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Review Article

Reflective Processes Carried Out with The Help of Spinal Neurons

Bon E. I*., Maksimovich N.Ye., Malykhina A.V.

Grodno State Medical University, Gorkogo St, Grodno, Republic of Belarus.

***Corresponding Author:** Elizaveta I Bon., Grodno State Medical University, Gorkogo St, Grodno, Republic of Belarus.

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Abstract

The data regarding the functional properties of motor neurons, preganglionic neurons and various types of spinal interneurons currently create a more solid basis for understanding the integrative mechanisms that carry out the main forms of reflex activity of the spinal cord. It is known that the scope of functions that the spinal cord can perform depends to a large extent on whether the connections of the spinal structures with the structures of the brain are preserved. Thus, in a decerebrated animal one can constantly observe (but in a very distorted form) complex forms of motor activity created by the spinal cord; after spinalization these forms of activity are lost. The reason for this effect of spinalization can be twofold. In some cases, the disconnection of the spinal cord and the overlying structures breaks the reflex arc responsible for the creation of the corresponding activity and including certain supraspinal structures. Such disorders include, for example, irreversible shutdown of periodic discharges of motor neurons of the respiratory muscles. In other cases, spinalization does not destroy the neural mechanisms of spinal activity in itself, but the latter cannot be realized due to a change in the state of these mechanisms.

Keywords: brain; spinal cord; neurons

Introduction

The data regarding the functional properties of motor neurons, preganglionic neurons and various types of spinal interneurons currently create a more solid basis for understanding the integrative mechanisms that carry out the main forms of reflex activity of the spinal cord [2]. It is known that the scope of functions that the spinal cord can perform depends to a large extent on whether the connections of the spinal structures with the structures of the brain are preserved. Thus, in a decerebrated animal one can constantly observe (but in a very distorted form) complex forms of motor activity created by the spinal cord; after spinalization these forms of activity are lost. The reason for this effect of spinalization can be twofold. In some cases, the disconnection of the spinal cord and the overlying structures breaks the reflex arc responsible for the creation of the corresponding activity and including certain supraspinal structures. Such disorders include, for example, irreversible shutdown of periodic discharges of motor neurons of the respiratory muscles. In other cases, spinalization does not destroy the neural mechanisms of spinal activity in itself, but the latter cannot be realized due to a change in the state of these mechanisms. A disorder of this kind is, for example, the loss of rhythmic locomotor movements. Locomotor movements can be caused by stimulation of certain points of the diencephalon and midbrain [5]. They disappear after spinalization, but can be re-induced after the above-described introduction of DOPA, which stimulates increased release of norepinephrine from the severed descending monoaminergic pathways. Restoration of a complex form of spinal activity in this way clearly indicates the preservation of the necessary neural mechanism in the spinal cord separated from the brain, the functioning of which, however, turns out to be impossible without some tonic influences from the suprasegmental centers [1]. Finally, a number of forms of spinal cord activity may also occur after the latter has separated from the suprasegmental structures; it is obvious that their neural mechanisms are little dependent on the receipt of any tonic influences from above [12]. This chapter will examine mainly reflex processes that can be carried out entirely by the structures of the spinal cord, as well as the role that these structures can play in the execution of descending commands [3].

Stretch Reflexes (Myotatic Reflexes)

The stretch reflex is the only spinal cord reflex in which direct control of motor neurons occurs by signals from primary afferents. A detailed study of this reflex was carried out long before the type of synaptic connection between the corresponding afferent fibers and motor neurons was determined, although the exceptionally short latent period of the tendon reflex (the phasic form of the stretch reflex caused by a short blow to the tendon) has always inclined researchers to assume the simplicity of the central part of the corresponding reflex arc [12]. Extensive studies of the nature of the involvement of motor nuclei in the stretch reflex have shown

that it is characterized by high locality and the absence of significant aftereffects. The stretch reflex is most pronounced in the extensor muscles, which must be in a state of tonic tension in order for the organism to be able to resist the force of gravity. Although the involvement of motor neurons in the stretch reflex is a purely spinal process, for which one or two spinal segments are sufficient, descending influences from the brain stem can facilitate or, conversely, suppress it, which is especially clearly demonstrated by the example of an extreme increase in stretch reflexes during decerebrate rigidity. One of the factors of such amplification is the increase in the activity of γ-motoneurons under the influence of descending influences, which in turn leads to an increased discharge of stretch receptors and a corresponding intensification of monosynaptic excitation of α-motoneurons ("γ-rigidity") [1-5]. The stretch reflex is always associated with inhibition of the activity of antagonist muscles due to the activation of inhibitory interneurons by collaterals of group 1a afferents, described above. A group of synergistic and antagonistic muscles, united functionally by the stretch reflex system, is called a myotatic unit. Such functionally related groups are included as elements in more complex forms of motor activity - maintaining posture, locomotion, etc. The reasons that caused the emergence of a mechanism for direct control of motor neurons only from the primary endings of muscle spindle stretch receptors in the course of evolutionary development are unclear. Perhaps such a feature ensures exceptional locality and reliability of the functioning of this mechanism, which controls the length of the contracting muscle [2-4].

All other reflex reactions originating from the receptors of the motor apparatus are polysynaptic in nature. These include the elongation reaction, the crossed stretch reflex (Philipson reflex), and the flexion stretch reflex. The elongation reaction consists of inhibiting the tonic tension of the stretched muscle during strong stretching; it can be considered as a system of reflex control of muscle tension, protecting it from rupture during stretching. The reflex elongation reaction is associated with the excitation of Golgi tendon receptors, which are extremely sensitive to tendon tension. As indicated above, electrical stimulation of group 1b fibers coming from these receptors causes disynaptic IPSP in the motor neurons of the corresponding muscle. Such IPSPs are intense enough to interrupt monosynaptic EPSPs evoked in the same motor neurons by monosynaptic activation from muscle spindles. The reflex inhibitory action of tendon receptors involves synergist muscles of the entire myotatic unit; antagonist muscles, on the contrary, are facilitated by the appearance in motor neurons of disynaptic EPSPs generated via 1b-interneurons. Because of this distribution of synaptic effects, the elongation reaction is sometimes referred to as the "perverted myotatic reflex," although the latter has a completely different genesis [9-12]. The flexion stretch reflex was discovered as an independent reflex reaction under artificial conditions - with electrical stimulation of muscle nerves. Intensification of such stimulation to values involving group II fibers from the secondary endings of muscle spindles is accompanied by the appearance of additional inhibition of homonymous motor neurons. This inhibition is quite widespread and goes beyond the myotatic unit; it coincides in time with the appearance of quite widespread facilitation of flexor motor neurons. Temporal and spatial characteristics indicate a connection of all these effects with a special system of spinal interneurons (II-interneurons), although, according to new data from Kirkwood and Sears, monosynaptic activation of motor neurons is also possible. Synaptic responses evoked by selective electrical stimulation of group II afferents (group I afferents were blocked by passing a direct current) were studied by Cangiano and Lutzenberger in decerebrated cats. These studies confirmed the occurrence of IPSP in extensor motor neurons with such stimulation. According to Lundberg et al., group II fibers of both flexors and extensors can exert three types of synaptic action in the spinal cord: weak homonymous monosynaptic excitatory action, disynaptic excitatory action, and trisynaptic inhibitory action. In a spinal animal, only excitatory pathways to flexor motor neurons and inhibitory pathways to extensor motor neurons function; however, in the presence of suprasegmental influences, more complex relationships in the spinal cord can also be manifested [17]. The functional meaning of the flexion stretch reflex has not been elucidated; such elucidation is complicated by the difficulty of evoking this reflex in isolation with appropriate stimulation. Since the secondary endings of muscle spindles are typical stretch receptors, the reflex reaction arising from them cannot be classified as a group of protective flexion reflexes and most likely represents some component of spinal locomotor coordination. With direct electrical stimulation of muscle nerves, due to the difficulty of precisely limiting the group of afferents, one cannot, however, be sure that the synaptic processes arising in the spinal structures actually reflect only this reaction [12-17]. The complex nature of reflex reactions during electrical stimulation of group II fibers is also evident from the fact that, along with the somatic component, it often includes a vegetative (vasomotor) reaction characteristic of protective reflexes [14].

Reflexes Of Cutaneous Origin

The skin surface contains a rich set of receptor structures capable of perceiving various characteristics of mechanical and temperature stimuli. Despite this diversity, the main spinal reflex reaction that is described during skin stimulation is always the ipsilateral flexion reflex, accompanied by a crossed extensor reflex under sufficiently strong stimuli, as well as a pressor vascular reflex [1-4]. Apparently, such universality reflects the fact that the animal responds with a protective reaction of avoiding the stimulus not only in the case of undoubtedly damaging pain stimuli, but also to light, non-damaging mechanical or temperature stimuli ("caution"). If such irritation does not then become severe, then the protective reaction can be overcome by purposeful behavior; otherwise, it will also intensify and prevent possible damage to the organism [5-7]. The severity of the cutaneous flexion reflex depends greatly on the strength of the stimulation; with increasing intensity of the latter, a greater number of flexor muscles are drawn into the reflex, and their reflex response is also intensified (i.e., a greater number of motor neurons pass from the subthreshold border region into discharge). The same effect is observed with increasing electrical stimulation of the cutaneous nerves. It may be thought that it is not simply the result of drawing a greater number of afferents into activity, but reflects the different qualitative nature of the flexion reflex caused by more and less excitable cutaneous afferents. There are no precise data in this regard; it is only known that stimulation of group 8 afferents is characterized by the appearance of a fairly long afterdischarge of motor neurons, which is absent during stimulation of α-afferents. Special reflex effects, which have been described by a number of authors and which do not fit into the flexion reflex scheme, may also be associated with the activation of low-threshold cutaneous afferents. Thus, Hagbart and Naess, in a detailed study of the effect of local stimuli of various areas of the skin surface on the excitability of various groups of motor neurons (the excitability of motor neurons was tested by changes in their monosynaptic discharge), found that areas of the skin above antagonist muscles caused not an increase, but a decrease in the excitability of flexor motor neurons and an increase in the excitability of extensor ones. The functional meaning of this phenomenon remains unclear to this day. Pressure on the pads of the paw also causes not flexion, but extension of the limb (the so-called extensor push); this reaction is one of the components of locomotion [12]. The main characteristics of the flexion reflex were obtained for the case of stimulation of high-voltage cutaneous afferents, and they are well supported by the above information regarding the functional properties of AFR interneurons [14]. A characteristic feature of the flexion reflex is the

possibility of involving all the muscles of the limb in activity when any of the nerves is stimulated. The degree of such involvement varies greatly depending on the strength of the stimulus and the type of muscle; the tension developed by each muscle (i.e., the fraction of motor neurons of its nucleus entering the discharge) is also very variable. The flexion reflex is also distinguished by its time characteristics: it is a jerk-type reflex and cannot be reproduced with high frequency [15]. The neural basis for the irradiation of excitation in the spinal cord during the flexion reflex is primarily the features of the synaptic connections of the AFR interneurons, the projections of which are distinguished by a wide multiplication due to the branching of axons and the connection of neurons into successive chains. The presence of extensive contralateral projections creates opportunities for cross effects, which are a constant component of the flexion reflex. Further, a significant role is played by the involvement of propriospinal neurons in the activity, which are effectively excited by high-threshold afferents. As shown by the recording of focal potentials of various parts of the transverse section of the spinal cord, the propriospinal pathways that ensure the transmission of the activity of these afferents pass through various funiculi of the white matter. The signals they transmit initially activate the interneurons of laminae V–VII, i.e. cells of the same type as those that receive direct influences from high-threshold afferents. Only then does synaptic activity appear in motor neurons. It is also possible that activation of motor neurons is also carried out through interneurons of the medial part of the ventral horn (plate VII, according to Rexed). In animals with an intact central nervous system, in which the flexion reflex is less intense than in an animal after spinalization, the transmission of influences from the propriospinal pathways to segmental interneurons is to a certain extent hindered due, apparently, to the presence of some constant inhibitory influences from the supraspinal structures on these cells. At the same time, under these conditions, spinal-bulbospinal connections are involved in the activity, which are an additional mechanism for the extensive spread of activity of high-threshold afferents and the creation of generalized responses [10-12]. The jerky nature of the flexion reflex indicates the probability of the appearance in its central structures after synchronous activation of a rather long period of depression. One of the mechanisms for the appearance of such a depression may be the development of a powerful presynaptic depolarization of the corresponding primary afferents. This depolarization develops not only during excitation of high-threshold muscle and cutaneous afferents, but also during excitation of afferents of other origin (with the exception of afferents of group 1a). In parallel with the development of presynaptic depolarization, there is a depression of the EPSP caused in spinal AFR interneurons by a test afferent wave. Such depression is somewhat longer than the presynaptic depolarization measured by the electrotonic potential of the dorsal root. However, it is necessary to keep in mind that the electrotonic potential reflects the processes occurring in the intracerebral part of the afferent fibers in a significantly weakened form. Therefore, it is possible that the complete course of depolarization of the terminals under the influence of a wave from high-threshold efferents is longer than can be judged by the electrotonic potential of the dorsal root [10-12]. At the same time, there is reason to believe that presynaptic depolarization is not the only cause of depression of reflex reactions caused by repeated afferent wave from high-threshold afferents. Processes developing in the interneurons themselves may play a significant role in the development of such depression; this is indicated, in particular, by the fact that the more complex the chain of interneurons included in intraspinal transmission, the more severe the depression is. The nature of long-term changes in the efficiency of synaptic transmission in chains of interneurons, which is sometimes referred to as habituation, is still unclear. Various possible mechanisms have been discussed in the literature, but there are no precise data on this issue [7-12]. It should be noted that a number of authors noted the appearance of not depolarization but, on the contrary, hyperpolarization changes in the central terminals of primary afferents upon stimulation of high-threshold afferents. Based on such data, it was suggested that there is a special "gate" system in the spinal cord at the level of afferent inputs, which, with the help of presynaptic hyperpolarization, can facilitate the flow of pain signals to interneurons, separating them from non-pain signals. However, other researchers failed to detect presynaptic hyperpolarization upon stimulation of high-threshold afferents. The electrotonic potentials of the dorsal roots of the spinal cord upon selective stimulation of cutaneous afferents of group III were most thoroughly studied in decerebrated cats in the work of Gregor and Zimmermann. The authors blocked conduction along thicker afferents by passing a direct current. In all cases, the wave from high-threshold afferents caused only depolarization of the central terminals. This pattern of changes was preserved even after spinalization of the animals or giving them barbiturate anesthesia. The exclusively depolarizing nature of presynaptic changes in nociceptive fibers was shown by Vyklitsky et al. and Burke et al. by irritating the skin with strong radiant heat, and also by Whitehorne and Burgess by direct stimulation of the terminals of these fibers through a submersible electrode and identifying them by means of adequate stimuli [15-17]. The reason for the discrepancy between the widely known data of Wall et al. and the data of a number of other researchers is unclear. It should be borne in mind that the data on the absence of presynaptic hyperpolarization upon activation of high-threshold afferents were obtained independently in several laboratories. Therefore, the idea that the transmission of pain signals causing the flexion protective reflex is based on a special "gate" mechanism that operates by removing the presynaptic inhibitory effect of high-threshold afferents requires additional experimental confirmation for its acceptance. Rudomin et al. described hyperpolarization of primary afferents of group 1a under the influence of activation of low-threshold cutaneous afferents. This phenomenon apparently has no relation to the problem of transmission of nociceptive signals [12].

Motor Reflexes of Visceral Origin

The motor spinal reflexes caused by excitation of the afferents of the celiac nerve have been studied in greatest detail. They are called visceromotor effects. Amassian was the first to show that the discharge of motor neurons of the thoracic segments occurs only when the afferents of the Ay8 group are involved in the activity. A more detailed study by Downman established that such a discharge is polysynaptic in nature; to some extent it also involves motor neurons of the muscles of the extremities. As in the case of the cutaneous flexor reflex, after transection of the spinal cord in the cervical region, the visceromotor reflex is strengthened, which indicates the presence of tonic inhibitory control of the corresponding reflex arc from the suprasegmental structures, apparently the medulla oblongata [9-12]. The reflex discharges evoked by stimulation of the splanchnic nerve afferents are not identical in all motor neurons of the thoracic region. They develop mainly in neurons innervating the muscles of the chest wall and the abdominal wall, and are poorly expressed in motor neurons innervating the extensor muscles of the back. In this group of motor neurons, IPSP and mixed reactions of the EPSP-IPSP type arise. According to the latent period, the effects in motor neurons are divided into two components - "early" and "late". Transection of the spinal cord does not have a significant effect on the development of the early components of the reflex response and eliminates the late components. Therefore, there is reason to believe that the central mechanisms for the implementation of the latter are significantly different: if the early discharge is created by spinal neural mechanisms, then the late one requires the participation of supraspinal structures, in particular the spinobulbospinal system [12-17]. Visceromotor effects on motor neurons of the lower limb

muscles are detected both by the appearance of reflex discharges in the corresponding ventral roots and by changes in test segmental mono- and polysynaptic reactions after stimulation of the celiac nerve. For the appearance of the indicated changes, as in the case of the study of thoracic motor neurons, it is necessary to excite the high-threshold afferents of the Ay8 group. Intracellular recording of potentials of lumbar motor neurons showed that the afferent wave from the celiac nerve causes rather intense and prolonged EPSP in flexor motor neurons, divided into "early" and "late" components. In extensor motor neurons, synaptic changes are of a different nature. The early component, which in this case has a more significant latent period, can be de- and hyperpolarizing. The late component is always hyperpolarizing; IPSP in this case have a very significant amplitude and duration. After transection of the spinal cord, these prolonged IPSP do not occur, which indicates their possible connection with supraspinal influences [5-10]. Visceromotor effects, like cutaneous flexion reflexes, characteristically depend on the frequency of afferent impulses. Even with very rare repetitions (once per second), successive reactions weaken; a more or less constant level of individual reactions can be ensured only with stimulations once every 4-5 seconds. At a frequency of four to five times per second, successive responses soon disappear completely [8,9]. A comparison of this depression with a similar depression of polysynaptic reactions evoked in the same motor neurons by segmental high-threshold afferents shows that in the second case the depression has a shorter time constant; its depth is also significantly less. It is obvious that the severity of such depression increases with the increase in the complexity of the neural circuit transmitting influences to motor neurons, which confirms the above assumption about the possible role of long-term changes in the activity of interneurons in its origin [9-12]. Certain information about the nature of adequate visceral stimuli that can create the described visceromotor effects was provided by experiments with intracellular recording of motor neuron responses during controlled stretching of internal organs (bladder or rectum). In spinal animals, such stretching causes qualitatively identical depolarization changes in both flexor and extensor cells, the magnitude of which depends on the intensity of stretching. In decerebrated animals, the changes in flexor motor neurons remain the same, while in extensor neurons, with a weak stretching force, membrane depolarization develops, which, with increased stretching, is replaced by hyperpolarization or hyperpolarization-depolarization shifts. Particularly clear reciprocal changes are observed after giving such animals narcotic substances [10,12]. The presented data show that visceromotor effects largely coincide in their nature with flexion protective reflex reactions of exteroceptive origin. This coincidence is quite explainable in light of the above data regarding the features of the convergence of visceral and somatic afferent influences on the interneuron systems of the spinal cord. At the same time, visceromotor effects also have some special features that indicate a certain specificity [5-8].

Spinal Autonomic Reflexes

The neural structures of the spinal cord also provide reflex reactions of the internal organs, primarily the vascular system. Due to the difficulty of analyzing such a slow and widespread reaction as a change in blood pressure, the discharge of sympathetic pre- and postganglion neurons is usually analyzed, which largely reflects the vasomotor reaction. This discharge can be recorded in the sympathetic chain, renal and cardiac nerves, as well as in the white connecting branches of the thoracic and lumbar segments and the cervical sympathetic nerve. In animals with an intact central nervous system, it is divided into "early" and "late" components; the first of them is preserved after transection of the spinal cord under the oblongata (provided, of course, that the animal maintains a sufficiently high reflex excitability and, accordingly, a sufficiently high level of blood pressure). The late component, according to most authors, disappears after such a transection, which is considered as an indication of its connection with the activation of the suprasegmental structures [1]. However, Khautshishm and Lukoshkova obtained data on the possibility of the appearance in spinal animals of reflex responses of sympathetic neurons with very long latent periods. These data were initially obtained only for postganglionic neurons, but they were recently confirmed by Lebedev et al. for the discharge in the white connecting branch. Most researchers designate the "early" component of the sympathetic discharge as spinal, and the "late" as supraspinal, although in light of the above data the latter designation is inaccurate [12-15]. The early response of sympathetic preganglionic neurons was first analyzed by Beecham and Pearl on spinal animals and then by a number of authors on intact anesthetized animals. Gokin and coworkers showed that the appearance of a small short-latency discharge upon stimulation of the splanchnic nerve is associated with the activation of slowly conducting fibers of the Ap group ("super-early" discharge). The main activation of preganglionic neurons occurs upon excitation of the fibers of the At8 group. High cervical transection not only does not weaken these discharges, but, on the contrary, to some extent strengthens them. Upon stimulation of somatic afferents, the discharge is always associated with the activation of fibers of groups II and III; it does not occur upon activation of fibers of group I. The measurement of the central delay, given the complexity of the intraspinal pathway of the corresponding reflex arcs, can be carried out only approximately; apparently, for the main component it is 7-9 ms in the lower thoracic segments and is the same when stimulating both visceral and somatic afferents [11-16]. Unlike the early discharge, the late discharge of preganglionic neurons is a generalized reaction. If the early discharge occurs mainly in segments close to the entry point of the corresponding afferents, then the late discharge has the same character when stimulating the nerves of all four limbs. It is distinguished by significant variability in amplitude and duration compared to those of the early discharge [10-13].

It was initially suggested that the late discharge is lower threshold than the spinal discharge.

However, this difference is apparent and is also associated with the activation of group II and III fibers [12]. A characteristic feature of the late response, which is easily detected when recording the activity of individual efferent fibers, is the presence of an extremely long period of "silence" after the discharge. In the cardiac and renal nerves, after stimulation of the somatic nerves, it reaches 0.5-1.0 s. The depression is so strong that in the first 500-600 ms the test reflex discharge cannot be evoked at all. Apparently, supraspinal influences play a significant role in the development of such depression; after transection of the spinal cord under the medulla oblongata, its intensity and duration are significantly reduced [9-14]. The role of thin afferents of group IV (C) in the generation of sympathetic neuron discharge is not entirely clear. Such a discharge is not detected when single stimuli are applied to peripheral nerves, but it can be evoked by rare rhythmic stimulation (1-2 impulses/sec) and after preliminary tetanization. The excitatory action of C-fibers can also be detected by blocking A-fibers in the stimulated nerve using direct current. Kut and Perets-Gonzalets, using this technique, detected a discharge of postganglionic neurons in the cardiac and renal nerves in response to stimulation of group IV fibers in muscle nerves; it had a latent period of about 500 ms. Koizumi and coworkers found similar discharges in the renal nerve and white connecting branches when C-fibers of cutaneous nerves were stimulated [12,17]. In spinal animals, it is usually not possible to elicit sympathetic efferent neuron discharges by isolated C-fiber stimulation. However, in chronic spinal cats, these responses can be recorded four to six weeks after surgery. Thus, despite the exceptionally long latent period, they may be generated by purely spinal mechanisms [11-15].

Certain information is currently available regarding spinal reflex reactions carried out by parasympathetic neurons innervating the musculature of the bladder wall through the corresponding peripheral ganglia. It is known that the discharge of parasympathetic neurons always precedes spontaneous contractions of the bladder; the discharge frequency of these cells increases with an increase in intravesical pressure. As indicated above, excitatory effects on parasympathetic neurons during stimulation of high-threshold afferents occur in intact animals with a very long latent period, indicating the participation of suprasegmental structures in its occurrence. Along with such an excitatory effect, short-latency inhibition of parasympathetic neurons is observed, created by spinal mechanisms. Apparently, the functional role of such an inhibitory reflex influence is to create a certain background inhibition of the activity of these neurons, necessary to prevent uncontrolled urination. As the activation of receptor structures increases, excitatory influences begin to prevail over inhibitory ones in determining the nature of the activity of parasympathetic neurons. Along with direct inhibition, parasympathetic neurons are apparently also subject to recurrent inhibition, which has a similar functional significance. Spinal inhibitory neurons of these reflex arcs can be a substrate for descending signals during voluntary urination, which switch the state of parasympathetic neurons from one of the types of inhibition to excitation. It is characteristic that the indicated synaptic reactions in parasympathetic neurons can be caused by stimulation not only of the afferents of the pelvic nerve (i.e., the receptors of the bladder itself), but also by stimulation of the somatic afferents of various nerves of the hind limbs. Such stimulation causes both early inhibition of these neurons and their late (supraspinal) excitation; the latter can be even longer than in the case of stimulation of visceral afferents. At the same time, in the adjacent somatic motor neurons, stimulation of somatic high-threshold afferents causes EPSPs typical of the flexion reflex. Thus, we are not talking about nonspecific influences from somatic afferents on parasympathetic neurons, but about specific activation of a specialized spinal neural system that regulates the process of urination. Apparently, the receipt of signals from various afferent sources facilitates the inclusion of interneurons of this system in the activity and is essential for its effective operation [8-12]. In chronic spinal conditions, a significant change in the synaptic reactions of parasympathetic neurons is observed. Under these conditions, irritation of the celiac nerve causes early excitatory effects in some of them. In animals with an intact nervous system, such reactions are never recorded; probably, the corresponding intraspinal connections are under the tonic inhibitory influence of the suprasegmental structures. Early excitatory effects may be the basis of the imperfect urination reflex, which is observed in chronic spinal animals. And in this case, such effects can be caused by irritation of not only visceral but also somatic afferents. This coincides with the known data regarding the facilitation of the urination reflex in chronic spinal animals with various exteroceptive stimuli [6-12].

Involvement Of Spinal Neurons in The Execution of Commands Sent by Suprasegmental Centers

Spinal neurons are the executive apparatus for an immense number of responses of the organism of varying complexity, carried out through the suprasegmental structures. A description of the behavioral features of spinal structures in such reactions is beyond the scope of this chapter. However, it is advisable to stop in conclusion on the general patterns of activation of various types of neurons of the spinal cord upon receipt of motor commands through the main descending systems originating in certain suprasegmental centers [10-12]. As already indicated in the anatomical and histological review, descending systems can be connected both directly with spinal motor neurons and directly control the latter, and with various groups of interneurons [12]. The number of fibers of the corticospinal (and possibly also rubrospinal) tract in higher mammals (mainly in primates) that form direct connections with motor neurons is small and constitutes about 2% of the entire pyramidal tract in the macaque and about 8% of the entire pyramidal tract in humans. As Bernhard, Bohm, and Peterson first showed, direct stimulation of the precentral region of the cerebral cortex in monkeys can reveal a discharge of motor neurons in the lumbar segments with a latent period of only 4.7 ms, which is consistent with the assumption that it arises through the monosynaptic connections described. Monosynaptic EPSPs during discharge of the red nucleus are found in the motor neurons of the distal muscles of the limbs in monkeys. These same motor neurons can receive monosynaptic influences from the pyramidal tract. In humans, during neurosurgical operations, stimulation of the internal capsule with a stereotaxically embedded microelectrode also revealed the appearance of a facilitation of the testing monosynaptic reflex with a latent period, indicating the monosynaptic nature of the transmission of the corresponding descending signal to motor neurons [5-16]. After experimental destruction of the corticospinal tracts, there is no complete loss of voluntary limb movements; after the initial period of their impairment, their almost complete restoration occurs, but the fine movements of the phalanges, as well as the movements that ensure rapid decision-making, remain partially lost. Such data allow us to conclude that direct control of motor neurons by the corticospinal tract, which cannot be replaced by the action of other descending systems that establish indirect connections with the motor nuclei, is necessary only to ensure fine voluntary movements in the most highly organized animals [7-15]. In contrast to the lateral descending systems, the medial descending tracts in all vertebrates, starting with the lowest (cyclostomes), form well-developed monosynaptic connections with motor neurons. The presence of terminals of vestibulo- and reticulospinal fibers in the motor nuclei is traced morphologically; electrophysiological monosynaptic EPSPs in individual motor neurons upon stimulation of the Deiters nucleus have been described by a number of authors. In the overwhelming majority of cases, such EPSPs are recorded in extensor cells; in flexor cells, TG1SPs with a latent period arise, indicating the participation of spinal interneurons in their generation. Particularly effective direct activation of motor neurons by vestibulospinal tracts occurs in the cervical spinal cord, in particular in the extensor muscles of the neck; monosynaptic EPSPs of vestibular origin have also been described in this region, indicating the presence of direct inhibitory fibers in the vestibulospinal tract [10-15]. All these effects are ipsilateral, and the transition of vestibular descending influences to the contralateral side is already accomplished at the spinal level by activation of intercalated cells. The appearance of monosynaptic EPSPs in flexor motor neurons caused by activation of reticulospinal fibers of the ventrolateral funiculus was established by Grillner and Lund, as well as Shapovalov, Grantin, and Kurchav [9-13]. They are well expressed in the motor neurons of the flexors of the knee and ankle; the motor neurons of the extensors of these joints are simultaneously subject to inhibition. The motor neurons of the hip joint muscles and the finger muscles can receive monosynaptic excitatory influences of both reticular and vestibular origin, but they affect different cells of the same nucleus. Reticulospinal fibers also monosynaptically excite the motor neurons of the thoracic and cervical spinal cord. In the cervical region, monosynaptic EPSPs of vestibular and reticular origin can be observed in the same motor neuron; such motor neurons innervate muscles that cannot be assigned to one of the antagonistic groups - flexors or extensors - and cause head movements [11-17]. Experimental switching off of the vestibulospinal pathways causes a characteristic motor effect - a drop in the reflex tone of the extensor muscles; this effect is especially noticeable in the case of a preliminary increase in such tone (for example, during decerebration). Apparently, direct vestibulomotor neuron connections are the main mechanism supporting it

due to tonic facilitating synaptic influences on the corresponding motor neurons [12]. Vestibulomotor neuron connections are represented mainly by axodendritic synapses, and therefore their efficiency during the natural rhythmic activity of neurons of the Deiters nucleus is hardly so high as to independently cause the impulses of motor neurons. However, the resulting depolarization of their membrane obviously significantly increases the efficiency of all other synaptic influences converging on the same cells, primarily from muscle afferents of group 1a. Elimination of tonic influences from the Deiters nucleus, on the contrary, passively inhibits motor neurons due to the loss of facilitating depolarization (disfacilitation). Such disfacilitation is well expressed upon stimulation of the anterior lobe of the cerebellum, the Purkinje cells of which in turn exert a direct inhibitory effect on the vestibular neurons. As a result, hyperpolarization develops in the extensor motor neurons of the spinal cord, which, unlike normal IPSP, does not change with a change in the membrane potential of the cell or the injection of chloride ions into it and reflects the loss of tonic depolarizing synaptic action. There is still no such clear idea regarding the functional role of monosynaptic influences of the reticulospinal tracts; it is known, however, that upon stimulation of some subcortical nuclei of the cerebellum, facilitation of the flexor motor neurons develops. It is possible that this effect is mediated through the reticular formation and the reticulospinal tracts. Recently it has been shown that direct descending pathways also enter the spinal cord from such structures as the fastigial nucleus of the cerebellum and the dorsal nuclei of the hypothalamus [5-8]. The functional significance of these projections has not yet been fully understood. Indirect transmission of suprasegmental signals to motor neurons is accomplished to a significant extent through the system of short propriospinal neurons, which receive synaptic inputs primarily from descending pathways. The functional properties of propriospinal neurons and the features of their connections with descending fibers and subsequent neurons create the possibility of significant integration of signals arriving through various subdivisions of descending tracts, as well as a certain transformation of the temporal structure of the signal. The system of short PS neurons obviously also produces spatial redistribution of processed signals among motor nuclei and coordination of their activity.

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