REVIEW ARTICLE

Typical forms of pathological processes of the nervous system

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ABSTRACT

The proposed article is an experience of creating a review on the typical forms of pathology of the nervous system. Much attention is paid to the importance of pathological mechanisms in certain diseases. This is important for understanding the pathogenetic basis of the disease. The article will undoubtedly be of interest to a pathophysiologist, a clinician, and even a pathologist. It will prove to be a useful tool for teaching one of the leading disciplines of medical science pathological physiology.

Keywords: Neuron; Pathological system; Dominant; Pathological mechanisms

INTRODUCTION

Generators pathologically enhanced excitation concept and general characteristics

One hyperactive neuron cannot cause a disorder in the activity of the central nervous system. Such an effect occurs when exposed to a sufficiently powerful stream of impulses that can overcome the mechanisms of regulation and inhibitory control of other parts of the CNS and cause their pathological activity. Such a powerful stream of impulses is produced by a group of hyperactive neurons that form a Generator of Pathologically Enhanced Excitation (GPEE) [1].

GPEE is an aggregate of hyperactive interacting neurons that produces an excessive stream of impulses. The intensity and nature of this flow do not correspond to the incoming signal and are determined only by the features of the structural and functional organization of the GPEE. Due to the fact that the GPEE neurons mutually activate each other, the GPEE is able to work autonomously and self-sustain its activity without the need for constant additional stimulation from the outside [2].

Arising from damage to the nervous system, GPEE becomes an endogenous mechanism for the development of the pathological process. It underlies a variety of nervous disorders related to different areas of the nervous system. Therefore, its formation has the character of an almost universal pathogenetic mechanism. It is a typical pathological process that takes place at the level of inter-neuronal relations [3].

The electrophysiological expression of GPEE activity is the total potentials of its constituent neurons. As an example of such potentials, we can cite the electrical activity recorded in the area of the GPEE in the giant cell nucleus of the medulla oblongata and in the epileptic focus in the cerebral cortex, which is one of the types of GPEE [4-7].

Pathogenetic significance of GPEE

The main pathogenetic significance of GPEE is that it hyper activates the CNS section in which it originated, as a result of which this section acquires the significance of a pathological determinant that forms a pathological system. Since pathological systems underlie the corresponding nervous disorders (neuropathological syndromes), the formation of GPEE is the initial link in these disorders, arising at the level of inter-neuronal relationships. Experimentally, this is proved by the fact that, by creating GPEE in certain parts of the CNS, it is possible to cause the corresponding neuropathological syndromes (for example, various types of pain and convulsive syndromes, Parkinsonism, a number of emotional and behavioral disorders, etc.) [8].

Formation and activity of GPEE. GPEE can be formed under the action of various substances of exogenous or endogenous nature, causing either a violation of the mechanisms of inhibitory control (which entails dis-inhibition and hyper activation of neurons), or direct hyper activation of neurons. In the latter case, the inhibitory mechanisms are preserved, but they are functionally ineffective and unable to normalize the activity of neurons. In all cases, an obligatory condition for the formation and activity of the GPEE is the insufficiency of inhibition of its constituent neurons [9,10].

LITERATURE REVIEW

Pathological determinant

Concept and general characteristics: The formation of GPEE does not always result in the occurrence of pathological reactions. With the blockade of the spread of the excitation generated by it, numerous [11-13].

An example of the formation of GPEE during a primary violation of inhibition can be generators that arise under the action of tetanus toxin, strychnine, penicillin, and other convulsants. An example of the formation of GPEE during primary hyper activation of neurons can be generators that occur during enhanced and prolonged synaptic stimulation, under the action of excitatory amino acids (in particular, glutamate), during shallow ischemia and post-ischemic reperfusion of the CNS, with corresponding changes in receptor, membrane and metabolic processes. GPEE can also occur during differentiations of neurons, after transection of nerves, spinal cord, and other things, which, for example, are associated with different pain syndromes.

In the early stages of GPEE development, when the inhibitory mechanisms are still preserved and the excitability of neurons is low, GPEE is activated by sufficiently strong stimuli coming through a certain input to the group of neurons that make it up. In the later stages, when there is a profound insufficiency of inhibitory mechanisms and the excitability of neurons is significantly increased, the GPEE can be activated by various stimuli from different sources, as well as be activated spontaneously due to the activity of significantly and stably altered neurons, which can be almost constantly active (the so-called trigger neurons).

The nature of the activity of the GPEE formed in different parts of the CNS and under different conditions is not the same. It is determined by the features of the structural and functional organization of the GPU. In this regard, different generators, as well as the same GPEE, at different stages of

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their development, produce pulse flows that are different in nature and duration. This is related to the peculiarities of the pathological reactions caused by the generator and the course of seizures in certain pathological syndromes.

Other mechanisms of inhibitory control of GPEE are functionally isolated and do not cause systemic pathological effects. Pathology occurs if a CNS section hyper activated under the influence of GPEE actively influences other CNS formations, involves them in a pathological reaction and combines them into a new, pathodynamic organization a pathological system. Such a department of the CNS also determines (determines) the nature of the activity of the pathological system, therefore it acquires the significance of a pathological determinant.

The pathological determinant in the nervous system is the altered formation of the central nervous system, which forms the pathological system and determines the nature of its activity. The role of a pathological determinant can be played by any formation of the central nervous system (a department, a nucleus, a set of nuclei, a nerve center, etc.).

Pathogenetic significance of the pathological determinant

The pathological determinant is a formative, key and control link of the pathological system. The appearance of a pathological determinant means the next stage of the pathological process after the formation of the GPU. The determinant itself is an endogenous mechanism for the further development of the pathological process. The occurrence of a determinant belongs to the category of typical pathological processes that occur at the system level. Since the determinant determines the nature of the activity of the parts of the system and their interaction within the system, it is an expression of the principle of intra-system relations.

An example of a pathological determinant in the cerebral cortex is a powerful epileptic focus, under the influence of which a complex of scattered, weaker foci of epileptic activity (EPA) is formed. Such a focus not only forms an epileptic complex, which is a pathological (epileptic) system, but also determines the nature of the activity of other foci and the entire complex as a single system. If the determinant focus is suppressed with the help of pharmacological agents or surgically removed, then the complex disintegrates and separate epileptic foci reappear in its place.

The emergence and activity of the pathological determinant

The determinant can unite the structures of the CNS into a pathological system and determine the nature of the activity of these structures and the system as a whole, provided that its influences are able to overcome the mechanisms of regulation of the structures subordinate to it. This ability is acquired by a hyperactive formation of the central nervous system, producing a sufficiently powerful functional package. In most cases, the hyper activation of this formation is carried out by the GPEE that has arisen in it. The weakening for any reason of the mechanisms of regulation of those structures that perceive the influence of the determinant contributes to the implementation of these influences. So, in the formation of the epileptic system in the form of a complex of epileptic foci, primarily those areas of the cerebral cortex that were changed under the influence of convulsants (zones 2 and 3) are involved; zone 4, not exposed to convulsants, remained uninvolved in the complex.

In the early stages of the development of nervous disorders, the pathological determinant is activated by specific modal stimuli, *i.e.* stimuli that are adequate for the formation of the central nervous system, which has become a determinant (for example, light stimuli, if the determinant is formation in the system of the visual analyzer, pain if the determinant has arisen in the system of pain sensitivity, etc.). This pattern also extends to disorders of higher nervous activity, to neurotic reactions: their determinant is activated under the action of the same stimuli that caused its formation (for example, the same conflict neurotic situations, etc.). These features determine the specifics of provoking influences that cause seizures and manifestations of the corresponding nervous disorders. In the later stages, the determinant can be activated by stimuli of various modalities, and therefore seizures can be provoked by various influences. In addition, the pathological determinant can be activated spontaneously in connection with spontaneous activation of GPEE.

Departments of the central nervous system, experiencing a long-term influence of a pathological determinant, over time can themselves become determinants. Initially, such a secondary determinant is dependent on the primary: It disappears if the primary determinant is eliminated. In the future, the secondary determinant may acquire an independent pathogenetic significance. Usually the next link of the same pathological system becomes a secondary pathological determinant. But it can be an education related to another physiological system; in this case, a new pathological system is formed from this physiological system. Sometimes the secondary determinant turns out to be stronger than the primary one and becomes the leading one. The establishment of primary and secondary determinants is important for understanding the pathogenetic features of nervous disorders, their correct diagnosis and pathogenetic therapy.

The pathological determinant is the most resistant link in the pathological system. With the general suppression of the pathological system with the help of pharmacological agents or with its natural reduction, the determinant structure is preserved even when other formations have already normalized and left the pathological system. However, due to the disappearance of the system, this formation loses its determinant value, retaining some activity due to the activity of the already weakened GPEE present in it. Preservation in this state or even in the form of traces of the former determinant is a risk factor for relapse. With new pathogenic influences that violate the mechanisms of control and enhance the activity of GPEE, the determinant structure is the first to be reactivated, which contributes to the restoration of the pathological system. So there is a relapse of nervous disorders [14,15].

DISCUSSION

Pathological system

Concept and general characteristics: The pathological system is a new pathodynamic organization that arises in the CNS under conditions of damage. The main biological sign of a pathological system is its maladaptive or direct pathogenic significance for the organism. This feature significantly distinguishes the pathological system from the physiological system, the activity of which has an adaptive value and is aimed at achieving the result necessary for the body.

In some cases, the pathological system arises as a result of hyper activation and out of control of the physiological system, in others by involving damaged and undamaged CNS formations in a new, previously non-existing structural and functional organization.

The factor that forms and determines the activity of the pathological system is the pathological determinant. The emergence of a pathological system is the next stage in the endogenization of the pathological process and the mechanism for its further development. Their formation belongs to the category of typical pathological processes that are realized at the level of systemic relations.

A good example of the activity of the pathological system is the pathological scratching reflex. It occurs when the GPEE is created in the brachial section of the spinal apparatus of the scratching reflex. Under these conditions, this apparatus becomes a pathological determinant, which turns the physiological scratching reflex into a pathological one. Animals begin to comb with their hind paws the reflex projection zone on the forelimb. These scratching occur spontaneously. Over time, as the pathological system develops, they become more frequent, prolonged and violent, and may end in tissue tearing. The animal cannot stop these scratchings, despite their inefficiency, uselessness and harmful effect. Violent movements of this kind are observed in many forms of pathology of the human nervous system.

Pathogenetic significance of the pathological system

Pathological systems underlie a variety of nervous disorders related to various areas of activity of the nervous system, so their formation is of almost universal pathogenetic significance.

The activity of the pathological system is expressed in the form of a neuropathological syndrome or symptoms. Each syndrome has its own pathological system. Simple, linear pathological systems manifest as symptoms or monomorphic syndromes. An example of a relatively.

Multilink, branched pathological systems serve as the pathogenetic basis for complex polymorphic syndromes. The latter can also be an expression of a complex of different pathological systems that have a common primary pathological determinant. Examples of such pathological systems include parkinsonism, emotional and behavioral disorders, etc.

Thus, the consistently implemented basic pathogenetic triad "GPEE pathological determinant pathological system" is an endogenous mechanism for the occurrence of various nervous disorders, manifested as the corresponding neuropathological syndromes. This mechanism underlies the experimental reproduction of various neuropathological syndromes.

One of the important pathogenetic mechanisms of the functioning of the pathological system is that it suppresses the activity of physiological systems, including antisystems, and compensatory processes. This mechanism contributes to the development of the pathological process, especially with the continued action of the etiological factor. It ultimately leads to disorganization of the activity of the central nervous system, which is more significant in the later stages of the process.

The emergence, development and activity of the pathological system represent the functional side of the pathological process at the level of intersystem relations. Its basic basis is the processes carried out at the neuronal and inter-neuronal levels. Taking into account the specifics of these processes, in particular their neurochemical nature, in each form of pathology of the nervous system is essential for understanding the pathogenesis of this form and developing appropriate pathogenetic therapy.

Functional organization and features of the activity of the pathological system

The structure of the pathological system. The key system-organizing and control link is the pathological determinant with its mechanism of hyper activation in the form of GPEE. Intermediate and central efferent links develop activities that correspond to the characteristics of the activity of the pathological determinant. If the pathological system has access to the periphery, then its structure also includes a peripheral organ, which becomes a target organ. In this case, the activity of the pathological system manifests itself in the form of an altered function of the organ a pathological effect. If the final link of the pathological system is the structures of the brain, then its effect is expressed in the violation of the corresponding functions of the brain.

From all links of the pathological system there are negative feedbacks to the same links and to the determinant. However, unlike the physiological system, where such connections regulate the activity of the system, they are functionally ineffective in the pathological system, since they do not correct (or correct poorly) the pathological determinant, which, due to the insufficiency of inhibitory mechanisms, has gone out of control. Inhibitory mechanisms are relatively insufficient in other parts of the pathological system; therefore, as a whole, it practically goes beyond the general integrative control of the CNS. Along with this, due to constant activity, positive connections between parts of the pathological system are strengthened, and the conduction of excitation through these connections is facilitated. As a result, over time, the pathological system becomes more and more resistant to regulatory influences from the brain and to therapeutic effects. It works according to a hard-coded principle, realizing the enhanced influence of a pathological determinant.

In the early stages of the process, the pathological system, following the pathological determinant, is activated modally by stimuli specific to it; in the later stages, it can be activated by various, including random, stimuli, as well as spontaneously. Therefore, in the later stages, seizures characteristic of the activity of this pathological system (for example, epileptic seizures, emotional affects, attacks of pain, etc.) can be provoked by various stimuli, occur spontaneously, becoming more and more frequent, prolonged and intense.

In the initial stage, the pathological system is completely dependent on the pathological determinant; it is activated when the determinant is excited and disappears when the determinant is eliminated. In the later stages, due to the strengthening of its structure, the pathological system is less dependent on the primary determinant and can continue to operate even after the removal of the determinant.

Elimination of the pathological system

Unlike a physiological system, which, after reaching a programmed biologically useful (adaptive) result, is eliminated as a functional organization, which provides the possibility of the formation and activity of new functional systems, a pathological system can operate indefinitely. This is due to the preservation of the pathological determinants and the consolidation of positive connections between parts of the pathological system.

The elimination of the pathological system is due to the weakening of the influence of the pathological determinant and the activation of anti-systems. It can occur naturally when endogenous sanogenetic mechanisms are mobilized and under the action of pharmacological therapeutic agents that suppress the activity of the pathological system, disrupt synaptic connections between its parts and activate sanogenetic mechanisms. In all cases, the elimination of the pathological system is carried out according to a single pattern: there is a consistent normalization, starting from those parts of it that are least influenced by the determinant. Therefore, the reduction of the pathological system occurs due to the exit from it of the parts of the system that are most distant from the pathological determinant. The pathological determinant remains the last. With its disappearance, a local, weakened GPEE may remain, which does not cause significant pathological effects. Then the GPEE also disappears. When traces from the former pathological system are activated, the latter can recover. This process begins with the reactivation of the pathological determinant, which contributes to the restoration of the pathological system.

Reduction of the pathological system due to efferent parts with appropriate therapeutic effects leads to the disappearance of clinical symptoms or syndromes, since under these conditions it cannot manifest itself as a violation of the functions of the target organ. However, at the same time, other parts of the pathological system remain and the threat of its restoration and the entire syndrome remains, which occurs with new pathogenic influences. Treatment aimed at normalizing only the efferent links and the target organ of the pathological system is not pathogenetic, but symptomatic.

At the same time, this reduction of the pathological system may turn out to be clinically effective, especially under benign conditions, when the possibility of new pathogenic influences is prevented. In addition, the reduction of the pathological system leads to a decrease, to some extent, in the resistance of the remaining part of it due to a decrease in the number of positive connections that strengthen this system, which contributes to the elimination of the pathological system. It is also very important that the disorganizing influence of the pathological system on other CNS systems is reduced.

An essential way to eliminate the pathological system is to eliminate the pathological determinant. Under these conditions, the pathological system disintegrates. This effect takes place in the early stages of development. At the later stages, due to the possibility of the formation of secondary determinants, as well as in connection with the fixation of the pathological system, the latter can either be restored or continue to exist after the elimination of the primary pathological determinant. Fixation of the pathological system leads to chronicity of the pathological process and the corresponding nervous disorders.

The fight against pathological systems, especially with complex and chronic forms, is very difficult and not always effective. It requires complex pathogenetic therapy aimed at eliminating the pathological determinants and normalizing other parts of the pathological system, activating anti-systems, strengthening general control and other sanogenetic mechanisms, and should be combined with etiological therapy to prevent the action of pathogenic factors that support pathological systems.

Disturbances of dominant relationships

The concept and general characteristics of the dominant: The dominant, according to A.A. Ukhtomsky, is the currently dominant functional structure of the central nervous system the center, the constellation of centers, the physiological system. The dominance of this structure over others is due to the conjugated inhibition of these structures. Dominant relationships are essential for the activity of the nervous system: due to the inhibition of other systems, the currently operating physiological system does not experience interference in the form of influences from other systems, which ensures the achievement of the programmed result without distortion, to the extent necessary.

Violations of dominant relationships can occur in various forms of pathology of the nervous system, they are a typical pathological process that occurs at the level of intersystem relationships.

Types of violations of dominant relations and their pathogenetic significance

Violation of dominant relations is expressed either in the form of their insufficiency, or in the form of their excessive strengthening. In both cases, pathology occurs.

When dominant relations are insufficient, the activity of the currently active system is disturbed by the influences on it from other systems. Under these conditions, the result of the activity of this system does not correspond to the one that should be achieved. With a deep violation of dominant relations, such a result cannot be achieved at all.

With an excessive strengthening of dominant relations, the pathology consists in the fact that the physiological systems and other structures of the central nervous system experience inadequately strong inhibition associated with the activity of the dominant system. A hyperactive pathological system acquires the significance of a pathological dominant: It causes inhibition of physiological systems and, as a result, disorganization of the CNS activity.

Normally, the physiological dominant and determinant are the working principles of the nervous system. The dominant, due to the conjugated inhibition of other systems, provides the possibility of the activity of the currently active system, while the determinant determines the features of the activity of this system. The dominant is the principle and mechanism of intersystem relations, the determinant is the principle and mechanism of intrasystem relations. A structure that plays the role of a determinant in relation to the parts of a given system, that is, an activating part of the system and determines the nature of their activity, at the same time can play the role of a dominant in relation to the structures of another system, suppressing their activity. The combined implementation of the principles of the dominant and the determinant is a necessary condition for the activity of the physiological system and the achievement of the desired result by it. Violation of this combined exercise!' of these principles is a pathology and one of the endogenous mechanisms of development of nervous disorders.

Diseases of nervous regulation

Concept and general characteristics: Disorders of function arise not only as a result of damage to the substrate for its implementation molecular and cellular processes, but also as a result of a violation of the regulation of these processes. If dysregulation plays the main pathogenetic role, the resulting functional disorders have the character of a dysregulatory pathology or regulation diseases. In cases where dysregulatory pathology is associated with a violation of the nervous regulation, diseases of the nervous regulation occur.

In diseases of regulation, and in particular in diseases of nervous regulation, the initial link in the development of the pathological process is changes in the apparatus of regulation of the activity of the organ, which becomes the target of pathological influences from the altered apparatus of regulation. In some cases, primary damage to the target organ may be the initial pathogenetic link in regulatory diseases, but the resulting changes in regulation become so significant that dysregulation becomes of leading importance and is the main pathogenetic mechanism.

Pathologically altered regulation of the activity of the organ is an endogenous pathogenic factor that causes the development of secondary or further changes in the target organ.

Violations of the nervous regulation may be due to changes in both the central and peripheral links of the regulatory apparatus. In both cases, these links become pathological determinants, causing the emergence of pathological systems, the peripheral efferent part of which is the target organ. The clinical expression of the activities of these PS are the corresponding syndromes. In the event that internal organs serve as target organs, neurovisceral pathology occurs. If autonomic centers become a pathological determinant, the resulting syndromes are centrogenous autonomic pathology.

Diseases of nervous regulation constitute an extensive class of diverse disorders. These include neurogenic forms of cardiac arrhythmias and hypertension, vegetative diencephalic paroxysms, neurogenic dyskinesias of internal abdominal organs (stomach, intestines, gallbladder, fallopian tubes, uterus, etc.), some forms of gastric and duodenal ulcers, bronchial asthma, diabetes, glaucoma, various kinds of vegetative crises, etc.

Those forms of pathology that are often referred to in everyday life as "neuroses of the internal organs" (for example, "cardiac neurosis", "gastric neurosis", etc.) are diseases of nervous regulation. They refer to neurotic disorders with certain pathological systems, the target organs of which are the corresponding visceral organs. Why this or that organ becomes an effector depends on the pathogenetic structure of the pathological system, on the direction in which its development will go, which nerve formations will become its efferent links. The involvement of one or another formation in the structure of the pathological system depends on whether the mechanisms of regulation of this formation will be overcome by the influence of the determinant. The own mechanisms of regulation of the target organ are also important. Thus, under normal conditions, with experimental cardiac arrhythmias caused by the creation of GPEE in certain parts of the central nervous system, disturbances in the rhythm of the activity of the heart begin to appear only with a sufficiently long action of GPEE. If you first cause changes in the reactivity of the heart or its damage, even minor ones that do not manifest themselves, then under these conditions, with the formation of GPEE in the same parts of the CNS, arrhythmias occur quickly, and their nature may depend on the characteristics of damage to the heart.

CONCLUSION

Diseases of regulation, including diseases of nervous regulation, the doctor usually refers to a functional pathology, saying that there are no organic changes. This conclusion does not reflect the essence of the process. In regulatory diseases, the corresponding structural changes occur primarily in the regulatory apparatus. In the target organ, pronounced structural changes can manifest themselves a second time, at later stages of the process. But even in the early stages in an organ, for example, in the heart, there may be hidden changes that are revealed under pathogenic influences from the altered apparatus.

Thus, the data presented in the review on typical forms of pathological processes of the nervous system represent a fundamental basis for further study of this system, deepening and detailing the pathogenesis of diseases, allowing you to create a basis for clinical research.

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