The results are evidence that burn trauma is accompanied by disturbances of the microcirculation, hemoconcentration, and increased viscosity of the blood, which are especially marked in vessels with low pressure. Changes in the mesenteric microcirculation coincided with changes in the dynamic viscosity of the blood and hematocrit index determined in vitro. These disturbances were more marked after extensive and deep burns with a fatal issue than after moderately severe burns, the consequences of which were less serious. This investigation confirms the important role of changes in the blood rheology and disturbances of the microcirculation in the early period of burns.

LITERATURE CITED

1. V. I. Udovichenko, Patol. Fiziol., No. 1, 73 (1978).
2. Yu. M. Shtykhno and A. M. Chernukh, Patol. Fiziol., No. 4, 6 (1974).
3. S. E. Bergentz, L. E. Gelin, S. M. Rudenstam, et al., Acta Chir. Scand., 126, 289 (1963).
4. W. G. Bigelow, R. D. Heimbecker, and D. G. Harrison, Arch. Surg., 59, 667 (1949).
5. A. M. Ehrly, Med. Welt (Stuttgart), 29-30, 1167 (1971).
6. L. E. Gelin, Acta Chir. Scand., 122, 287 (1961).
7. C. Jelenko, W. D. Jennings, W. R. O'Kelley, et al., Arch. Surg., 106, 317 (1973).
8. R. E. Schoen, C. H. Wells, and S. N. Kolmen, J. Trauma, 11, 619 (1971).

PERMEABILITY OF TISSUE–BLOOD BARRIERS OF THE SMALL INTESTINE DURING PERFUSION WITH CERTAIN PRESERVATIVE SOLUTIONS

V. P. Kulik, S. N. Genyk,
V. M. Krysa, R. V. Goncharenko,
and V. M. Kostyuk

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The effect of some preservative solutions on changes in permeability of tissue–blood barriers of isolated loops of small intestine was studied in laboratory albino rats during perfusion of their vessels with 0.85% sodium chloride solution, with Ringer–Locke, Hanks', and Collins-2 solutions, and with the Soviet preparations Gemodez and Aminopeptid. The volume of fluid flowing from the vessels, penetration of perfusion fluid into the lumen of the intestine, and its elimination through the serous membrane were determined. It was concluded that the least disturbance to the tissue–blood barriers of the small intestine is observed during perfusion of its vessels with Collins-2 solution. This method is recommended as a test for comparing the properties of preservative solutions.

KEY WORDS: small intestine; tissue–blood barriers; perfusion; preservative solutions.

Of the many methods of keeping organs and tissues viable in vitro the most promising at this stage seems to be their preservation in cold liquid media. In this connection many solutions balanced with the extracellular or intracellular fluids and containing electrolytes, carbohydrates, amino acids, and antibiotics, have been studied [1, 6, 8]. However, no general criterion for comparison of these solutions could be found in the accessible literature.

It has been shown [2, 3, 7, 9, 10] that during perfusion of the vessels of the small intestine with various solutions the latter penetrate through the vascular wall and tissue–blood barriers into the lumen of the intestine and emerge on its serous membrane. The writers have used this phenomenon to compare the properties of preservative solutions used in experimental and clinical transplantology.

Department of Normal Anatomy, Patrice Lumumba Peoples' Friendship University, Moscow. Department of Clinical Surgery, Ivano-Frankovsk Medical Institute.

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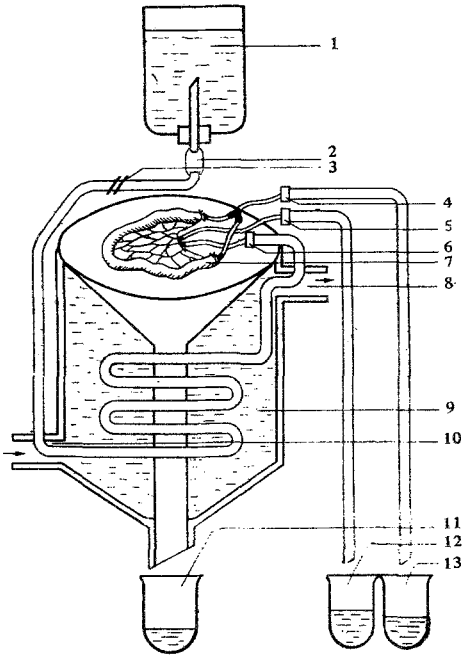

Fig. 1. Diagram of microperfuser with heat exchanger for perfusing blood vessels of isolated loops of small intestine. 1) Reservoir with perfusion fluid; 2) dripcock; 3) regulator of rate of flow of perfusion fluid; 4) cannulas for draining perfusion fluid from intestinal lumen; 5) cannula for draining vascular component of perfusion fluid; 6) cannula conducting perfusion fluid into mesenteric artery; 7) intestine placed on surface of heat exchanger; 8) connecting tube of heat exchanger for outflow of heat carrier; 9) heat exchanger with heat carrier; 10) connecting tube for inflow of heat carrier into heat exchanger; 11) vessel for collecting extraintestinal component of perfusion fluid; 12) receiver for collecting vascular perfusion fluid; 13) receiver for collecting intestinal perfusion fluid.

It is convenient to use the small intestine because fluid passing out of the vessels does not accumulate in the tissues, as it does in the liver, kidneys, pancreas, and other parenchymatous organs, but penetrates through tissue–blood barriers and enters the lumen of the intestine [11, 13, 14]. A certain proportion of the solution escapes to the serous membrane of the intestine and into its lymphatic system. This fraction was conventionally called the extraintestinal component of the perfusion fluid.

EXPERIMENTAL METHOD

Experiments were carried out on 37 laboratory albino rats weighing 200-250 g. The animals were divided into seven groups with at least five rats in each group. The animals of group 1 (seven rats) were used to develop an experimental model. In the remaining six groups changes in the tissue–blood barriers were studied during perfusion of the intestine with 0.85% sodium chloride solution, with the Soviet preparations Gemodez (see below) and Aminopeptid (a product of enzymic hydrolysis of bovine blood proteins), and Ringer–Locke, Hanks', and Collins-2 solutions.

Perfusion systems of different designs [4, 5, 8, 12] are used to perfuse the small intestine. In the present investigation the writers used a microperfuser consisting of a reservoir for the test fluid, a system of tubes, and a regulator of the perfusion fluid drip system and heat exchanger, on top of which the isolated loop of intestine for perfusion was placed (Fig. 1). Fluid escaping through the serous membrane of the intestine and lymphatic ducts was collected through the central outlet of the heat exchanger. The vessel containing perfusion fluid was placed 150 cm above the level of the heat exchanger, equivalent to a pressure of 110 mm Hg. The experiment continued for 1.5-2 h. The rate of flow of the solution was kept stable at 5 ml/min.

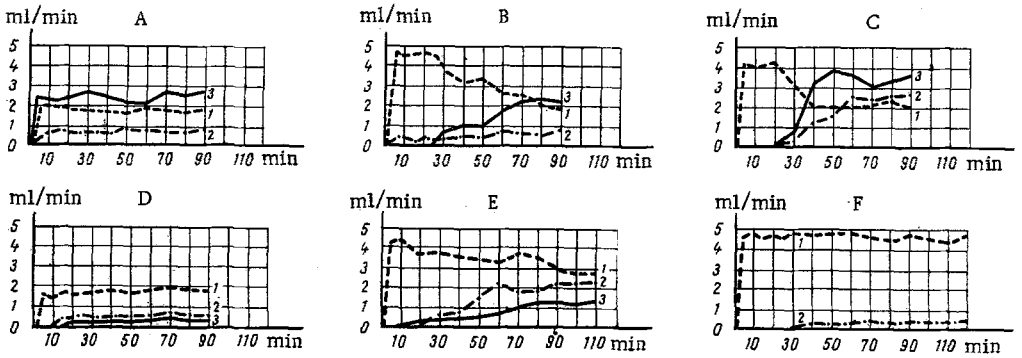

Fig. 2. Dynamics of permeability of tissue–blood barriers of small intestine to various perfusion solutions. A) 0.85% sodium chloride solution; B) Ringer–Locke solution; C) Gemodez; D) Aminopeptid; E) Hanks' solution; F) Collins-2 solution. 1) Vascular, 2) intestinal, and 3) extraintestinal components of perfusion fluid.

Under intramuscular thiopental anesthesia laparotomy was performed, the small intestine mobilized, and the intestinal contents removed by flushing out the lumen with 0.85% sodium chloride solution (20 ml). To prevent thrombus formation in the intestinal vessels, 500 units heparin was injected into the lungs. The cranial mesenteric artery and vein were isolated and cannulated. The intestine was then placed on the top surface of the heat exchanger and perfusion carried out with the various test solutions at 20°C. The duration of ischemia from the time of ligation of the mesenteric vessels until the beginning of perfusion did not exceed 10 min.

EXPERIMENTAL RESULTS

During perfusion of the vessels of the small intestine with 0.85% sodium chloride solution large quantities of the perfusion fluid were seen to penetrate into the lumen of the intestine and also to escape through the serous membrane. The surface of the intestine remained moist throughout the experiment. Outflow of the solutions through the vessels, the lumen of the intestine, and its outer surface reached equilibrium 10 min after the beginning of the experiment and was maintained throughout the rest of its course (Fig. 2A).

During perfusion with Ringer–Locke solution during the first 25 min only a very small volume of perfusion fluid entered the lumen of the intestine. Not until 30 min after the beginning of perfusion was an increased flow of perfusion fluid observed into the lumen of the intestine, and fluid also penetrated through the serous membrane to appear on the surface, while at the same time there was a decrease in the outflow of perfusion fluid from the vessels (Fig. 2B).

Gemodez, among the components of which are low-molecular-weight dextran and electrolytes balanced with the extracellular fluid, did not penetrate for 20 min into the lumen of the intestine. The outer surface of the intestine was dry, and to prevent further drying it was necessary to moisten it constantly. Only after 20 min did Gemodez begin to penetrate into the lumen of the intestine and to appear on its serous membrane. The outflow of perfusion fluid increased until 60 min, when dynamic equilibrium was established between the vascular, intestinal, and extraintestinal components (Fig. 2C).

Aminopeptid penetrated in minimal quantities into the lumen of the intestine over a period of 1.5 h. The outer surface of the intestine remained dry until the end of the experiment. Only in the last 20 min was a very small increase in the outflow of perfusion fluid through the lumen of the intestine observed. Another special feature must also be mentioned. At the maximal rate of supply of perfusion fluid the inflow and corresponding outflow volumes of vascular perfusion fluid were only half of those observed with noncolloidal solutions (Fig. 2D).

Curves of similar character were found in the case of perfusion with Ringer–Locke and Hanks' solutions. However, by contrast with Ringer–Locke solutions, less of the Hanks' solution penetrated through the outer surface and more was eliminated into the lumen of the intestine. After 70 min dynamic equilibrium began to be established between the three components and was maintained until the end of the experiment (Fig. 2E).

Least disturbance of permeability of the tissue-blood barriers was found with perfusion of the intestinal vessels with Collins-2 solution. A uniform and stable outflow of perfusion fluid through the vessels was ob-

served throughout the 2 h of the experiment. The solution did not begin to enter the lumen of the intestine until 30 min after the beginning of the experiment. A considerable increase in the outflow of perfusion fluid into the lumen of the intestine was not observed before the end of the experiment. An extraintestinal component of the perfusion fluid likewise did not appear (Fig. 2F).

Comparison of the results indicates a general rule which can be applied to the various groups of preservative solutions. The least satisfactory preservative solution was 0.85% sodium chloride solution. Within a few minutes it caused disturbances of the tissue–blood barriers and penetrated in large quantities into the lumen of the intestine and appeared on its serous membrane [1, 2].

Solutions balanced with the extracellular fluid (Ringer–Locke and Hanks' solutions, Gemodez) were retained for the first 15-20 min in the blood stream, but then penetrated through the tissue–blood barriers of the small intestine; increased permeability was observed until 60-70 min after the beginning of perfusion. Later equilibrium was established between the outflowing components of the perfusion fluid.

Aminopeptid, which has a high specific gravity, high viscosity, and high osmotic pressure, despite identical parameters of perfusion with the other solutions tested, had only half the inflow and outflow perfusion volumes. These same properties can probably account for its minimal ability to penetrate through tissue–blood barriers.

The least disturbance of permeability of the tissue–blood barriers of the isolated loop of small intestine was found when Collins-2 solution, balanced with intracellular tissue fluid, was used. This can be attributed to prolongation of the energy metabolism of the isolated loop of intestine as a result of a decrease in the rate of breakdown of the energy resources in the cellular energy depots and a decrease in the energy expenditure of the cell on work required to maintain ionic, chiefly Na^+, gradients [6]. Preservation of the cell energy resources at the same time reduces disturbance of the tissue–blood barriers, and it is this, perhaps, which prevents penetration of perfusion fluid from the blood stream into the intestinal lumen and on to the serous membrane.

LITERATURE CITED

1. A. A. Vishnevskii, V. F. Portnoi, and L. N. Cherkashchenko, Vestn. Akad. Med. Nauk SSSR, No. 8, 16 (1973).
2. M. A. Meshcheryakova and T. Z. Efimova, in: Transplantation of Organs and Tissues and Problems in Plastic Surgery [in Russian], Moscow (1973), pp. 35-36.
3. O. B. Murashova, in: Transplantation of Organs and Tissues and Problems in Plastic Surgery [in Russian], Moscow (1973), pp. 37-38.
4. A. I. Parfenov and A. A. Zhgun, Med. Tekhnika, No. 1, 35 (1976).
5. A. A. Pisarevskii and N. A. Super, Med. Tekhnika, No. 1, 10 (1975).
6. V. I. Shumakov, E. Sh. Shtengol'd, and N. A. Onishchenko, Preservation of Organs [in Russian], Moscow (1975).
7. C. Chaussy, "Studien über Motilität und Resorptio am isolierten hämoperfundierten Darmsegment," Dissertation, Munich (1972).
8. J. Kachelhoffer, C. Mendel, P. Wong, et al., C. R. Soc. Biol., 163, 2449 (1969).
9. D. S. Parsons and G. Powis, J. Physiol. (London), 217, 641 (1971).
10. L. Johnson, Acta Chir. Scand., 139, 184 (1973).
11. L. Johnson, Acta Chir. Scand., 139, 72 (1973).
12. L. Johnson, Acta Chir. Scand., 138, 834 (1972).
13. H. G. Windmueller, A. E. Spaeth, and C. E. Ganote, Fed. Proc., 36, 177 (1977).