system have stronger antiaggregating properties than vessel walls of the venous system. In some experiments the walls of veins had virtually no antiaggregating action. The antiaggregating activity of the walls of different arteries, incidentally, also varied: The walls of the carotid artery had the strongest activity, walls of the femoral artery the weakest.

The results of the comparative study of the response of platelets from the aorta and posterior vena cava to vascular antiaggregating factor are given in Table 2. Platelets, isolated both from the aorta and the posterior vena cava, responded equally to the antiaggregating action of the walls of all vessels studied.

High aggregating activity of the platelets was exhibited in the aorta despite the considerable antiaggregating action of the vessel wall. Meanwhile in the posterior vena cava, function of both platelets and vessel wall was at a much lower level. Platelets from both parts of the vascular system responded equally to vascular antiaggregating factor. A mechanism ensuring different levels of aggregating activity of platelets in arterial and venous blood probably exists. It can be tentatively suggested that a definite role in the realization of this mechanism is played by the presence of the arteriovenous oxygen difference, which is responsible for the more intensive rate of metabolism in platelets of the arterial system compared with the venous. Hence there is an arteriovenous difference with respect to function of blood vessel–platelet hemostasis, which plays a definite role in the regulation of the state of blood aggregation in the body.

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EFFECT OF DESGLYCINEARGININE-VASOPRESSIN ON EXCITABILITY OF DEFENSIVE COMMAND NEURONS IN *Helix lucorum*

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Vasopressin and its analogs can influence learning and memory processes [3, 11]. To explain the mechanisms of this effect, the action of vasopressin on nerve cell function must be studied. It has recently been shown that vasopressin analogs have most frequently an excitatory action on spontaneous neuronal activity and also induce or potentiate renal activity. This effect has been described both in higher vertebrates [1, 9, 10] and an invertebrates [3, 5-7]. However, the participation of vasopressin and its analogs in the regulation of evoked activity has received little study [8].

The object of this investigation was to study the effect of desglycinearginine-vasopressin (DG-AVP) on excitability of command neurons involved in the organization of the defensive reflex in snails.

EXPERIMENTAL METHODS

Experiments were carried out on a preparation of the isolated CNS of the snail *Helix lucorum* L. Activity of identified neurons LaPa4, RPa3, and LPa2 [2]. Excitability of neurons

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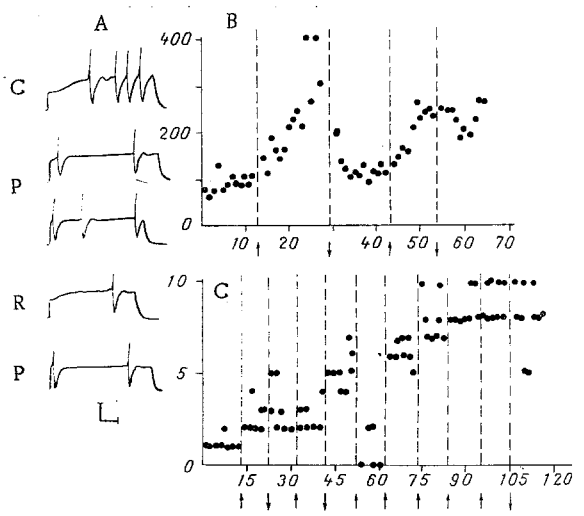

Fig. 1. Increase in excitability of neurons under the influence of DG-AVP. A) Examples of responses of neuron LPa2 to depolarizing pulses in control (C), during application of DG-AVP (P), and on rinsing to remove the preparation (R). Calibration: 20 mV, 400 msec; B) abscissa, serial No. of stimulus: ordinate, time course of value of 1/LP (in % of mean control level, average for 17 neurons). Arrow pointing upward — beginning of application of DG-AVP; arrow pointing downward — beginning of rinsing; C) ordinate, values of 1/LP recorded in one neuron LPa3 (in Hz). Remainder of legend as to Fig. 1B. Here and in Figs. 2 and 3, amplitude of AP not shown fully.

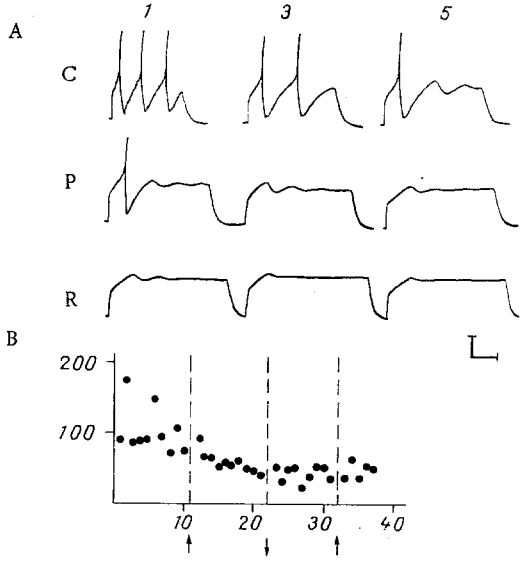

Fig. 2. Diminution of response of neurons to intracellular stimulation. A) Legend as to Fig. 1A. Numbers above are serial numbers of stimulus. Calibration: 10 mV, 400 msec; B) time course of value of 1/LP (ordinate, in % of mean control level). Each point represents average for 10 neurons. Remainder of legend as to Fig. 1B.

TABLE 1. Changes in Evoked Unit Activity after Application of DG-AVP

Types of neurons		No. of neurons	Measurements of 1/LP after DG-AVP, %	No. of neurons in which were recorded				
				increase in number of AP	decrease in number of AP	no change in number of AP	increase in amplitude of pacemaker potential	decrease in amplitude of pacemaker potential
Neurons responding by increase in excitability	LPa3	10	127—439	5	5	—	7	1
	RPa3	4	127—282	2	1	1	2	—
	LPa2	3	102—840	2	1	—	—	1
Neurons responding by decline of excitability	LPa3	3	0—79	—	3	—	—	3
	RPa3	5	0—86	—	4	1	—	1
	LPa2	2	95—96	—	1	1	—	1

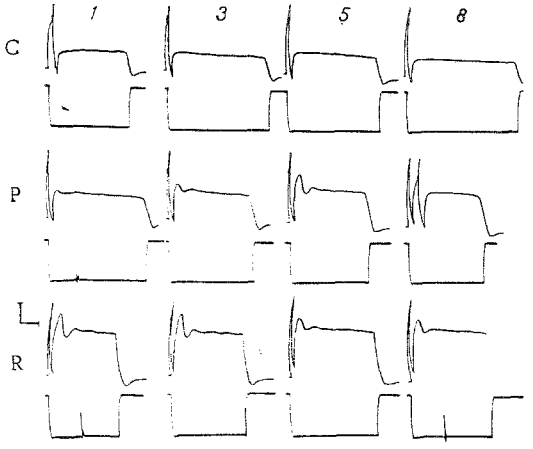

Fig. 3. Time course of spike responses and pacemaker potentials of neuron LPa3. Legend as to Fig. 1A and Fig. 2A. Calibration: 10 mV, 400 msec. Current (bottom trace) 30 nA.

was assessed on the basis of latent period (LP) of the action potential (AP) arising on passage of an above-threshold depolarizing current through the neuron. The depolarizing current was passed either through the recording electrode, using a bridge circuit, or through a second independent electrode, inserted into the same cell. The current was measured in the circuit of the reference electrode. The experiment consisted of several series: The first, control series was followed by alternation of series with perfusion with DG-AVP and series in which the compound was rinsed out. The duration of each series was 20-30 min. Intracellular stimulation was applied with an interval of 2-4 min. DG-AVP (1 μM) was added to the perfusion medium, consisting of Ringer's physiological saline for cold-blooded animals. The perfusion system allowed for complete change of the medium in the experimental chamber in about 1 min. The action of the substance was assessed by comparing values of 1/LP during perfusion with physiological saline, during application of DG-AVP, and during rinsing to remove the preparation. The significance of any changes obtained was calculated by Student's test.

EXPERIMENTAL RESULTS

Activity of 27 neurons was recorded. Depending on the results of the control series these neurons could be divided into two groups (Table 1). In one group (10 cells) diminution of the response toward the end of the control series, expressed as a decrease in the number of AP (sometimes their disappearance) in response to intracellular stimulation and a reduction in amplitude of the pacemaker potential, was clearly visible. In the other group (17 cells) either changes in reactivity were absent or there was actually a small increase in the response toward the end of the control series. Additional DG-AVP to the medium in micromolar concentrations had different effects on neurons of these groups. If the response in the control series was reduced, excitability was depressed as shown by the value of 1/LP after addition of DG-AVP, and in the other case, excitability was increased. Examples of

such responses are shown in Fig. 1A (enhancement) and Fig. 2A (diminution). The time course of the increase in excitability differed significantly from the time course of the opposite process. The time course of the values of 1/LP as a percentage of the mean control level, averaged for 17 neurons responding by increased excitability to application of DG-AVP, is illustrated in Fig. 1B. In 13 of them there was a significant ($P < 0.01$) increase in 1/LP by 1.2-8.4 times compared with the control, on the very first addition of DG-AVP to the medium. On rinsing out the preparation with physiological saline excitability declined, but not to the original level, and it increased again on addition of further DG-AVP (Fig. 1B). In some experiments this repeated addition caused a more rapid and stronger effect than in the first series. Rerinsing in this case was less effective. In one case prolonged alternation of the series for 5 h led to a permanent increase of excitability (Fig. 1C), which was not abolished by rinsing. Incidentally, during the action of DG-AVP the time course of the increase in excitability differed in different experiments. In most cases the action did not achieve its peak effect until after 10-15 min, and sometimes actually during rinsing to remove the preparation. In some cases a small increase in LP was even observed during the first minutes of action of DG-AVP. This could explain the fact that changes were not significant in four of the 17 experiments. Differences between the control series and the end of the next series in which DG-AVP was given, and the beginning of the rinsing series, likewise were significant for these neurons and indicated that the increase in excitability took place in these neurons also.

As already stated, experiments on 10 of the 27 neurons were segregated into a separate group. Excitability of these neurons, according to values of 1/LP, decreased gradually throughout the period of recording (Fig. 2B); application of DG-AVP, moreover, had no appreciable effect on the time course of this process. The increase in LP was accompanied by a decrease in the number of AP and a reduction in the amplitude of the pacemaker potentials in response to intracellular stimulation. Interruption of stimulation (20 min) led to restoration of the response. This decline in the response was evidently due to habituation of the neuron to repetitive stimulation [4]. Figure 2A shows clearly that this process had already begun in the control series. In all 10 neurons of this group processes of endoneuronal habituation coincided with an increase in LP in the course of stimulation.

Most neurons in which a significant increase in excitability was recorded responded by activation of the pacemaker mechanism, as shown by an increase in amplitude of the regular oscillations, and it led ultimately to an increase in the number of AP to a stimulus of fixed magnitude (Fig. 3). This effect was suppressed by rinsing the preparation and potentiated on further application of DG-AVP. A reduction in the parameters mentioned above was observed in only three neurons of this group.

The action of DG-AVP on spontaneously inactive command neurons on *Helix lucorum* may thus lead, under certain conditions, to an increase in their excitability. The ongoing process of endoneuronal habituation evidently masks the "excitatory" action of DG-AVP and it may perhaps even be potentiated during application of DG-AVP. The change in excitability of the neurons was probably due to the effect of DG-AVP on the pacemarker mechanism.

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