PHYSIOLOGY

ELECTROPHYSIOLOGICAL CHARACTERISTICS OF THE INTEROCEPTIVE REFLEX ARC

COMMUNICATION II. CHARACTERISTICS OF THE REGIONAL VASCULAR REFLEXES ARISING DURING STIMULATION OF THE MECHANORECEPTORS OF THE URINARY BLADDER

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In previous communications [1, 2] we made an attempt to compare the true and composite reflexes arising during stimulation of the mechanoreceptors of the urinary bladder. An example of a true reflex, changes in the tone of the ureter were studied, and of a composite reflex — changes in the general arterial pressure.

It was shown that as the strength of stimulation of the mechanoreceptors of the urinary bladder increased, there was a corresponding increase in the intensity of afferent impulses in the pelvic nerve, whereas the efferent impulses to the ureter, like the tone of the ureter, are altered in different ways depending on the strength of stimulation: weak stimuli (3 - 25 mm of mercury) cause an increase in the efferent impulses to the ureter registered in the central end of the pudendal nerve and contraction of that organ. Stimuli of moderate strength (25 - 30 mm of mercury) sometimes cause alternate periods of intensification and weakening of the efferent impulses and rhythmic contraction and relaxation of the ureter. Stronger stimuli (over 25 mm of mercury) cause inhibition of efferent impulses and relaxation of the ureter. Only still greater intensity of stimulation causes reflex changes in the general arterial pressure, i.e. appearance of a composite reflex.

The aim of the present work, which dealt with the interrelation between the true and composite reflexes bringing about the complex reaction of the blood pressure, was to trace the movement within the central nervous system of the process of excitation caused by stimulation of the mechanoreceptors of the urinary bladder. For this purpose we attempted to determine the order in which the following vessels became involved in the reaction — the vessels of the urinary bladder whose mechanoreceptors were being stimulated, the vessels of the kidney as a region functionally connected with the stimulated interoceptive zone, and the vessels of the skin of the hind limb and head as regions anatomically and functionally unconnected with the stimulated zone — and to compare these changes in the individual vascular regions with the reaction of the general arterial pressure.

The majority of present-day workers [5, 6, 7, 8 and others] consider that the reflex regulation of blood vessels is carried out by sympathetic fibers by way of changes in tonic efferent impulsation. These impulses were recorded by several authors [3, 4, 9], who called them "slow" tonic impulses and they either have a constant rhythm or they change synchronously with respiration or with the rhythm of the heart and they differ in different nerves only in frequency or amplitude. These authors consider that the main function of the sympathetic system is to maintain the tone of the blood vessels, i.e. to maintain a constant vasoconstrictor action. However, certain authors [3] could not detect any form of change in these impulses during stimulation of different internal organs.

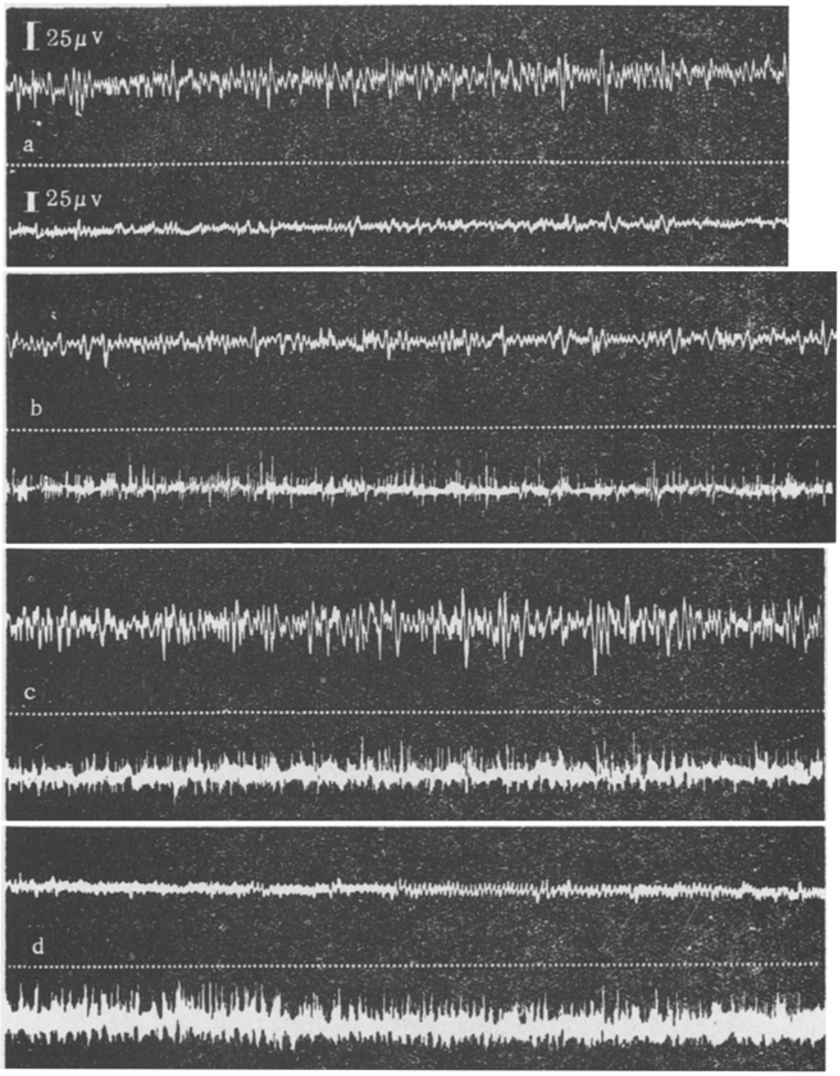

Fig. 1. Reflex changes in the efferent impulsation in the hypogastric nerve and the magnitude of the afferent impulsation in the pelvic nerve during stimulation of varying intensity of the mechanoreceptors of the urinary bladder.
a) Initial efferent impulsation with a rhythm of 68 per sec, amplitude 29 μv. No afferent impulsation present under these conditions (before stimulation); b) pressure inside the urinary bladder increased to 22 mm of mercury. Efferent impulsation with a rhythm of 40 per sec, amplitude 21 μv. Afferent impulsation 130 per sec; c) pressure inside the urinary bladder increased to 56 mm of mercury. Efferent impulsation with a rhythm of 80 per sec, amplitude 37 μv. Afferent impulsation 250 per sec; d) pressure inside the urinary bladder increased to 74 mm of mercury. Efferent impulsation with a rhythm of 20 per sec, amplitude 10 μv. Afferent impulsation 300 per sec. Inscriptions on the oscillograms from above down: action potentials of the central segment of the hypogastric nerve, time marker 0.01 sec, action potentials of the peripheral segment of the pelvic nerve.

EXPERIMENTAL METHOD

In acute experiments on 52 cats (under urethane anesthesia) the arterial pressure in the carotid artery and the pressure inside the urinary bladder were recorded by a mercury manometer. Meanwhile recordings were made on a cathode-ray oscillograph of the impulses in the peripheral end of a branch of the pelvic nerve (afferent

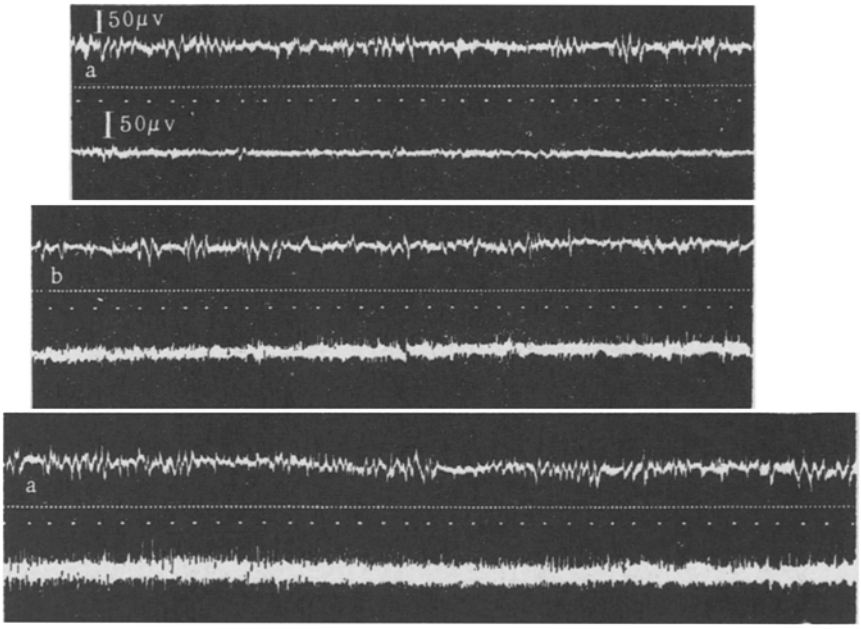

Fig. 2. Reflex changes in the efferent impulsation in the renal nerve and the magnitude of the afferent impulsation in the pelvic nerve during stimulation of varying intensity of the mechanoreceptors of the urinary bladder.
a) Initial efferent impulsation with a rhythm of 45 per sec, amplitude 58 μv. Afferent impulsation in the pelvic nerve absent under these conditions (before stimulation); b) pressure inside the urinary bladder increased to 28 mm of mercury. Efferent impulsation with a rhythm of 45 per sec, amplitude 73 μv. Afferent impulsation 300 per sec. Inscriptions on the oscillograms from above down: action potentials of the central end of the renal nerve, time marker 0.01 sec, time marker 0.05 sec, action potentials of the peripheral end of the pelvic nerve.

impulsation from the mechanoreceptors of the urinary bladder) and the impulses in the central ends of one of the following four nerves: hypogastric, renal, saphenous and the efferent fibers of the cervical sympathetic nerve. In recording the impulses we made use of immersible electrodes as suggested by P. A. Kiselev.

Since the majority of the nerves investigated are mixed, to make sure that the impulses recorded are, in fact, concerned with the regulation of vascular tone, a special series of experiments was carried out in which the vessels of the skin and kidney were perfused. In these experiments the reaction of the vessels were judged by the number of drops flowing from the vein. An increase in the rate of flow of drops indicated vasodilatation, a reduction – vasoconstriction.

EXPERIMENTAL RESULTS

The efferent impulses to the vessels of the urinary bladder were recorded in the central end of the hypogastric nerve, for it is known from morphological investigations [10] that it is in this nerve that the efferent nerve fibers pass to the vessels of the urinary bladder.

In the absence of stimulation of the mechanoreceptors of the urinary bladder a "slow" stream of impulses is observed in the nerve (Fig. 1, a). Weak stimulation of the mechanoreceptors of the urinary bladder (4 - 22 mm of mercury) leads to inhibition of the efferent impulses in the hypogastric nerve (Fig. 1, b). This inhibition develops after a relatively long latent period (from 1 to 4 sec) and reaches its maximum gradually without any concomitant rise of arterial pressure.

The range of variation of intensity of stimulation within which inhibition of efferent impulsation arises in the hypogastric nerve was very small in each experiment. Further increase in the intensity of stimulation of the

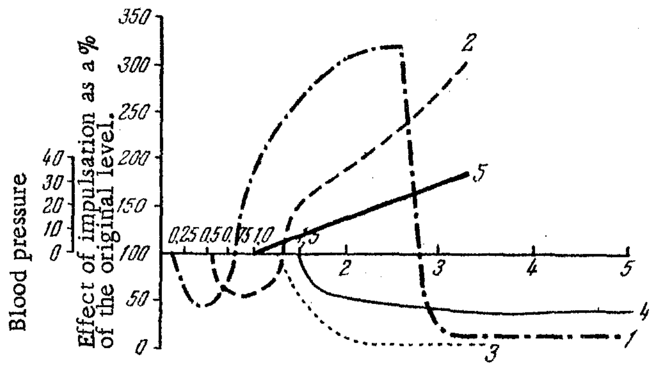

Fig. 3. Relationship between the changes of efferent impulsation in different nerves and the magnitude of the reflex rise in the general blood pressure and the intensity of stimulation of the mechanoreceptors of the urinary bladder.
Along the horizontal axis are represented the strengths of stimulation of the mechanoreceptors of the urinary bladder in relative units. One unit is taken to be the strength of stimulation causing threshold changes in the general blood pressure. Along the vertical axis are represented the changes in the general blood pressure in mm of mercury and changes in the efferent impulsation in different nerves as percentages of their original value. The initial level is taken as 100%. For an explanation, see text. 1) Efferent impulsation in the hypogastric nerve; 2) the same in the renal nerve; 3) the same in the saphenous nerve; 4) the same in the cervical sympathetic trunk; 5) changes in the general arterial pressure.

mechanoreceptors of the urinary bladder then causes strengthening of the efferent impulses in the central end of the hypogastric nerve (Fig. 1, c).

This strengthening of impulsation has a short latent period (0.3 - 1.5 sec) and reaches its maximum quite quickly. The range of intensity of stimulation of the mechanoreceptors of the urinary bladder within which strengthening of the efferent impulsation arises in the hypogastric nerve is very great and varies between 10 and 80 mm of mercury.

It is important to mention that strengthening of the efferent impulsation arises during stimulation of an intensity still inadequate to cause reflex changes in the blood pressure and only after further strengthening does it cause an increase in the blood pressure.

If, however, the pressure in the urinary bladder is increased still further, inhibition of the efferent impulses in the hypogastric nerve develops (Fig. 1, d). This inhibition is characterized by a shorter latent period (0.3 - 1.5 sec) than the inhibition observed by stimulation of low intensity. In all cases the intensity of afferent impulsation in the pelvic nerve increases in proportion to the strength of stimulation (see Fig. 1, a, b, c, d).

We now turn to the analysis of the changes in the efferent impulses in the renal nerves which, in the absence of stimulation of the mechanreceptors of the urinary bladder, consisted of a series of "slow" potentials (Fig. 2, a). Only the first two stages are demonstrated by the changes in the efferent impulsation in the renal nerves: inhibition and strengthening, and furthermore for each of these stages to appear it was necessary to apply more intensive stimulation of the mechanoreceptors of the urinary bladder than for the appearance of the corresponding stages in the nerves of the urinary bladder. The first stage — the stage of inhibition — arose during stimulation at a strength of 15 - 35 mm of mercury (Fig. 2, b). At the time of appearance of a reflex rise of blood pressure, some degree of inhibition of the efferent impulsation to the kidney could still be observed.

Further increase in the intensity of stimulation caused strengthening of the impulses (Fig. 2, c) which lasted as long as the intensity of stimulation of the mechanoreceptors of the urinary bladder continued to increase.

We did not observe the development of a stage of inhibition of the impulses in the renal nerves on further strengthening the stimulation of the mechanoreceptors of the urinary bladder.

From recordings of the efferent impulses in the nerves supplying the skin of the hind limb and in the cervical sympathetic nerve we were able to find only the first stage of changes of impulsation and in the cutaneous nerves this stage appeared at strengths of stimulation (25 mm of mercury and higher) slightly greater than those which caused reflex changes in the arterial pressure.

In the cervical sympathetic nerve inhibition of impulsation developed at still higher strengths of stimulation (28 mm of mercury and higher).

In the special experiments with perfusion of the skin and kidney, which were mentioned above, it could be demonstrated that the efferent impulses recorded in the nerves supplying these regions are directly related to to the vessels. According to our observations, constriction of the vessels corresponded to strengthening of the impulsation and dilatation of the vessels to its inhibition.

DISCUSSION OF RESULTS

As seen from the facts which have been given, changes in the impulsation in the efferent nerves appear in a definite sequence. In order to understand the phenomena described, let us take as an initial unit the intensity of stimulation of the mechanoreceptors of the urinary bladder at which changes in the arterial pressure first appear. If this system is used, the changes in the impulsation in the various efferent nerves may be compared in a composite diagram as illustrated in Fig. 3, in which are included the results of all the experiments.

Figure 3 illustrates clearly the gradual implication of the individual vascular regions in the reaction as the intensity of stimulation of the mechanoreceptors of the urinary bladder is increased.

Examination of Fig. 3 shows that 0.25 conventional units is sufficient to implicate in the reaction the vessels of the urinary bladder itself. When the intensity of stimulation reaches 0.5 conventional units, the vessels of the kidney are implicated in the reaction. The vessels of both the urinary bladder and the kidney respond characteristically at this time by dilatation.

Further increase in the intensity of stimulation leads to strengthening of the impulsation to the vessels of the bladder and kidneys, i.e. vasoconstriction develops.

Increase of impulsation in the hypogastric nerve begins slightly earlier (0.7 conventional units) than in the renal nerves. It begins in the latter at a time when the first signs are observed of the reaction of the arterial pressure and requires 1.2 conventional units. At this time, as seen from the figure, the skin vessels and the vessels innervated by the cervical sympathetic nerve are included in the reaction. Obvious depression of impulsation is observed in these nerves.

Further study of Fig. 3 shows that as the intensity of stimulation increases and the magnitude of the reflexes affecting the arterial pressure becomes greater, the impulsation in the cutaneous and sympathetic nerves is still further depressed, the impulsation in the renal vessels continues to increase, whereas in the hypogastric nerves inhibition of impulsation reappears and is deeper than was observed at first.

The facts presented may thus be considered to show convincingly a movement of the process of excitation, to involve in the first place the centers most closely connected functionally with the particular interoceptive zone. In this way the vascular changes are confined to a relatively small area and changes in the general arterial pressure do not take place. Meanwhile, as the intensity of stimulation of the mechanoreceptors continues to rise, more and more new structures in the central nervous system become involved in the reaction in a definite order. This can be seen from the fact that soon after the vessels of the bladder are included in the reaction the renal vessels follow suit. If, however, the mechanism of the observed effects consisted purely of the gradual involvement of more and more new structures, it would be expected that an extraordinarily widespread involvement of the whole blood stream might take place which would not be without danger to the animal.

Our results show that side by side with the successive involvement of more and more new centers there are mechanisms which limit this irradiation.

Even at the very beginning, when the vessels of only the urinary bladder and kidneys were involved in the reaction, we can discern these mechanisms in the fact that initially the impulsation in the hypogastric and renal nerves is not strengthened but is inhibited (see Fig. 3). In other words, even in the beginning inhibition is created which limits the area of vasodilatation and therby prevents an increase in the arterial pressure.

When the rising intensity of stimulation causes this block to give way, the depression of the efferent impulsation is replaced by strengthening, but at this time inhibition is arising in other regions of the central nervous system, as shown by depression of the impulsation to the vessels of the skin and of the region innervated by the cervical sympathetic nerve (see the inhibition of the impulsation in these nerves shown in Fig. 3). With an even greater rise in the intensity of stimulation, inhibition of the efferent impulsation to the vessels of the urinary bladder arises once more and in consequence they are dilated. It is possible that this dilatation plays the part of a specific "protective" mechanism in preventing spasm of the vessels which may lead to prolonged ischemia of the organ.

Constant impulsation from the mechanoreceptors of the urinary bladder thus not only creates conditions for the irradiation of excitation but also promotes the onset of inhibition. The interaction of these two processes ultimately determines the reaction of the arterial pressure.

SUMMARY

Acute experiments were performed on cats. The changes of the efferent impulsations were studied in the sympathetic fibers innervating various vascular areas in vesicular mechanoceptor stimulation of different intensity.

Low intensity stimulation is required to produce changes in the impulsation of the nerves innervating the blood vessels of the urinary bladder proper (hypogastric nerve). With the rise of the intensity of stimulation these changes begin to spread to the more remote (in functional condition) vascular areas. At first the change of the efferent impulsation occurs to the renal vessels, then to the blood vessels of the skin of posterior extremity and, finally, to the vessels innervated by the cervical sympathetic nerve. With the rise of intensity of stimulation of the vesicular mechanoceptors these changes in the efferent impulsations in the hypogastric nerve undergo 3 phases: the first phase corresponds to the inhibition of the efferent impulsation, the second to its intensification and the third – to inhibition. Only the first two phases are observed in the renal nerve and only one in the skin and cervical sympathetic nerves. The intensification of the impulsation always leads to constriction while its inhibition – to the dilatation of the vessels.

LITERATURE CITED

[1] T. S. Lagutina, Proceedings of a Conference of the Young Scientists of the Institute of Normal Physiology of the AMN SSSR, pp. 51-52, Moscow, (1955).*

[2] Idem, Byull. Eksptl. Biol. i Med. No. 7, 3-8 (1957).

[3] E. D. Adrian, D. W. Bronk, G. Phillips, J. 74, 115, 133 (1932).

[4] D. W. Bronk, L. K. Ferguson, D. Y. Soland, Proc. Soc. Exper. Biol. a. Med. 31, 579-580 (1934).

[5] B. Folkow, Physiol. Rev. 35, 629-656 (1955).

[6] B. Uvnas and B. Folkow, Acta Physiol. scand 15, 389-406 (1948).

[7] B. Folkow, G. Ström and B. Uvnas, Acta Physiol. scand 17, 327-338 (1949).

[8] B. Folkow, G. Ström and B. Uvnas, Acta Physiol. scand 21,145-158 (1950).

[9] B. Gernandt, G. Liljestrand and Y. Zottermann, Acta Physiol. scand. 11,239-247 (1946).

[10] O. R. Langworthy and E. L. Murphy, J. Comp Neurol. 71, 487-509 (1939).

* In Russian.