

Revascularisation of Arterial Prosthesis and Autovenotransplant

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ABSTRACT. After implantation of prosthesis in the artery, incapsulation and formation of neointima, there develops “vasa vasorum”, which penetrates into the prosthesis from the outside and from the inner side – from the prosthesis lumen. Internal initiated vessels, contacting with the external, anastomose with each other. In the prosthesis internal lining original capillary network of hexahedral form is produced. This geometric form depends on decussation of longitudinal and transversal filaments of prosthesis fibers.

Practically the importance of neointima vascularization lies in the fragility of capillaries, resulting in small hemorrhages formation. Neointimal capillary topography, their exclusive location to prosthesis lumen, these small hemorrhage foci can simulate a coagulation chain reaction in the prosthesis.

After autovenoplasty in the artery and after restoration of blood flow the autovenotransplant sharply extends under arterial pressure, and there was no case of rupture or aneurism development in the transplanted veins. In some weeks the transplanted vein changes sharply, the wall thickenes and, which is of great importance, vascularization of vein changes and becomes similar to the vascularization of the artery, into which the vein has been transplanted. © 2007 Bull. Georg. Natl. Acad. Sci.

Key words: blood vessel alloplasty, autovenoplasty.

On the basis of large experimental material (dogs) the animal organism's response to vascular plastic reconstruction of arteries and veins has been studied.

Besides various methods of morphological investigation the injection of blood vessels with Indian ink-gelatine mass according to M. Komakhidze's method has been used. The latter added significant originality to the studies carried out.

The organism's response to encapsulation-implantation of different kinds of substitutes: autoveins, hard and soft-porous artificial prosthesis – has been examined.

Testing of hard artificial substitutes of arteries (polyvinyl alcohol and polychlorvinyl tubes) showed that they were suitable only for short-term blood vessel substitution, because of their rapid thrombosing. It is caused by the absence of porosity in the tube walls and their internal surface smoothness, on which fibrin deposit is not retained, as well as significant activation of the blood

coagulating system. After hard tubes occlusion and thus their exclusion from the blood circulation, the parameters of blood coagulating system return to the initial level. The problem was solved by the usage of synthetic fibres: nylon, dacron, teflon, terylene, ivalon, orlon, vinon, PTFE-Gore-Teks, kapron, lavsan (Soviet manufacture). A significant step was the usage of goffered artificial tubes, offered by Edwards and Trapp (1955), which facilitated selection of the necessary length of artificial vessels, but not all have taken a positive view of this improvement (Weibel, Szilagy, 1959; Chvapil, Krajihnek, 1966).

Searches and efforts to improve artificial substitutes of an artery continued, each attempt being pronounced as a stage of development. Watanuki (1978) invented felt-like dacron, the firm Meadox (France) created high-perforated, knitted micro-vellur artificial vessels (Perini et al. 1995).

However, long-term testing nevertheless gave preference to factory manufactured goffered dacron porous tubes.

Testing of various soft-pored artery substitutes (capron, lavsan, dacron) has shown that the process of their incorporation principally follows one and the same scheme. This process is subdivided into three consecutive periods or stages: the first one - formation of fibrinous cover, the second - fibrin organization and development of the prosthesis connective tissue capsule, and the third - definitive inner lining - prosthesis neointima formation. All authors studying artificial arteries mark these stages [1-5, 8, 9, 11-15, 19, 21].

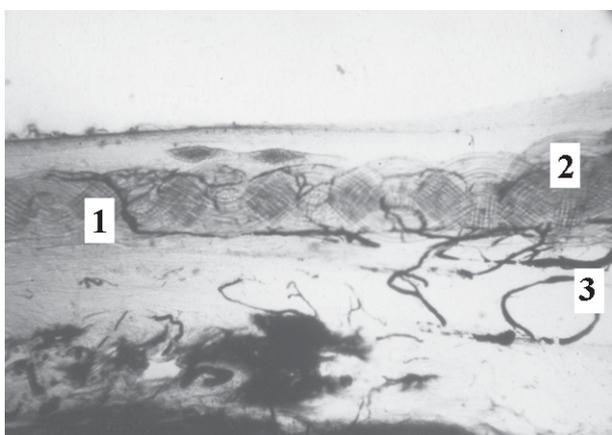


Fig. 1. Prosthetics of the dog's abdominal aorta with smooth capron tube. Duration of experiment (DE) 85 days. Longitudinal cut of a capron tube - 1, injected capillaries in the external prosthesis capsule - 2, capillaries between capron middle fibres - 3, capillaries in the internal lining of prosthesis 4 x 35.

The first stage, i.e. blood coagulation on the prosthesis wall immediately follows after blood circulation starts. In the crimped prosthesis fibrin fills the deepenings and makes the tube surface smoother. Fibrin closely attaches to the prosthesis filaments and contains blood cells. The second stage, i.e. connective tissue germination, occurs in fibrin very quickly after prosthesis inclusion; it initiates from the prosthesis ends and extends to the middle part, and simultaneously - from the surrounding tissues, developing from the outside into the internal part. The whole second stage should be considered as two periods: usually during the first fortnight the fibrinous cover organization process is carried out mainly owing to cell element germination (fibroblasts, epithelioid, plasmatic and lymphoid cells).

Later (2-3 months) the inner and upper cover of the prosthesis takes the form of dense fibrous connective tissue. Fibrous cover of the prosthesis is substituted by connective tissue in two ways: from the ends of the prosthetic vessel and on account of surrounding tissues. That is why demands to prosthesis porosity are

determined as the demands to hemostasis, and the necessity of connective tissue germination through pores.

The third stage - formation of the definitive inner lining of the prosthesis, accompanied by formation of neointima endothelial cover is shown to develop gradually. Usually the layer of endothelial cells grows from the anastomosis edges, extending to the middle part of the prosthesis, and ends with total endothelialization of the whole internal surface. This process develops with various intensity in different patients and thus the terms of its completion might be determined relatively from several months to one year. The cells lying on the growth edge are large, of irregular form, polynuclear, close to anastomosis location they do not differ from the ordinary endothelial cells, as well as the cells of completely formed neointima. Its endothelialization runs quicker from the central anastomosis edge and a bit slower - from the peripheral anastomoses.

It is rather disputable whether neointimal cover cells are true endothelial cells. But practically it is unimportant from the prosthesis full value, because these cells carry out endothelial function.

Connective tissue having developed round the prosthesis is rich in blood vessels. For the first fortnight prosthesis connective tissue capsule contains a large amount of fissures, which are filled with the contrast mass by vessel injection. Later the wall round the fissures is found to be organized, endothelialized, and the fissures reduce to vessel capillaries, precapillaries, and their walls become of typical structure corresponding to the vessel gauge.

Small blood vessels like *vasa vasorum* combined with connective tissue fibers germinate inside through prosthesis pores and make the internal lining vascularized (Fig. 1). But another internal lining vascularization source exists. These are vessels initiated from the pros-

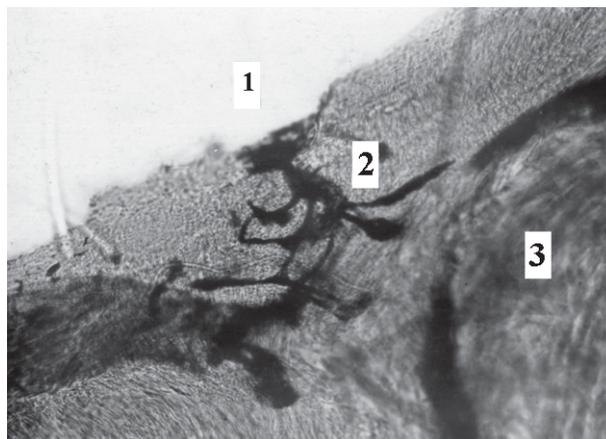


Fig. 2. Prosthetics of the dog's abdominal aorta with goffered lavsan tube. DE 17 days. Inner lining vascularization by the vessel growing from the prosthesis lumen - 1, newly organized vessels are of flask-like form - 2, prosthesis tissue - 3. Vessels injection, x 100.

thesis lumen (Fig. 2). These internal *vasa vasorum* not only provide the internal lining blood supply, but simultaneously are responsible for its endothelialization (Fig. 3).

Internally initiated vessels penetrate outside through the prosthesis pores, contacting with the external *vasa vasorum*. These and those vessels widely anastomose with each other.

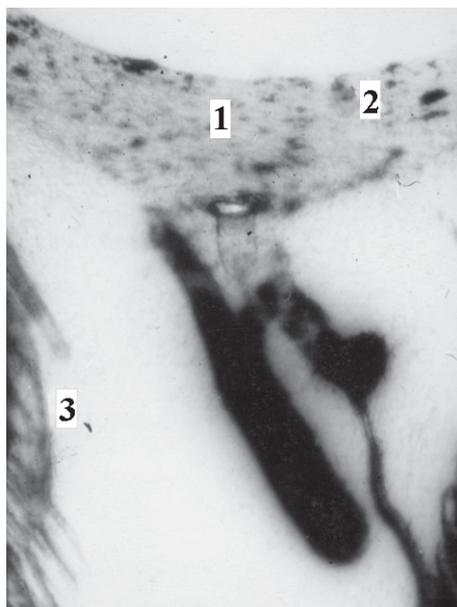


Fig. 3. Prosthetics of the dog's abdominal aorta with gofferred lavsan tube. DE 80 days. Inner lining vascularization by the vessel initiated from the prosthesis lumen - 1, neointima - 2, prosthesis tissue - 3. Vessels injection, x 100.

The blood vessels vascularizing the prosthesis internal lining make in its thickness the original capillary network of hexahedral form (Fig. 4). This geometric form of the capillary network internal lining depends on the prosthesis. The prosthesis filaments being closely interwoven form eminences at the longitudinal and transversal filaments decussation spot, and as well as

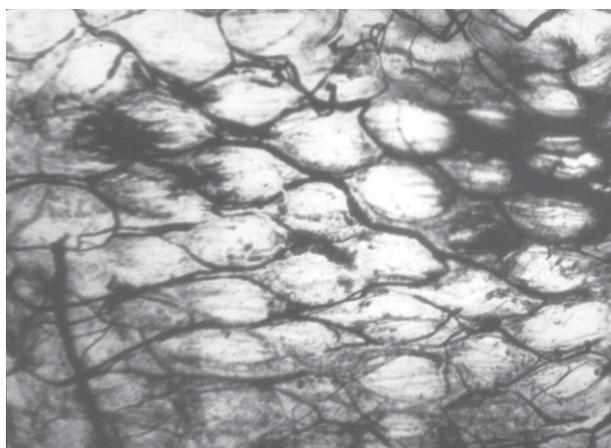


Fig. 4. Prosthetics of the dog's iliac artery with smooth capron tube. DE 201 days. Injected capillary network anseae in the inner prosthesis lining are of hexagonal form, x 50.

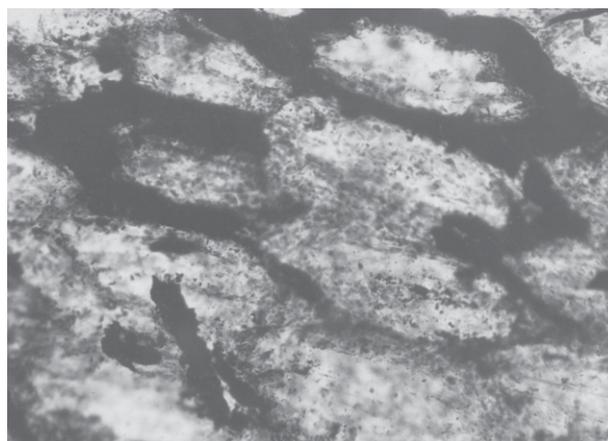


Fig. 5. Prosthetics of the dog's abdominal aorta with gofferred Dacron tube. DE 127 days. Hexagonal anseae of the capillary network in the inner prosthesis lining. In the centre at the point of injection defect the capillary endothelial lining is observed, x 100.

deepenings-grooves on the decussation edges, where the blood pressure upon them seems to be rather weak and this is the cause of vascular network peculiar form (Fig. 5).

Practical importance of neointima vascularization is obvious in the following: capillaries are the most fragile part of the vascular system and that is why they easily break, especially newly formed, which results in small hemorrhages formation. In our case, owing to neointimal capillary topography, i.e. their exclusively close location to the prosthesis lumen, which does not take place in the arterial and vein intima, these small hemorrhage foci can stimulate blood coagulation chain reaction in the prosthesis. Thus neointima vascularization appears to be one of the causes of arterial prosthesis late thrombosis.

The chance of thrombosis after alloplasty by artificial prosthesis is even greater in an artery of small calibre than an artery of greater calibre (aorta), owing to the arrangement of newly developed capillaries directly under neo-endothelium, which does not take place in normal vessels and in an artery. We decided to test

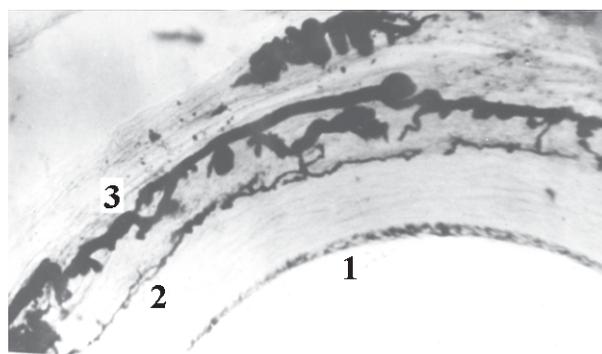


Fig. 6. Capillary network in the wall of the dog's common carotid artery. Arterial lumen - 1, perimuscular capillary network - 2, periadventitial capillary network - 3. x 35.

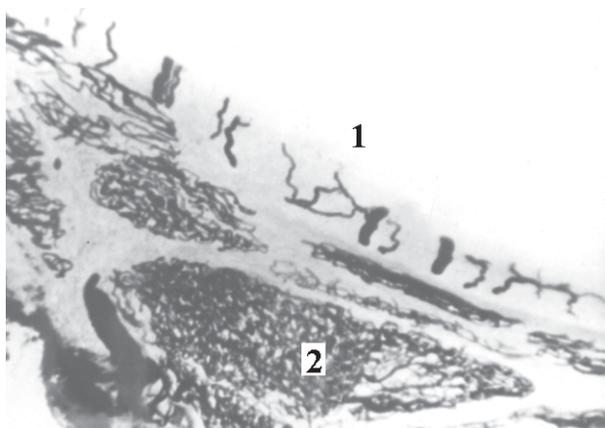


Fig. 7. Capillary network in the dog's v. saphena magna - 1, perivascular network - 2, x 35.

the possibility of replacement of an artery with own vein.

Veins proper (jugularis, saphena magna) had been transplanted into various peripheric arteries (a. a. carotis com., femoralis, renalis) and in all the cases autotransplanted vein wall thickening had been observed, and which is of great importance, preformation of its wall vascularization that became similar to vascularization of artery in which the vein had been included.

This conclusion is made due to the difference between arterial and venous blood supply. The arterial wall has two circular plexuses: perimuscular and periadventitial (Fig. 6), but the vein has a single vascular network, which is located in adventitia (Fig. 7). As soon as vein begins to carry out the arterial function and blood is pulsating in it upon arterial pressure, vascu-

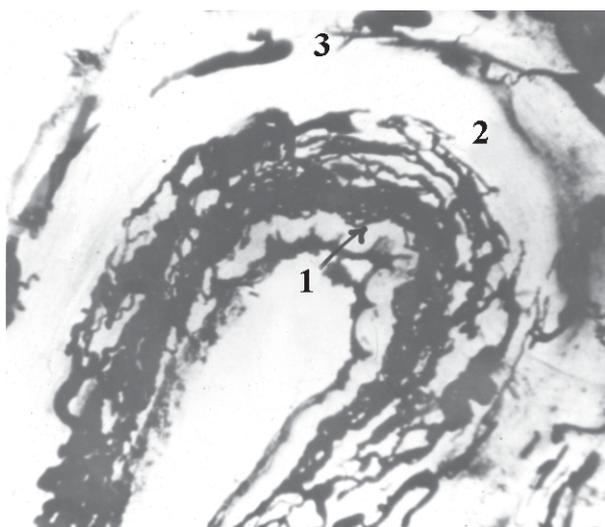


Fig. 8. Subcutaneous jugular vein implanted into the common carotid artery of the dog. DE 91 days. Capillary network in the transplant wall is sharply enlarged, no difference between periintimal - 1, perimuscular - 2 and periadventitial network - 3 gauges, x 35.

lar plexuses in its altered wall develop, which are similar to perimuscular and periadventitial plexuses of arteries (Fig. 8).

The first operations (1954) were made on the common carotid artery in which a segment (4-5 cm) was stitched in a piece of jugular or femoral vein (10 experiments). Following autvenoplasty was made on a hip. A segment of femoral arteries (3-4 cm) was replaced also by a femoral subcutaneous vein.

Each time after restoration of bloodflow, autovenotransplant sharply extended under the influence of arterial pressure and it seemed that it could burst but it never occurred; in one experiment development of aneurysms of a wall of the transplanted vein did not take place either. In some weeks the transplanted vein changed sharply, condensed and externally did not differ any more from the artery in which the stitch was.

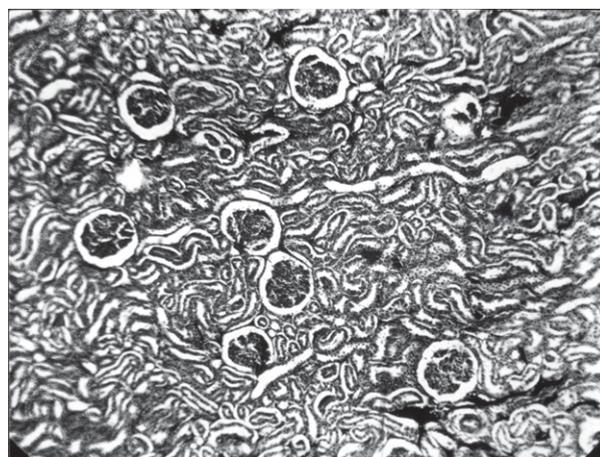


Fig. 9. Dog's right intact kidney after autovenoplast of left renal artery. (DE) 205 days. Glomerules are injected completely. Hematoxylin-eosin. x 80.

Becoming convinced of the stability of the autotransplant venous site, we decided to check the possibility of replacement of kidney arteries by a vein (1963). Pressure of blood in system kidney arteries is selectively high in comparison with other arteries of the same calibre. Renal hypertension less submits to hypotensive therapy, the reason of this pathology is often a change - narrowing, thrombosis of kidney arteries and other cases demanding surgical intervention.

Autovenoplasty of the left kidney was made to 44 dogs. In the beginning the right kidney was not touched. When we became convinced that at successfully executed operation the left kidney externally had not changed, at the second moment, some months later, the right intact kidney was removed. Dogs lived with one autovein transplanted kidney some years (2 dogs - 9 m, 1 dog - 1 y, 2 dogs - 116 m, 1 dog - 3 ys, 6 m, 1 dog - 3 ys, 11 m, 1 dog - 4 ys, 1 dog - 8 ys); we discontinued the experiment to carry out histologic research. One dog pupped, two dogs ran away from an open-air cage. At a

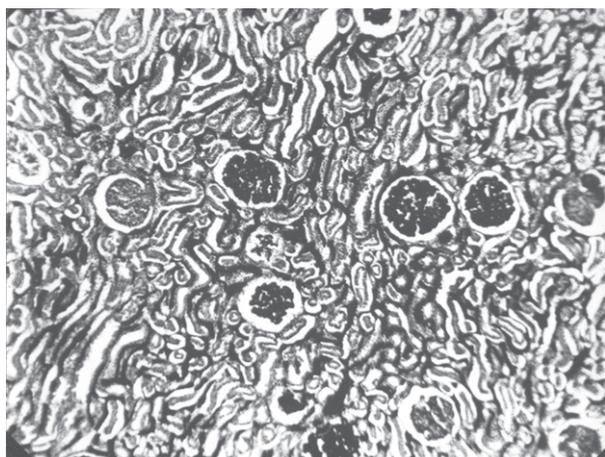


Fig. 10. Dog's left kidney after autovenoplasty of its artery. DE 205 days. Glomerules are well injected. Hematox.-eosin. x 80.

successful result the pressure of arterial blood kept within the limits of norm, nitrogen in blood 22-35%. At autopsy kidneys did not show signs of great damage of structure (Figs 9, 10), of ultrastructure (Figs 11, 12) as well as the vascular system (Figs. 13, 14, 15).

The duration of deenergizing of a kidney from blood circulation is of great value; 30 minutes are desirable,

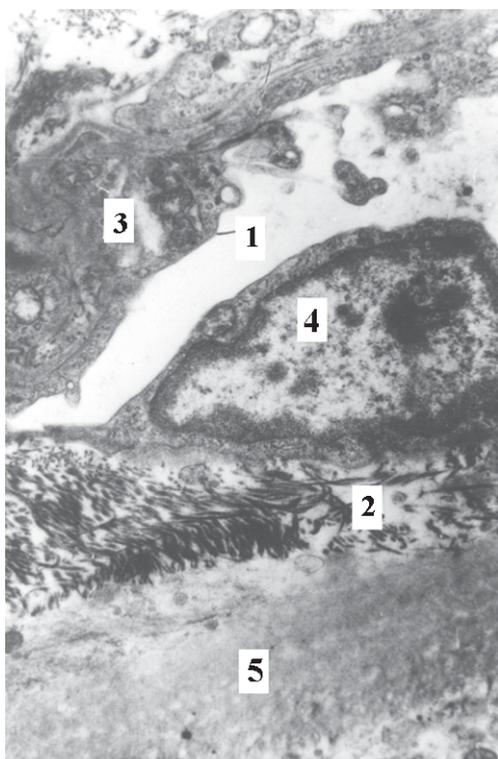


Fig. 12. Dog's left kidney after autovenoplasty of its artery. DE 2 years 9m. peritubular capillary is separated from the urinary tubules by rough collagen fascicles - 2, endotheliocyte is condensed - 3 and has no fenestra, endotheliocyte nucleus is unchanged - 4, basal membrane of the urinary tubule is significantly thickened - 5. x 7000.

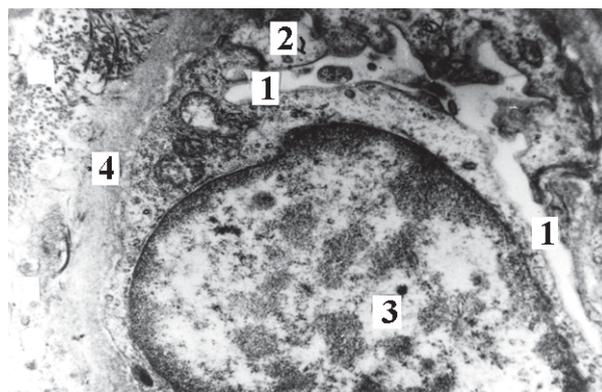


Fig. 11. Dog's left kidney after autovenoplast of its artery. DE 9 months. Peritubular capillary lumen, fissural one - 1, endotheliocyte cytoplasm is endemic - 2, nucleus - 3 protrudes into the lumen, pinocytosis is absent, basal membrane and pericapillary space contain collagenic fibrillae - 4. x 9000.

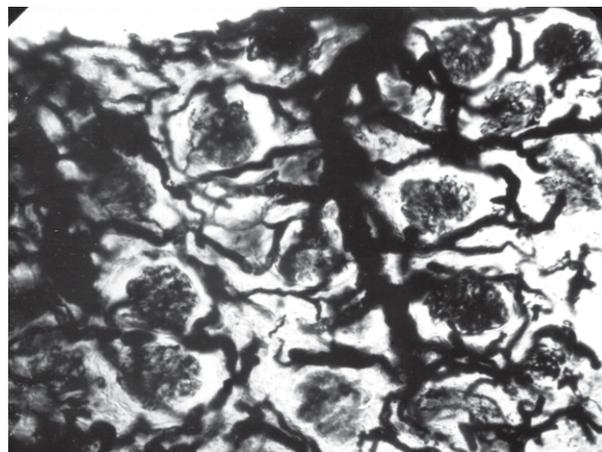


Fig. 13. Dog's left kidney after autovenoplasty of its artery. DE 200 days. All the intraorganic vessels, including glomerules, are well injected, x 120.

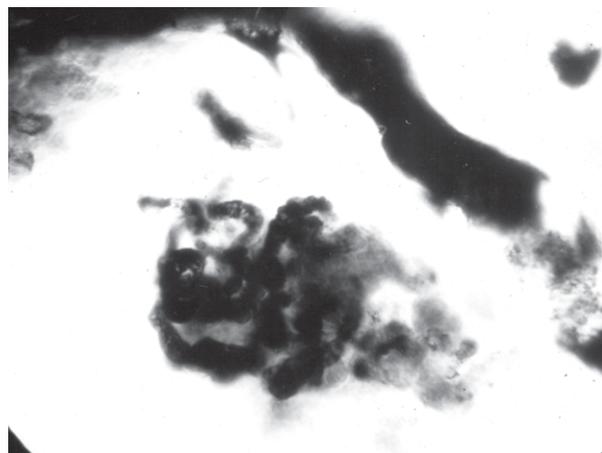


Fig. 14. Dog's left kidney after autovenoplasty of its artery. DE 200 days. Glomerular vessels are significantly extended and well injected. x 300.

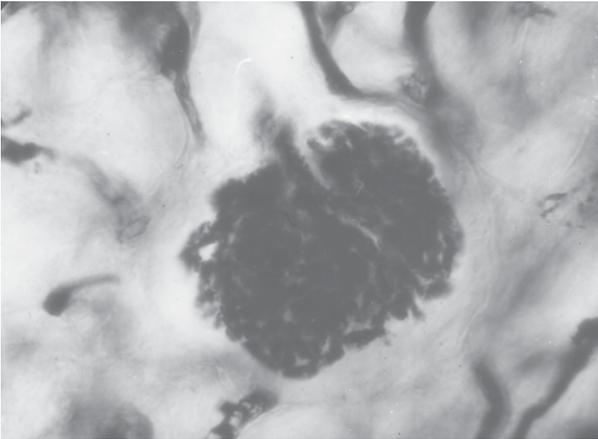


Fig. 15. Dog's right intact kidney. Glomerular vessels are well injected, all the ansae are filled with contrast substance, x 300.

not longer. The calibre of a dog's renal arteries is very small, diameter of 2-3 mm, the form of branching is also important. After operation complications in the system of intestinal vessels and the portal system, and during roentgeno-vasocinematography are frequent.

Especially obvious is the full value of autovenotransplant in the case of renal artery substitution. After successful operations the dogs endure the second kidney ablation and live for several years with a single kidney, whose artery had been replaced by the autovein. The structure and ultrastructure as well as the function of such kind of kidney are preserved.

Autovenoplasty of renal artery is necessary not only at renal hypertension, but also at transplantation of a kidney. Unfortunately, the viability of the replaced vein is not great [7, 10, 16-18, 20].

სამედიცინო მეცნიერებანი

არტერიის პროთეზის და აუტოვენის ტრანსპლანტაციის რევასკულარიზაცია

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ნაშრომში ნაჩვენებია სისხლის ძარღვების ახალგანვითარება და ჩაზრდა არტერიის ხელოვნური შემცვლელის გარშემო განვითარებულ ქსოვილებში, მათ შორის ნეონტიმაში (შიგნითა გამონაფენში), რასაც არა აქვს ადგილი არც არტერიაში და არც ვენაში. გამოთქმულია აზრი, რომ ნეონტიმაში ახლად ჩაზრდილი სისხლძარღვები შეიძლება იყოს ერთ-ერთი მიზეზი არტერიის პროთეზის გვიანი თრომბირებისა, ნეონტიმასთან – სანათურთან ახლო მდებარეობის გამო.

არტერიის საკუთარი ვენით შენაცვლების შემდეგ ვენის კედელი სწრაფად იცვლება, განიცდის „არტერალიზაციას“ და უძლებს მაღალ არტერიულ წნევას, რის გამოც ეს აუტოტრანსპლანტაცია საიმედოა და შეიძლება გაკეთდეს მსხვილ არტერიებშიც.

მონაცემები არტერიების ხელოვნური პროთეზებისა და საკუთარი ვენით არტერიის შეცვლის რევასკულარიზაციის შესახებ მეოცე საუკუნის ბოლო ათწლეულების მრავალრიცხოვან ლიტერატურაში არ არის. ამიტომ წერილს ვურთავთ მხოლოდ ჩვენი ზოგიერთი შრომის დასახელებასაც.

REFERENCES

1. *M.V. Anichkov et al.* (1963), *Vestnik khirurgii*, 2, 41-44 (Russian).
2. *V.A. Zmur et al.* (1959), *Vestnik khirurgii*, 4, 71-79 (Russian).
3. *M.I. Perel'man, Yu.A. Rabinovich* (1965), *Vestnik khirurgii*, 3, 7-11 (Russian).
4. *B.V. Petrovskii* (1963), *Vestnik khirurgii*, 3, 10-17 (Russian).
5. *Yu.G. Shaposhnikov* (1961), *Primenenie autoven, podkreplennykh perlonovymi odnosloinymi obolochkami, dlya plastiki perifericheskikh arterii v eksperimente, v/ch. pp.63032* (Russian).
6. *C.D. Campbell* (1980), *J. Surg.* 4, 2, 227.
7. *C.D. Campbell et al.* (1979), *Ann. Surg.* 190, 6, 740-742.
8. *C. D. Campbell et al.* (1976), *A preliminary report. Surgery*, 79, 5, 485-493.
9. *M. Chvapil, M. Krajicek* (1966), *Statni zdravotnistkie nakladet. December* 153, 163 (Czech).
10. *W. A. Dale* (1959), *Autogenous Vein Grafts and Related Aspects of Peripheral Arterial Disease*, Berlin.
11. *W. A. Dale* (1974), *Surgery*, 76, 6, 849-866.
12. *R. C. Darling* (1980), *J. Surgevry*, 4, 4, 2, (229).
13. *M. E. De Bakey, G. M. Lawrie* (1979), *Ann. Surg.* 189, 3, 303-305.
14. *J. Descotes, J. Brudon* (1986), *Lyon Chirurg.*, 82, 3, 145-146 (French).
15. *J. Descotes et al.* (1985), *Lyon Chirurg.*, 81, 1, 59-60.
16. *J. Descotes et al.* (1974), *Lyon Chirurg.*, 70, 4, 259-260.
17. *R. M. Dickerman et al.* (1980), *Ann. Surg.*, 182, 5, 639-644.
18. *J. P. Gamondes et al.* (1981), *Lyon Chirurg.*, 77, 4, 231-235.
19. *J.W. Hallett et al.* (1980), *Ann. Surg.*, 191, 4, 430-437.
20. *X. Martin et al.* (1985), *Lyon Chirurg.*, 81, 322-326.
21. *N. S. Martinez et al.* (1957), *Surgery*, 42, 6.

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