

ROLE OF PLATELET FUNCTION AND ANTIAGGREGATING ACTIVITY  
OF BLOOD VESSEL WALLS IN REGULATION OF THE STATE  
OF BLOOD AGGREGATION

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Much attention is currently being paid to the study of the role of platelet and vascular wall function in blood clotting processes. Interaction between platelets and the vessel wall is known to be determined partly by the balance between the proaggregating agents of platelets (thromboxane  $A_2$ ) and antiaggregating compounds synthesized by the vessel wall ( $PGI_2$ ). Prostacycline synthetase activity of arterial walls has been shown to be higher than that of veins [2, 4]. Having regard to the concept of the difference between hemostatic potentials in different parts of the blood stream [1], considerable interest must be aroused by a combined investigation of the primary, blood vessel-platelet component of the hemostasis system in the regulation of the state of blood aggregation.

The aim of this investigation was to study platelet aggregating activity in arterial and venous blood and also the antiaggregating properties of arterial and venous walls in different parts of the circulatory system in dogs.

#### EXPERIMENTAL METHODS

Blood from the aorta and posterior vena cava was studied from the comparative aspect in experiments on 15 mongrel dogs of both sexes, weighing 8-12 kg and anesthetized with pentobarbital (30 mg/kg body weight). Blood was taken by means of plastic catheters introduced into the vessel. As blood stabilizer a 3.8% solution of sodium citrate in the ratio of 1:9 was used. Platelet-rich plasma was obtained by centrifugation of the blood at 800-1000 rpm for 3-5 min. Platelets were counted by Nikolaev's method and platelet aggregation was studied by Born's method on an Elvi-840 aggregometer, and recorded on a Logos-176 Omniscrite Recorder automatic writer. A solution of ADP (from Fluka) in a final concentration of  $10^{-3}$  M was used as aggregating agent. The aggregating properties of the platelets were assessed in relation to two parameters: degree of aggregation (in %) and velocity of aggregation, measured as the tangent of the angle of slope of the aggregation curve. The antiaggregating action of the vessel wall was determined by the method in [3]. A sample of vessel wall weighing 5 mg was incubated for 5 min at room temperature in 0.5 ml of platelet-rich plasma, obtained from

TABLE 1. Comparison of Antiaggregating Activity of Blood Vessel Walls from Different Parts of the Vascular System ( $M \pm m$ )

| Vessels             | Antiaggregating activity, % | P                                       |
|---------------------|-----------------------------|---|
| Carotid artery      | $15 \pm 2.6$                | —                                       |
| Aorta               | $21 \pm 1.5$                | $>0.05$                                 |
| Femoral artery      | $32 \pm 5.6$                | $<0.02$ ; $>0.05$                       |
| Jugular vein        | $61 \pm 4.4$                | $<0.001$ ; $<0.001$ ; $<0.001$          |
| Posterior vena cava | $71 \pm 7.0$                | $<0.001$ ; $<0.001$ ; $<0.001$ ; $>0.2$ |

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TABLE 2. Antiaggregating Action of Vessel Walls from Different Regions on Platelets from Arterial and Venous Blood ( $M \pm m$ )

| Vessels             | Antiaggregating activity, % |                     | P    |
|---------------------|-----------------------------|---------------------|------|
|                     | platelets from blood from   |                     |      |
|                     | aorta                       | posterior vena cava |      |
| Carotid artery      | 16±5,4                      | 14±3,0              | >0,2 |
| Aorta               | 23±2,9                      | 20±1,6              | >0,5 |
| Femoral artery      | 31±7,4                      | 32±10,5             | >0,5 |
| Jugular vein        | 64±5,8                      | 58±7,2              | >0,5 |
| Posterior vena cava | 70±5,9                      | 76±12,5             | >0,5 |

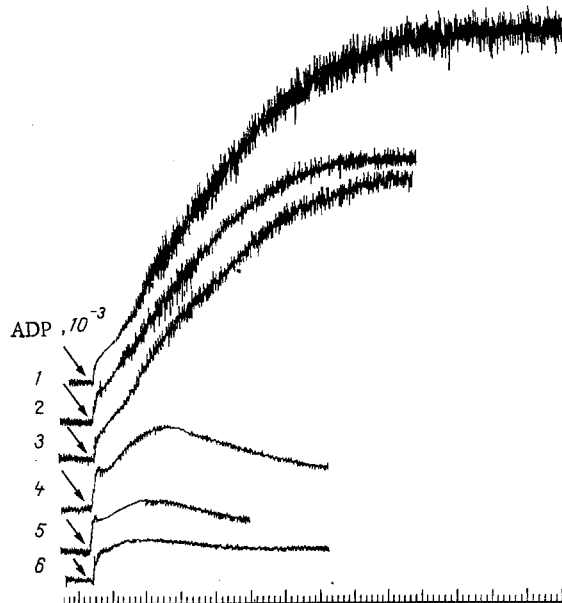


Fig. 1. Aggregation of platelets from arterial blood in original state (1) and after incubation with wall of posterior vena cava (2), jugular vein (3), femoral artery (4), aorta (5), and carotid artery (6).

arterial (aorta) or venous (posterior vena cava) parts of the circulation, and the aggregation process was subsequently recorded and expressed as a percentage of the control. Initial aggregation in the corresponding plasma was taken as the control. Antiaggregating activity of vessels of the arterial (aorta, carotid and femoral arteris) and venous (posterior vena cava and jugular vein) system. The results were subjected to statistical analysis and the significance of differences estimated by Student's test.

#### EXPERIMENTAL RESULTS

The study of the aggregating activity of the platelets revealed a significant difference between aggregation values in arterial and venous blood: Aggregating activity and the velocity of aggregation of platelets from the aorta were higher than those of platelets from a vein ( $69 \pm 4\%$ ,  $1.1 \pm 0.3$  and  $42 \pm 6\%$ ,  $0.7 \pm 0.2$ , respectively). The number of platelets in the arterial and venous blood was approximately the same, although in some experiments there was a tendency for venous blood to contain more cells:  $(170 \pm 18) \times 10^9$  cells/liter in the aorta and  $(173 \pm 15) \times 10^9$  cells/liter in the vein.

The results of investigation of the antiaggregating action of the vessel walls are given in Table 1 and Fig. 1. As will be clear from Table 1, vessel walls of the arterial

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 in 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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