure
d. increase in the number of red blood cells
e. Excitement
f. Euphoria
g. periodic breathing
h. inhibition