



CONTRIBUTION AUTONOMIC NERVOUS SYSTEM THE DEVELOPMENT OF TREMOR AND THERAPEUTIC POSSIBILITIES

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Article History

Received: 09.08.2025

Accepted: 15.10.2025

Published: 01.11.2025

Abstract: - Tremor is the most common hyperkinesia in clinical neurology. Accumulated information indicates that tremor often occurs when the integrative activity of the central nervous system is disrupted with the participation of structures related to nonspecific brain systems. The authors tried to analyze their own and published clinical and experimental data on the effects on tremorogenesis of the autonomic nervous system, as well as evaluate possible pathogenetic ways of correction.

Keywords: tremor, autonomic nervous system, diagnosis, therapy.



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Introduction

Tremor (T) is the most frequent movement disorder observed in clinical practice and one of the most common hyperkinesia, which manifests itself in the form of non-targeted, involuntary, stereotyped oscillatory movements of body parts as a result of contractions of the corresponding muscles [1,2,3,4].

The international group for the study of T characterizes it as a rhythmic involuntary oscillatory movement of at least one functional area of the body [2]. It is distinguished from other hyperkinesia by its regularity and repeatability with a relatively constant frequency [4,5].

Violation of the integrative activity of the central nervous system in vegetative dysfunctions affects various systems, including motor, sensory, adaptive, exploratory and other types of behavior. The interactions of the cortex, subcortical structures, hypothalamic nuclei, limbic-reticular complex (LRC), segmental-peripheral formations of the nervous system that arise in this case imply a single physiological mechanism of further regulation of the activity of functional systems [6,7].

In the motor sphere, adaptive response involving different levels of the autonomic system (ANS) is most often manifested by changes in the provision of spatial orientation, coordination, and

tremorogenesis [1,6,7]. Focusing attention on these functional systems in clinical neurovegetology is justified by informativeness, the possibility of objectification, and the studied pathomorphological features of movement disorders.

The purpose of the work is to analyze own and literary clinical and experimental data on the effects on tremorogenesis of the central nervous system and possible pathogenetic ways of correcting trembling hyperkinesia.

T can occur in isolation or in combination with other clinical manifestations accompanying neurological, endocrine, somatic diseases, various intoxications, etc. regardless of the "pacemaker", the levels of interest of various departments of the nervous, autonomic and other systems that can participate in the implementation of T.

There is information on the selection of four tremorogenic pacemakers (cortical, striopallidal, truncal and cerebellar) [8].

There are indications of the pathophysiological mechanisms of T generation: mechanical, reflex, central nervous system, as well as in cerebellar dysfunction, and the first two refer to peripheral, and the rest to central tremor mechanisms [9]. All of them are provided and supported by the activation of various departments and levels of the central nervous system, which intervene in the work of the neuromuscular system [10], stimulate

Cite this article:

Kalashnikov, V. Y., Stoyanov, O. M., Oprya, Y. V., Melnyk, Y. V., Kugel, Y. I., (2025). CONTRIBUTION AUTONOMIC NERVOUS SYSTEM THE DEVELOPMENT OF TREMOR AND THERAPEUTIC POSSIBILITIES. *ISAR Journal of Medical and Pharmaceutical Sciences*, 3(11), 1-6.

some reflex arcs. Participate in the formation of the "central oscillator" T in the form of neural circuits or, possibly, the spontaneous activity of a group of neurons in the CNS with synchronization by means of interneuronal connections. When forming kinetic variants of T, the dependence of purposeful movements on feedback from the periphery with the participation of the cerebellum increases [8,11].

An even more diverse clinical implementation of T in functional somatoform, psychosomatic disorders involving the stem, diencephalic, subcortical, and temporal-limbic levels of the brain [12,13]. In addition to this, significant indirect effects are found on the part of the central nervous system [14, 15].

Shaking hyperkinesia is often a sign of impaired autonomic regulation or is associated with certain structures of the central nervous system [16,17]. T can be registered with breakdowns of noradrenergic, dopaminergic, serotonergic, cholinergic mechanisms that regulate the activity of the central nervous system. Thus, the imbalance of brain neurotransmitters is the biochemical basis of tremor [18,19,20], the same mechanisms are basic in various dysfunctions of the central nervous system. The most, and to the fullest extent, these violations are found in physiological T (FT) with labile characteristics when autonomic regulation changes. It is known that it suddenly increases during emotional excitement, anxiety, fatigue, hypothermia, alcohol consumption, abstinence, which are necessarily combined with vegetative-vascular changes. All this determines the objectivity of information about the balance of the autonomic supply of T, in cases of dysfunction of the central nervous system, both sympathetic and vagal activation of its departments with corresponding changes in tremorogenesis is observed [17].

It is known that the cells of the striatum are similar to neurons of the vegetative type [21,22], when irritated, the body changes blood pressure, thermoregulation processes, etc.

Unilateral thalamo-striatal-hyperthermic syndrome is described.

An important structural and functional union of striopallidary and vegetative systems is the body of Luys. Its connections with the pale globus (tractus pallidohypothalamicus), striatum, substantia nigra (tractus hypothalamicus descendens), segmental vegetative apparatus, etc. have been studied. [14,23].

Structural commonality of the striopallidary and limbic systems - development from the anterior brain bubble, the presence of single histological formations; the functional inclusion of the reticular formation (RF) of the upper brain stem in the LRK and the vegetative accompaniment of the motor act may explain the frequency of the birth of tremulous hyperkinesia with dysfunction of non-specific systems [7]. At the same time, a number of known neuropsychological disorders can be explained by the direct involvement of the basal ganglia in cognitive disorders and affective processes [24].

It has been shown that the activation of the RF, especially its part near the tractus rubrospinalis, contributes to its development and strengthening [25]. A similar condition is formed when the hypothalamus nuclei are irritated, which affect the activity of gamma neurons of the anterior horns of the spinal cord [18,26]. In addition, tremorogenesis is affected by suprasegmental control, nuclei of the thalamus opticus, hyperexcessive

thalamocortical synchronizing influences, as well as cortical effects on peripheral autonomic structures (including through the striatum).

Indicated for the role of the cerebellum in the regulation of autonomic, sensory functions, emotions [14,27], as well as in the implementation of T [13,28], especially in the case of disruption of the cerebellofrontal projections, often with the occurrence of cognitive-affective cerebellar syndrome [26, 29] (with damage to mainly middle structures - the so-called "limbic cerebellum") with pronounced vegetative accompaniment.

It is also necessary to take into account the participation of the sympathetic nervous system in the vegetative support of the striated muscles [22].

In our opinion, it is necessary to pay more attention not to the morphological substrate T, but to violations of metabolic processes, primarily to neurotransmitter imbalance in the form of a relative excess of catecholamines, histamine, a lack of serotonin, glycine and other neurotransmitters [22,29,30,31,32]. In some works, the occurrence of T is associated with an increase in adrenergic and possibly GABAergic neurotransmission [33].

Increased sensitivity to acetylcholine of damaged brain structures responsible for tremorogenesis was noted [34,35].

Our experimental studies, devoted to the influence of monoaminergic neurotransmitters on reserpine-induced T in rats, confirm their active participation in tremorogenesis and autonomic dysfunctions. Suppression of the activity of serotonin, dopamine, and adrenergic neurotransmission leads to preservation or strengthening of the generalization and amplitude of T. When activating the neurotransmitter systems listed above, the expression of T significantly decreased. With serotonergic stimulation - by 33.6% ($p < 0.05$). Noradrenergic stimulation revealed a more pronounced decrease in T ($p < 0.05$) compared to previous indicators. The maximum reduction in tremor intensity and amplitude was achieved in animals with activation of dopaminergic neurotransmission. The obtained data on monoaminergic neurotransmission and its role in tremorogenesis have pathogenetic connections with the functioning of the central nervous system — piloerection, salivation, muscle dystonia, anorexia, etc. (as one of the main mediators). In addition to nigrostriatal deficiency, it is necessary to take into account the possible mediator imbalance of other monoamines. At the same time, the development of new schemes for the complex treatment of tremulous hyperkinesias, taking into account neurotransmitter disorders, which can be corrected through central suprasegmental structures [35,36], is gaining considerable relevance.

Therefore, the imbalance of brain neurotransmitters is the biochemical basis of tremor [36,37] and the basis for various dysfunctions of the central nervous system. It is fully manifested in FT with labile characteristics of changes in autonomic regulation. The fact of sudden development of T in conditions of emotional excitement, anxiety, fatigue, hypothermia when drinking alcohol, abstinence with pronounced vegetative-vascular accompaniment and vice versa is known [38-43].

Postencephalic T is often accompanied by thalamic pains and autonomic symptoms [38,39]. With essential T, anxiety may be associated with hyperactivation of the noradrenergic system [35,36].

In angioneurology, at the initial manifestations of chronic brain ischemia, T is the main or only symptom of motor disorders [44,45].

Diagnostics. We conducted a hardware study of tremulous hyperkinesis in 76 patients with cerebral angiodystonia and chronic brain ischemia with autonomic dysfunctions. The influence of vegetative changes on the characteristics of T was noted. An increase in the integrative tremographic index gives reason to think about the growth of maladaptive tendencies of motor components as a result of the transition from functional disorders of the central nervous system to organic ones [43,46].

The occurrence and some characteristics of T in acute hypoxic, toxic, hypertensive encephalopathies, dyscirculation in the vertebrobasilar pool, vertebral artery syndrome are described [12,19,22,42,43,47].

In psychovegetative movement disorders, T is accompanied by pronounced emotional and affective manifestations. At the same time, it is necessary to adequately evaluate both the characteristics of hyperkinesis and the syndromic environment to exclude organic suffering [48,49]. In fact, such manifestations are often classified as unclassified tremor syndromes.

In neurophysiology, tremorography is widely used for diagnosing neurovegetative disorders, studying the mobility of nervous processes that characterize the functional state of the motor analyzer in the process of maladaptation.

Small-amplitude T is the most frequent sign of autonomic regulation disorders [50]. The so-called emotional T with pronounced lability of the central nervous system has been described [22,51]. With vegetative crises, fear, anxiety, combined with cardialgias, chill-like T is registered. Fatigue, hypothermia, emotional excitement increase FT. It is known that tremor can occur as a side effect when taking antidepressants, especially tricyclics [12,52], sympathomimetics (phenamine), xanthines (caffeine, theophylline, euphylline), metoclopramide, bronchodilators, synthetic thyroid hormones, cytostatics, immunosuppressants, antidepressants, estrogen compounds [29,52] and others. Moreover, the intensity of T sharply increases with the combined use of catecholamines (including amphetamine) [52], neuroleptics, valproates, dopaminergic agents, levodopa, psychostimulants, glucocorticoids, hypoglycemic drugs, butyrophenones, phenothiazines (especially in combination with tricyclic antidepressants), lithium drugs and others [29]. The development of T in metabolic disorders has been shown - in conditions of hypoglycemia, thyroid dysfunction, withdrawal of alcohol, drugs, benzodiazepines, intoxication with salts of heavy metals (mercury, arsenic, lead, bismuth), carbon monoxide [52-54]. A number of chemicals and pharmacological drugs cause pathological variants of trembling hyperkinesis: rest T can be provoked by neuroleptics and other drugs that block dopamine receptors, manganese [12], chronic carbon disulfide poisoning; T action occurs with alcoholism, alcohol withdrawal, thyrotoxicosis, valproate overdose [29,30,55-57].

Developed ways of correction

The use of complex electropharmacological correction of autonomic dysfunctions, including direct effects on the suprasegmental apparatus of the autonomic dysfunctions (Patent of

Ukraine No. 88797) recorded the alignment and stabilization of the average values of tremometric characteristics. Their reduction correlated with the sympatholytic effect of the proposed therapy - sympathicotonia ($P<0.05$); sufficient vegetative support and satisfactory adaptation ($P<0.05$); the elimination of crises during the paroxysmal course of vegetative dysfunctions was observed.

Also, T correction was performed in 40 people who suffered a craniocerebral injury by direct influence on suprasegmental structures with a vegetative stabilizing purpose using the method of endonasal administration of ethyl methyl hydroperidine succinate 2.5% solution. When using the proposed treatment complex in the main group, the changes were maximal ($P<0.05$). At the same time, this kind of positive changes occurred in the majority of people with initial sympathicotonia (90.0%), eutonia (50.0%) and a minimal effect in vagotonia (12.0%) ($p<0.05$). This circumstance once again confirms the sympatholytic effect and direct influence on tremorogenesis mainly caused by vegetative-vascular disorders, which are characteristic in the remote period of TBI [58].

It is known that one of the early syndromes in vascular pathology is movement disorders in the form of T. 101 patients with chronic brain ischemia and vegetative dystonia syndrome were examined. It has been proven that the severity of this kind of motor disorders depends on the state of the central nervous system, especially against the background of sympathotonic influence. A combination of antioxidants and nootropic drugs was used (hamalate B6, endonasal electrophoresis with a 2.5% solution of ethyl methylhydroxypyridine succinate, and then its oral use, which made it possible to reduce or eliminate subjective experiences, normalize sympatho-parasympathetic connections, comorbid psycho-emotional, cognitive disorders, to achieve the adequacy of the functioning of cerebral hemodynamics and to improve its full perfusion, as well as to inhibit the realization of hyperkinetic manifestations in both studied groups [59]. It is advisable to additionally apply low-intensity transcerebral magnetic stimulation of the prefrontal and motor cortex of the brain of the dominant hemisphere in this type of T in cerebral angiodystonias [60].

The data of clinical observations of adolescents with autonomic disorders and associated tremulous hyperkinesis are given. Aminophenylbutyric acid (phenibut) was used for their correction. Under the influence of therapy, the general clinical condition of the patients, cerebral hemodynamics, psychometric tests, "vegetative portrait" significantly improved, hyperkinetic syndrome stopped, including increased FT. According to the authors, the inclusion of aminophenylbutyric acid is pathogenetically justified, because provided a vegetotropic, sympatholytic, adaptogenic effect, as well as an antiparoxysmal effect similar to tranquilizers for vegetative crises. According to the authors, phenibut is a stimulant of dopamine production, enhances dopaminergic neurotransmission, which is facilitated by the presence of β -phenylethylalanine in the molecule of the drug, a modulator of the mediator function [61]. In addition, improvement of the "feedback" mechanism in cortical-subcortical neuronal projections and increased activity of pallidum-striatal, limbic-reticular and brain stem structures is accompanied by the elimination of psycho-emotional manifestations of their crisis course, which affect the occurrence and maintenance of tremor [5].

Taking into account that T is one of the leading signs of vegetative dysfunctions, a therapeutic complex developed by us including nicergoline, endonasal electrophoresis of ethyl methylhydroperidine succinate followed by its oral administration, sertraline, a complex of balanced vitamins of group B, β -adrenoblockers (propranolol) was used for its correction and sedative drug of plant origin. It is shown that, in addition to the objectification of autonomic dysfunctions with the help of T registration, the latter is associated with a violation of vascular regulation, blood circulation, as well as functional maladaptation of the body in similar conditions. Under the influence of the developed original T treatment complex, an improvement in metabolism, severity of biochemical and neurotransmitter processes, as well as blood circulation in the responsible structures of the central nervous system was noted. Mutual potential vegetative stabilizing and anti-stress effects accompanied by the elimination of manifestations of psychovegetative dysfunctions affecting the occurrence and maintenance of tremor [62,63].

A similar therapeutic complex was used in the treatment of patients with a tremor-rigid form of post-traumatic parkinsonian syndrome, we used an original medication method in combination with physiotherapeutic procedures. The obtained results indicate the pathogenetic effectiveness of the therapy [64].

Discussion

Based on the above T is often a sign of impaired autonomic regulation or is associated with certain structures of the central nervous system. Therefore, the commonality of multilevel provision of vegetative regulation of T, involvement of the same structural and functional formations of the CNS, and even the occurrence of T as a sign of impaired vegetative regulation dictates the need for further study of tremulous hyperkinesia in neurovegetology.

The obtained information makes it possible to objectively assess the balance of the vegetative supply of T and reveal the degree of involvement of the formations of the nervous system in the pathological process in various diseases of the motor sphere, which is necessary for the diagnosis of a number of diseases, prognosis and adequate pathogenetically justified correction.

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