was observed, but the degree of the increase was less marked than in the first group of patients (Fig. 1B: f, h).

The more rapid and complete recovery of combined electrical activity of the anterior abdominal wall muscles after herniotomy accompanied by autoalloplastic repair with knitted Lavsan gauze fabric thus indicates that the anterior abdominal wall muscles recover their functional properties and that the allograft and the capsule forming around it have no atrophic effect on the muscle; these findings, in turn, indicate that allografting of the anterior abdominal wall is a physiologically correct and adequate procedure. Defects of the anterior abdominal wall in patients undergoing autoalloplasty, incidentally, were much greater than those in patients treated by autoplasty, but the EMG indices were nevertheless restored more completely after the operation in the patients of the first group.

It can be concluded from this investigation that autoalloplasty of the anterior abdominal wall has definite advantages in patients with giant ventral hernias over autoplasty, when the sutured muscle fibers are in a functionally less advantageous state.

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MECHANISM OF THE ANTIEXUDATIVE ACTION OF HEPARIN

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In experiments on rats inflammation of the skin was induced by application of xylene. Exogenous and endogenous hyperheparinemia was shown not to affect the dynamics of the microcirculatory changes in the zone of inflammation in the initial stage of its development and vascular permeability was significantly reduced. It is suggested that a decrease in vascular permeability plays the main role in the mechanism of the antiexudative action of heparin.

KEY WORDS: heparin; capillary permeability; microcirculation.

One of the many biological effects of heparin is its antiexudative action [7, 8]. The leading factor in the mechanism of exudation, of course, is increased capillary permeability. The effect of heparin on vascular permeability in inflammation has been inadequately studied and data in the literature on this matter are contradictory [1, 2, 6].

The object of this investigation was to study the effect of exogenous and endogenous hyperheparinemia on vascular permeability and on the dynamics of changes in the microcirculation in an inflammatory focus.

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TABLE 1. Concentration of T-1824 in Skin (in $\mu g/mm^2 \times 10^3$) after Application of Xylene in Control Rats and Rats with Hyperheparinemia

Group of number of animals	Concentration of T-1824 at different times after application of xylene			
	5 min	10 min	15 min	20 min
Control (7)	4,5±0,58	5,5±0,93	4,7±0,64	13,9±1,73
Experimental 1 – exogenous hyperheparinemia (9)	4,6±0,79	3,8±0,56	3,6±0,25	6,7±0,7 †
Experiment 2 – endogenous hyperheparinemia (8)	3,7±0,18	5,1±0,56	5,5±0,93	8,9±0,53*

* $P<0,05$.
† $P<0,01$.

EXPERIMENTAL METHOD

Experiments were carried out on rats. Inflammation of the skin was induced by application of xylene by Wilhelm's method [11]. Vascular permeability was assessed from the outflow of proteins labeled with T-1824 [11] and uranin (sodium fluorescein). Uranin was injected intravenously as a 0.05% solution (500 µg/100 g body weight). The appearance of luminescence in the inflammatory focus and its dynamics were recorded by means of a reflected light microscope (the "Diagnoskop" manufactured at the Krasnogvardeets factory), modified by the writers; intensity was determined from the density of the negatives. The dyes were injected 5 min before application of xylene. Exogenous hyperheparinemia was produced by intravenous injection of heparin (from Richter) in a dose of 300 units/kg body weight 10 min before application of xylene. Endogenous hyperheparinemia was induced as follows. Protamine sulfate (from Roche) was injected intravenously in a dose of 10 mg/kg, and 5 min later the heparin concentration in the blood [10] was sharply reduced (1.2 ± 0.64 unit/ml compared with 6.0 ± 0.90 units/ml in the control; $P < 0.001$), but starting from the 10th minute hyperheparinemia was observed (9.5 ± 0.39 units/ml).

Consequently, in the experiments with inflammation, in order to create endogenous hyperheparinemia, the protamine sulfate was injected 10 min before application of xylene.

The microcirculation in the inflammatory focus was studied with the MLK-1 luminescence contact microscope (LOMO) in polarized light (magnification 80).

EXPERIMENTAL RESULTS

The maximal exudation of the dye T-1824 into the skin was observed in the control rats 20 min after application of xylene (Table 1). In hyperheparinemia, both endogenous and exogenous, the degree of disturbance of vascular permeability in the inflammatory focus (20 min) was significantly less than in the control group.

In the experiments in which uranin was injected, the earliest disturbances of vascular permeability in the inflammatory focus could be recorded. As Table 2 shows, luminescence in the zone of inflammation appeared in the control rats 9 sec after application of xylene and reached a maximum after 68 sec. In endogenous and exogenous hyperheparinemia, lumines-

TABLE 2. Time of Appearance of Maximal Luminescence in Inflammatory Focus in Control Rats and Rats with Hyperheparinemia

Group and number of animals	Time of appearance of luminescence, sec	Time of maximal luminescence, sec
Control (8)	9,0±0,6	68,0±0,41
Experiment 1 – exogenous hyperheparinemia (6)	29,0±1,7	111,0±4,7*
Experiment 2 – endogenous hyperheparinemia (5)	58,0±3,36	252,0±44,9*

* $P<0,001$.

cence appeared much later in the zone of inflammation, and the development of maximal luminescence also was shifted in time (Table 2).

The quantity of dye determined in the zone of inflammation can depend not only on vascular permeability, but also on the volume of blood in the vessels and the state of the lymphatic drainage. The state of the microcirculation was therefore studied in the zone of inflammation by recording the dynamics of the number of functioning capillaries (in 1 mm^2) and the time of appearance of the first signs of stasis, namely swelling and stopping of the blood flow in the capillaries. In the control rats the number of functioning capillaries was increased only a few seconds after application of xylene, and the changes were statistically significant after 5 min (control 35.0 ± 0.59, experiment 67.0 ± 5.95; $P < 0.01$).

During the next 25 min the number of functioning capillaries remained virtually unchanged. The first signs of stasis were observed on average after 26 min. Endogenous and exogenous hyperheparinemia did not affect the dynamics of the indices of the microcirculation in the inflammatory focus. Consequently, the decrease in the exudation of dye into the inflammatory focus observed in rats with hyperheparinemia was due primarily to the effect of heparin on vascular permeability.

There is information in the literature on inactivation of histamine under the influence of heparin [9]. The present writers also showed previously [1, 3-5] that heparin reduces the effect of histamine and serotonin on vascular permeability. These mediators play a role mainly in the early phase of inflammation and, for that reason, the decrease in capillary permeability observed in rats with hyperheparinemia during xylene inflammation of the skin can be explained by inactivation of the mediators of inflammation.

The decrease in vascular permeability thus plays the principal role in the mechanism of the antiexudative action of heparin.

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