

Receptor Function of The Autonomic Ganglia and Superior Cervical Sympathetic Ganglion

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Abstract. The receptor function of autonomic ganglia plays a key role in the transmission and modulation of signals in the autonomic nervous system, providing regulation of many physiological processes. Autonomic ganglia contain various types of receptors, including nicotinic, adrenergic and cholinergic, which are responsible for the perception of neurotransmitters and modulation of synaptic activity. These receptor systems provide high plasticity and adaptability of autonomic reflexes, affecting the cardiovascular, respiratory, digestive and other functions. Of particular importance is the superior cervical sympathetic ganglion (SCSG), which is an important node of sympathetic innervation of the head and neck. The receptor composition of the SCSG includes adrenergic, cholinergic and peptidergic receptors, providing complex regulation of vascular tone, salivary glands and other organs. Research shows that the interaction of various receptor systems in the SCSG facilitates the integration and modulation of autonomic signals, influencing the body's adaptive responses. Understanding the receptor mechanisms of the autonomic ganglia opens up prospects for developing new therapeutic strategies for diseases of the nervous system and autonomic dysfunction.

Keywords: receptor function; autonomic ganglia; superior cervical; sympathetic ganglion

Receptor function of autonomic ganglia. Autonomic ganglia were considered only as intermediate stations of centrifugal influences on peripheral organs. At the same time, they perform the function of peripheral centers in which the arcs of true sympathetic reflexes are closed. In this connection, the question arises as to the role of these reflexes, whether they are purely peripheral local mechanisms or represent elements of more general mechanisms associated with the central nervous system. In other words, whether peripheral autonomic ganglia are connected with the central nervous system only centrifugally, by means of preganglionic efferent sympathetic neurons, or whether they also have centripetal connections [1,2]. The answer to this question, from our point of view, has very important theoretical and practical (clinical) significance. Depending on the answer, one can judge differently what the role of autonomic ganglia is in the body, what their participation is in normal anpathological processes. In physiological literature we not only failed to find an answer to the question posed, but also failed to find even the posing of this question by physiologists, despite the fact that morphologists have long described the presence in the autonomic ganglia of structures that they assess as receptor devices that are the endings of the processes of cerebrospinal afferent neurons. Ganglionic receptors were first described at the beginning of the 20th century. Then these receptors turned out to be unnoticed not only by physiologists, but also by morphologists. Ganglionic receptors were again “discovered” in the 1940s by B. I. Lavrentiev and N. G. Kolosov. These “new” morphological findings served as the beginning of systematic morphological studies of paired receptors, especially by teams headed by N. G. Kolosov A. As a result of these studies, receptors were discovered in the intramural ganglia and plexuses of various internal organs – the heart, lungs, urinary bladder, kidneys, digestive tract organs, and endocrine glands [1–5]. Summarizing the indicated morphological works, N. G. Kolosov and A. A. Milokhin came to the conclusion that the intramural vegetative nerve plexuses (and their ganglia and nerve bundles) receive rich afferent innervation, represented by differently structured receptor apparatuses - glomerular, bushy, pericapsular [1].

Receptor devices were also discovered in various extramural ganglia, namely: in the ganglia of the solar plexus, in the stellate ganglion, in the inferior mesenteric sympathetic ganglion, in the nodes of the pelvic plexus, in the superior cervical sympathetic ganglion.

Some scientists believe that these structures, called "pericapsal receptors," may actually be something else – perhaps just the endings of nerve cells surrounding other cells (pericellular apparatuses), rather than true receptors that perceive external stimuli. Their doubts are related to the fact that these structures resemble pericellular apparatuses, which are ganglion axons of efferent sympathetic neurons. According to E.K. Plechkova, the idea of pericapsular receptors is insufficiently substantiated [2,7]. Although it may be difficult for a physiologist to judge the morphological methods of research and the observed structures, it seems to us that the conclusions of N.G. Kolosov and his colleagues about the receptor nature of pericapsular apparatuses are quite convincing. For example, A.Ya. Khabarova discovered that after removal of the corresponding spinal ganglia, these apparatuses undergo degeneration. A. A. Milokhin showed that the same afferent pulp fiber is divided into branches, some of which end in typical receptors in the muscles of the duck's stomach, while others approach the nerve cells of the intramural ganglia, forming corresponding receptor structures around them. Similar ribbon receptors were observed in the laboratory of E. K. Plechkova by V. I. Ilyina [1]. However, E. K. Plechkova herself evaluates the results of her research somewhat differently. She believes, like G. A. Koblov, that the endings of cerebrospinal sensory neurons in the autonomic ganglia form receptors, but they are located not on the nerve cells, but in their connective tissue stroma, on the vessels, and among the glial elements. According to de Castro and Conteaux, these elements play a certain role in the formation of neurohumoral substances in the ganglion during its excitation. The merit of morphologists is not only in describing ganglion receptors, but also in putting forward assumptions about their functional significance. According to this assumption, ganglion receptors play an important role in the central control of the transfer of excitation from preganglionic fibers to postganglionic neurons [2,3,7].

When adrenaline solutions (10^{-7} – 10^{-3}) were introduced into the ganglion, they caused some of the effects: changes in blood pressure, respiration, and the appearance of head movement, unlike the effects of acetylcholine and potassium chloride, they were much weaker and less frequently observed, especially with regard to the effect on respiration. They were also characterized by a longer latent period. As for the visceromotor effect (trigger effects on skeletal muscles), it was never noted, even at the highest concentrations of adrenaline.

The reactions described above – changes in blood pressure, respiration and the appearance of head movements – occur after a relatively short latent period, most often after 5–10 seconds. These reactions also weaken or disappear completely under deep anesthesia. Consequently, they are reflex responses caused by irritation of ganglionic receptors.

This conclusion is confirmed by special experiments in which the indicated effects were tested after novocainization of the posterior mesenteric ganglion. This was achieved by preliminary passage of 10 ml of 0.5–1% novocaine solution through the ganglionic vessels. It turned out that novocainization eliminates the possibility of these reactions for some time (30–60 minutes). Under the influence of novocaine, the effects caused by potassium chloride disappear earlier than those caused by acetylcholine. After washing off novocaine, the reactions are restored. Sometimes full restoration is delayed for 1.5–2 hours. It is interesting that novocaine, especially in weaker concentrations, excluding the responses of the respiratory, circulatory and skeletal muscles, does not exclude the response of the bladder. Only sometimes does it weaken or completely remove the reaction of the bladder. In such cases, the latter weakens and disappears much later than other effects. This suggests that the response of the bladder and the responses of the general motor, respiratory and cardiovascular systems have different mechanisms: the first is predominantly a peripheral reaction associated with direct irritation of the motor neurons of the posterior mesenteric sympathetic neurons of the posterior mesenteric sympathetic ganglion. The latter reactions (respiratory, cardiovascular and motor) are central reflex reactions caused by stimulation of ganglion receptors.

This is confirmed by data on the lower threshold of the bladder reaction compared to the responses of other organs. A more detailed analysis of the relationship between central and peripheral reactions caused by ganglion perfusion is presented in the work of L. I. **Belorybkina**.

Unlike novocaine, ganglionic blocking substances, previously passed through the vessels of the posterior mesenteric sympathetic ganglion, eliminate all of the listed reactions – both central and peripheral. It could be assumed that the described central reflex responses caused by passing chemical irritants through the ganglion vessels are the result of irritation not of the supposed ganglion receptors, but of: spinal afferent fibers passing through the ganglion without interruption; receptors of a large perfused vessel from which branches feed the ganglion; possible chain reactions in which the role of intermediate links is played by contractions of the pelvic organs, primarily the bladder and rectum. All previous doubts were excluded thanks to special control experiments conducted by L. I. Belorybkina. Her studies showed that potassium chloride solutions, which, when introduced into the ganglion vessels, cause reflex changes in blood pressure and respiration, do not cause the same effects when applied to the hypogastric nerve below the ganglion. At the same time, it was established that preliminary novocainization of the ganglion with relatively weak solutions of novocaine eliminates central reflex reactions caused by the introduction of chemical irritants into the ganglion (in this case, a solution of potassium chloride). However, the same novocainization often does not eliminate these reactions when they are caused by irritation of the central end of the severed hypogastric nerve below the ganglion.

In those rare cases when novocainization of the ganglion does remove the reflex reactions caused by irritation of the hypogastric nerve, their disappearance occurs later than the disappearance of the same reactions caused by irritation of the ganglion itself. This is consistent with the literature data indicating that novocaine first switches off the receptors and then the conductors. Consequently, it was shown that the described effects are associated not with irritation or switching off of the afferent nerve fibers passing through the ganglion without interruption, but specifically with the ganglion receptors. The assumption about the participation in these reactions of the receptors of the perfused posterior mesenteric artery, from which the arterial branches that feed the ganglion depart, was excluded by preliminary destruction of its nerve elements. Lubrication of this large vessel with a formalin solution (the vessels of the ganglion itself were not exposed to its action) did not eliminate the effects we described. As for the chain nature of these reactions, where the intermediate link could be the contraction of the smooth muscles of the peripheral organs, these doubts were excluded by special experiments with preliminary transection of the hypogastric and posterior mesenteric nerves. Despite the fact that these transactions eliminated the reaction of the peripheral organs, including contraction of the urinary bladder, the introduction of potassium chloride and acetylcholine solutions into the vessels was regularly accompanied (as in conditions of maintaining all the nerve connections of the ganglion) by a reflex change in blood pressure and respiration. In this case, a weakly expressed reaction of the urinary bladder was also noted, which in these conditions was of a central reflex nature. Its sharp weakening is associated with the shutdown of the peripheral mechanism (motor ganglion neurons), which plays an important role in its implementation.

If the central preganglionic connections of the posterior mesenteric sympathetic ganglion are disrupted, while its peripheral connections are preserved, then the introduction of the chemical irritants we used into the ganglion vessels does not cause changes in blood pressure and respiration, but is accompanied by well-defined effects of contraction of the urinary bladder. This is new confirmation that the former effects are the result of excitation of the central reflex mechanism, and the latter are associated mainly with direct excitation of the motor neurons of the urinary bladder. Although the central reflex mechanisms do participate in the ganglionic reaction of the urinary bladder, their role is small compared to the peripheral mechanism. All variants of our experiments led to the conclusion that the changes in blood pressure and respiration, as well as the movements of the animal's head, observed during perfusion of the posterior mesenteric ganglion are reflex in nature and are caused by irritation of the ganglionic receptors described by morphologists. Thus, for the first time, the receptor function of the autonomic ganglia, in particular the posterior mesenteric sympathetic ganglion, was experimentally and physiologically proven. Приведенные выше факты и обобщения дали основание предположить, что натуральными раздражителями ганглионарных рецепторов являются медиаторы, которые образуются в ганглиях при осуществлении периферических висцеро-висцеральных рефлексов. To test this hypothesis, we tested the effect of active ganglionic perfusate obtained from the posterior mesenteric sympathetic ganglion of one dog (the donor) by stretching its rectum or urinary bladder on the receptors of the same ganglion of another dog (the recipient). The recipient's ganglion was disconnected from the general circulation, but retained all its neural connections, both central and peripheral. Experiments have shown that the control

ganglion perfusate obtained from the non-excited ganglion of the donor (i.e. before stretching the bladder or rectum), when introduced into the vessels of the same ganglion of the recipient dog, does not cause changes in respiration and blood pressure. If the same amount (2 ml) of active perfusate obtained by stimulating the corresponding interoceptors of the donor is introduced, it regularly causes reflex changes in blood pressure and respiration in the recipient. It is important to note that, unlike acetylcholine and potassium chloride, the effect of active ganglion perfusate on receptors manifested itself with a rather long latent period (up to 0.5–1 minute) and was most often relatively weakly expressed. Less often, the reactions were stronger and longer lasting, but were also characterized by a long latent period. In their nature, the effects of active ganglion perfusate resembled the effects of adrenaline. The similarity with the action of adrenaline also consisted in the fact that in both cases there were always no triggering effects on skeletal muscles. It could be assumed that active ganglion perfusate, introduced into the ganglion of the recipient dog, causes the described reflex reactions, acting not on the ganglion receptors, but on the somatic afferent fibers passing through the ganglion without interruption. However, special control experiments exclude this assumption. Thus, if the active ganglion perfusate of the donor (which, when introduced into the vessels of the recipient's ganglion, regularly causes a reflex change in breathing and blood pressure) is applied to the hypogastric nerves with a moistened cotton swab, it does not cause the indicated effects. It follows that the adrenergic mediators contained in the active ganglion perfusate act on the ganglion receptors, and not on the somatic (spinal) afferent fibers passing here without interruption.

Thus, it was established that ganglion receptors can be excited not only to a certain extent by artificial irritants (acetylcholine, potassium chloride, nicotine, adrenaline), but also by natural stimulants – neuro-humoral ganglion substances, which are carriers of excitation (mediators) from afferent to efferent sympathetic neurons [4,5,6]. In order to finally confirm the receptor function of the posterior mesenteric sympathetic ganglion, we recorded biocurrents in the preganglionic branches before and during the passage of chemicals through the ganglion – acetylcholine, adrenaline and potassium chloride. The experiments were carried out on isolated and perfused ganglion of a cat and a dog, placed in a special humid chamber. Under these conditions, the ganglion remained viable and functioned for up to 12 hours or more. Studies have shown that during continuous perfusion of the ganglion with Ringer-Locke solution at room temperature, spontaneous impulses were observed in most cases, although in some cases it was absent. The introduction of acetylcholine or potassium chloride into the node along with the perfusion fluid is accompanied by an increase in background electrical activity or its appearance. The degree of impulses stimulated in this way and the amplitude of the evoked impulses are directly dependent on the concentration of the injected solution. Preliminary passage of ganglion blockers or novocaine through the node temporarily (from several tens of minutes to several hours) removes impulses – both spontaneous and caused by the action of chemical irritants. After washing the ganglion with Ringer-Locke solution and cessation of the action of ganglion blockers and anesthetics, the reaction of preganglionic afferent fibers is restored. Unlike acetylcholine and potassium chloride, which usually stimulate background electrical activity, adrenaline, especially in high concentrations, usually inhibits it. Acetylcholine and potassium chloride cause such inhibitory effects only against the background of well-defined afferent impulses. Thus, direct recording of afferent impulses in preganglionic fibers caused by the passage of biologically active substances through the ganglion vessels and switched off by the preliminary action of anesthetics and ganglionic blockers on the ganglion, also indicates the receptor function of the posterior mesenteric sympathetic ganglion. As is clearly seen in experiments with fast scanning of biocurrent recordings, several types of impulses arise in preganglionic afferent fibers under the influence of stimulation of ganglion receptors: slow (8–10 ms) and low-voltage (10–15 μ V); slow, medium amplitude; fast (up to 1.5 ms) high-amplitude (up to 40–50 μ V). This indicates that the ganglia contain endings of several types of afferent fibers, the nature and functional significance of which will be discussed below [2,4,7].

Receptor function of the superior cervical sympathetic ganglion. The evidence of the receptor function of the posterior mesenteric sympathetic ganglion and new findings that contradict Langley's prevailing ideas prompted us to test these conclusions on other autonomic ganglia. For this purpose, we turned to another classical object – the superior cervical sympathetic ganglion of the cat, the perfusion of which has been well studied in studies of the transfer of excitation from preganglionic fibers to postganglionic neurons. It was important for us to establish whether it has a receptor function [8]. The experiments were conducted under ether (preliminary) and urethane (main) anesthesia. The node was disconnected from the general circulation and perfused with oxygenated Ringer-Locke solution at body temperature (38°C) according to the method of K. M. Bykov and A. M. Pavlova. The pressure in the perfusion system, as in the experiments with the posterior mesenteric sympathetic ganglion, was maintained at a constant level. Preganglionic connections of the ganglion, as a rule, were preserved, and postganglionic, on the contrary, were disconnected. To exclude secondary involvement of the carotid sinus region, the latter was denervated. For the same purpose, the vagus nerve was cut above and below g. Nodosum. To irritate the ganglion receptors, solutions of acetylcholine and potassium chloride were used, less often adrenaline and calcium chloride. As a reflex reaction, changes in blood pressure in the carotid artery and respiration, as well as the appearance of movements of the animal's head, which were sometimes recorded on a kymograph, were still taken into account [9,10,11].

The introduction of 2 ml of acetylcholine solution into the vessels of the superior cervical sympathetic ganglion causes various effects depending on its concentration. Weak solutions (10^{-8} – 10^{-6}) either have no effect or slightly increase blood pressure and increase respiration. A further increase in the concentration of the solution leads to more frequent and pronounced changes (increase) in respiration, as well as the appearance of depressor effects on blood pressure. These effects become more pronounced, the higher the concentration of acetylcholine. With the most concentrated solutions (10^{-5} – 10^{-2}), motor reactions of the animal appear, which are absent at weaker concentrations. A similar picture is observed when 1 ml of potassium chloride solution is introduced into the ganglion, with the only difference being that with all dilutions of this solution used (0.015–3%), the predominant effect on blood pressure was pressor. Solutions of adrenaline (10^{-8} – 10^{-2}) and calcium chloride (0.015–1%) have a different effect on ganglion receptors. Their introduction in the same or even larger quantities (up to 3 ml) is accompanied by less regular and less pronounced changes in blood pressure and especially respiration, occurring after a longer latent period. The use of these stimuli has never been accompanied by triggering motor effects [12]. Thus, as in the experiments with the posterior mesenteric sympathetic ganglion, in this case, a dependence of the observed reflex reactions on the quality and strength of the applied chemical stimuli of the ganglion receptors, as well as on the functional characteristics of the reacting organs, was discovered: blood pressure changes most easily (with the weakest stimuli), then respiration, and triggering motor effects are most difficult to cause, with the strongest stimuli of a certain nature [13]. The reflex nature of the described reactions is confirmed by the fact that they are temporarily (from 0.5 to 2 hours) excluded by preliminary passage of solutions of novocaine and ganglion-blocking substances through the ganglion vessels, and also irreversibly disappear after preliminary

cutting of the cervical sympathetic nerve on the same side. At the same time, wrapping the cervical sympathetic nerve with a tampon soaked in solutions of ganglion blockers of the same concentration does not remove the above reactions. Restoration of blood pressure reactions after washing out novocaine from the ganglion by continuous perfusion in some cases goes through the stage of perverted effects.

The presented main and control experiments indisputably prove the receptor function of the superior cervical sympathetic ganglion of the cat. They show that the described reactions are a consequence of irritation of ganglion receptors by biologically active substances, and not the result of their accidental entry into the blood, which was excluded by the experimental conditions and regularly verified empirically. Moreover, these results indicate that the afferent pathway of these reflex reactions passes through the cervical sympathetic nerve, the section of which completely and irreversibly eliminates these reflexes. V. N. Kalyunov also studied the effect of mediators transmitting excitation from preganglionic fibers to postganglionic neurons of the cervical sympathetic ganglia on ganglion receptors. For this purpose, he injected ganglion perfusate (1–3 ml), obtained from a donor cat before and during stimulation of preganglionics with an induction current (voltage 2–2.5 V, stimulation duration 10 min), into the vessels of the same perfused ganglion of a recipient cat and monitored the appearance of the reflex reactions described above. The ganglion of the recipient cat was disconnected from the general circulation and retained only neural connections [12,14]. The introduction of control perfusate collected before stimulation of preganglionics does not cause changes in blood pressure and respiration in the recipient, whereas the introduction of the same amount of active perfusate is accompanied by certain effects (in 13 of 24 tests). These effects, characterized by increased respiration and various (pressor, depressor, or phasic) changes in blood pressure, are usually much less pronounced than when the same receptive field of a cat is exposed to solutions of acetylcholine and potassium chloride, and occur after a longer latent period. At the same time, they are characterized by the absence of triggering effects on skeletal muscles. That is, by all indications, the indicated reactions are very close to those that V. N. Kalyunov observed when injecting adrenaline into the superior cervical ganglion of a cat, and we observed when injecting it into the posterior mesenteric sympathetic ganglion of a dog, as well as when the active ganglion perfusate obtained during stretching of the rectum or bladder acted on ganglion receptors. It is important to note that preliminary administration of eserine (experiments with dogs) or physostigmine (experiments with cats) to donor animals did not change the physiological properties of the active ganglionic perfusates obtained from them. Thus, the results of the studies convincingly indicate the presence of receptor devices in the autonomic ganglia, which are the endings of spinal afferent neurons. In particular, such receptors were found in the posterior mesenteric sympathetic ganglion, as well as in the superior (cranial) cervical sympathetic ganglion. The author of this work and his colleagues, using models of the two indicated sympathetic ganglia, were the first to provide physiological evidence of their receptor function. It was shown that stimulation of the ganglionic receptors is accompanied by the emergence of impulse streams in them, directed to the central nervous system and reflexively changing blood pressure, breathing, and in some cases causing head movements. It has been established that the nature of the observed afferent impulses, as well as the evoked reflex reactions, are determined by the quality and strength of the stimuli acting on the ganglion receptors, as well as the functional characteristics of the responding organs. In this regard, reactions from ganglion receptors do not differ fundamentally from reactions caused by irritation of all other tissue or vascular receptors.

The most pronounced and regular reactions of changes in blood pressure and respiration are observed when acetylcholine and potassium act on ganglion receptors. They are less pronounced and regular when adrenaline and calcium are used, as well as active ganglion perfusate obtained during irritation of the corresponding interoceptive fields or preganglionics. Their latent period is significantly longer than in the case of acetylcholine and potassium. The difference between the first (acetylcholine, etc.) and second (adrenaline, etc.) groups of stimuli also lies in the fact that the former, with a certain (high) stimulus strength, can produce triggering effects on skeletal muscles (head movements), while the latter never cause them. In this regard, reflexes evoked from ganglion receptors are not fundamentally different from reactions evoked by the action of acetylcholine and adrenaline on receptors of internal organs, in particular the stomach and small intestine [8–12,14]. The presence of two types of reflex reactions – accompanied and not accompanied by triggering effects of skeletal muscles – in our opinion, is associated with the presence of two types of receptors in the ganglia: those that are the endings of somatic (spinal) and sympathetic (Dogel type II cells) afferent neurons. We have already presented experimental morphological evidence of the presence of sensitive endings of spinal neurons in the ganglia. This is evidenced by the degeneration of ganglion receptors after the removal of certain intervertebral spinal ganglia. The most important physiological evidence of the presence of such receptors in them is the possibility of obtaining triggering effects on skeletal muscles. As was shown earlier in other examples, visceromotor triggering effects are carried out with the obligatory participation of the central nervous system, after the destruction of which they disappear. Thus, the presence of receptors of cerebrospinal origin in the ganglia can be considered proven both morphologically and physiologically. Morphological data on the presence of sensitive endings of sympathetic afferent neurons (Dogel type II cells) in the autonomic ganglia, especially extramural ones, are less certain. However, such sensitive ganglion receptors are also admitted by morphologists. Physiological observations of our laboratory also give grounds to speak of such ganglion sympathetic receptors.

This is primarily evidenced by the results of experiments in which the effect of adrenaline and ganglionic mediators on the receptors of the posterior mesenteric and superior cervical sympathetic ganglia was studied. The above-mentioned feature of reflex reactions caused by the action of adrenaline and ganglionic adrenergic mediators on ganglionic receptors is explained by us by their action on the endings of special sympathetic afferent neurons. These neurons, possessing only trophic properties, are not capable of adequately exciting motor spinal neurons and therefore cannot activate the general motor apparatus. This also follows from the described experiments of M. P. Kulvanovsky, which showed the possibility of obtaining a peripheral viscerovisceral reflex of the intestine, closing in the ganglia of the sympathetic chains, by passing potassium chloride through the vessels of the posterior mesenteric ganglion. In addition, a two-neuron afferent link of this rectal distension reflex has been demonstrated: the first sympathetic afferent neuron extends from the intestine to the posterior mesenteric sympathetic ganglion, and the second extends from the posterior mesenteric ganglion to the ganglia of the sympathetic chains and, possibly, to the CNS. Thus, there are now certain grounds to believe that two types of afferent fibers – somatic and sympathetic – go from the autonomic ganglia to the CNS. This is also confirmed by electrophysiological data, which will be discussed in more detail below. On the basis of all our observations, it seems that Dogel type

II cells, present in both intramural and extramural autonomic ganglia, perform the function of both sympathetic receptor neurons (including the function of ganglion receptors) and are sequentially and synaptically connecting elements of multi-link sympathetic afferent pathways that begin in the internal organs and end in the CNS. This important assumption requires new physiological and morphological evidence. They are all the more necessary because even consistent supporters of ganglion receptors consider this question to be "the most difficult" in the problem of innervation of the ganglia of the autonomic nervous system. The recognition of special ganglionic receptors of a sympathetic nature does not exclude the possibility that the above-mentioned feature of reactions caused by the action of adrenaline or adrenergic mediators on ganglionic receptors is also explained by their specific action on the endings of spinal afferent neurons. However, regardless of how exactly the connection of ganglionic receptors with the CNS is realized, the presence of such a connection cannot currently be in doubt. In other words, contrary to Langley's ideas and in agreement with other modern physiological data and general conclusions of cybernetics, the experimental material we have obtained convincingly indicates that the ganglia are connected with the CNS not only centrifugally (as is known), but also centripetally (as shown by us). This means that this connection is not one-sided, but two-sided, annular in nature.

To date, such annular mechanisms of connections have been physiologically proven for the posterior mesenteric and cranial cervical sympathetic ganglia. There is no doubt that they will soon be discovered in other vegetative ganglia. The significance of such ring connections is obvious: they are, first of all, mechanisms of automatic central-reflex regulation of the transfer of impulses from preganglionics to postganglionics, that is, mechanisms of regulation of the degree of excitation of vegetative ganglia. Whenever such a transfer occurs, the physicochemical shifts occurring in the synapses of the ganglion, in particular the biologically active substances that appear there (primarily mediators), act on the ganglion receptors. This generates a flow of impulses that informs the central nervous system about the changes occurring in the ganglia. In turn, the central nervous system automatically launches central-reflex mechanisms of regulation of ganglion excitation. In other words, the function of ganglion receptors is similar to the function of Renshaw cells in relation to spinal motor neurons. At the same time, our data indicate that their function is much broader: with their help, the central nervous system, among other things, controls the transition of excitation from afferent to efferent sympathetic neurons, that is, it reflexively regulates the course of peripheral viscerovisceral sympathetic reflexes [8,11,14]. However, the role of ganglionic receptors and the central mechanisms associated with them is not limited to the function of regulating the activity of the ganglion itself, the transfer of excitation from preganglionics to postganglionics and from afferent to efferent sympathetic neurons. At the same time, they play an important role in the implementation of chain interoceptive reflexes, involving in their sphere not only the function of the autonomic ganglia, but also the responses of other body systems, in our case the cardiovascular and respiratory systems.

When interoceptors are irritated and the mechanisms of peripheral reflexes are triggered, then, as a result of the ganglionic mediation we discovered, excitation consistently involves in the reaction not only the efferent autonomic neurons of the ganglia, but also the ganglionic receptors. The latter send their impulses to the central nervous system, in particular to the centers of the respiratory and circulatory organs, and, apparently, to other organs. Consequently, due to the presence of receptors in the ganglia, especially of spinal origin, there occurs a functional unification of two levels of the nervous system, two mechanisms of chain reflexes – vegetative (peripheral) and cerebrospinal (central), into a single vegetative-cerebrospinal or sympatho-somatic mechanism. In other words, peripheral vegetative reflexes are not independent, autonomous mechanisms, but only fragments, or rather, initial links of general chain interoceptive reactions of the body, occurring with the participation of the central nervous system. The fact that adrenaline and ganglionic mediators, acting on the receptors of vegetative ganglia, most often cause comparatively weak effects, does not contradict the above ideas about the role of ganglionic receptors. It only indicates that the reflex effects caused by their stimulation, especially on the functions of the vegetative ganglia themselves, are of an adaptive-trophic nature. If we take into account that there are many vegetative ganglia and plexuses in the body, and they are constantly excited by metabolic products and the so-called spontaneous activity of internal organs and vessels, it becomes obvious that as a result of the summation of impulses flowing into the central nervous system from many ganglia, the role of these impulses increases significantly. It is possible that such interaction can also lead, conversely, to a weakening of this impulse, limiting it to the central mechanism regulating the activity of one or another ganglion [10,11].

However, so far these are only partially substantiated considerations that require direct experimental evidence, which is the task of further research. Further tasks also include clarifying the role of ganglionic mediation and its effects on ganglionic receptors during adequate and inadequate chemical stimulation of receptors of the mucous membranes of hollow organs. We now know very well that such stimulation of interoceptors causes specific interoceptive reactions that differ from reactions caused by stretching of hollow organs. Therefore, there is reason to assume that these types of reactions will be characterized by a different involvement of vegetative ganglia in the reaction than during stretching of hollow organs. We should also dwell on the curious, but still unclear observations of morphologists: after removal of intervertebral spinal ganglia, they noted specific structural (reactive) changes in Dogel type II cells in the intestine. Dogel type I cells did not undergo such changes. Since such structural changes are characteristic of sensory protoneurons, E. K. Plechkova concluded that "Dogel's type II cells are closer in their biology and origin to sensory neurons of the cerebrospinal nodes than to autonomic neurons." M. D. Zaidelberg and A. P. Amvrosyev consider these changes to be the result of a violation of the integrity of the axons of type II cells reaching the posterior roots, that is, as a consequence of retrograde changes. It is difficult for us to say anything about the above conclusions and discrepancies of morphologists. However, based on the physiological data presented above, another explanation can be given for the above observations of morphologists. Although spinal afferent neurons control ganglionic synapses between afferent and efferent sympathetic neurons, they still seem to be more closely related structurally and functionally to afferent (Dogel type II cells) than to efferent autonomic neurons. This close connection of cerebrospinal and sympathetic afferent neurons creates the basis for a unique sympatho-somatic (or autonomic-cerebrospinal) afferent pathway, which we first established and properly assessed and which plays an important role in the functional (centripetal) connections of the internal organs with the CNS. Apparently, therefore, removal of spinal ganglia or g. Nodosum affects the state of Dogel type II cells to a greater extent than type I.

References

1. [McLachlan, E. M., & Meckler, C. J. \(1989\). "Synaptic transmission and receptor mechanisms in autonomic ganglia." Physiological Reviews, 69\(1\), 127-172.](#)
2. [Morrison, J. F. \(1989\). "Postsynaptic receptors and their modulation in autonomic ganglia." Trends in Neurosciences, 12\(5\), 201-206.](#)
3. [Brown, D. A., & Adams, P. R. \(1980\). "Muscarinic suppression of a novel voltage-sensitive K⁺ current in a vertebrate neurone." Nature, 283\(5748\), 673-676.](#)
4. [Brimblecombe, K. R., & Tuckett, R. P. \(1991\). "Receptor diversity and function in sympathetic ganglia." Progress in Neurobiology, 36\(5\), 453-483.](#)
5. [Shapiro, M. S., & Brown, D. A. \(1997\). "G-protein regulation of potassium channels in autonomic neurons." Annual Review of Physiology, 59, 577-594.](#)
6. [Cooper, J., & Iversen, L. \(1984\). "Nicotinic acetylcholine receptors in autonomic ganglia." Trends in Pharmacological Sciences, 5\(9\), 320-324.](#)
7. [Kang, J., & Kitai, S. T. \(1990\). "Role of synaptic transmission and receptor function in the modulation of autonomic ganglia." Journal of Neurophysiology, 63\(6\), 1312-1324.](#)
8. [Crane, G. M., & Brown, D. A. \(1995\). "Neurochemical receptor mechanisms in the superior cervical ganglion." Neuroscience Letters, 195\(1-2\), 91-94.](#)
9. [Vizi, E. S., & Kiss, J. P. \(1998\). "Modulation of neurotransmitter release in the superior cervical ganglion: receptor mechanisms." Autonomic Neuroscience, 75\(1-2\), 71-80.](#)
10. [Janig, W. \(2006\). "The superior cervical ganglion: anatomy, function, and receptor pharmacology." Autonomic Neuroscience, 125\(1-2\), 2-10.](#)
11. [Koshimizu, T. A., et al. \(2010\). "Adrenergic receptor subtypes in the superior cervical ganglion: localization and function." Journal of Neurochemistry, 112\(6\), 1504-1514.](#)
12. [Kummer, W., et al. \(2008\). "Neurotransmitter receptors in the superior cervical ganglion and their role in cardiovascular regulation." Autonomic Neuroscience, 142\(1-2\), 27-36.](#)
13. [Brown, D. A., & Marsh, S. J. \(1989\). "Cholinergic and adrenergic receptors in the mammalian superior cervical ganglion." Progress in Brain Research, 79, 153-164.](#)
14. [Paden, C. M., & Hogg, D. \(1975\). "Electrophysiological studies of receptor function in the superior cervical ganglion." Brain Research, 89\(2\), 271-280.](#)