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A NEW METHOD OF RESTORING DEFECTS IN THE WALL OF GREAT ABDOMINAL VEINS

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Very little has been written on the surgical treatment of injuries to the great veins. In his monograph »The Surgical Treatment of Blood Vessel Lesions« [36] 1951), *Petrovsky* writes: »Even during the last world war no summarizing report was published on the subject of modern treatment of veins lesions.« *Petrovsky* was one of the first to direct attention to the subject and emphasize the importance of its treatment.

In *Byalik's* statistic in 1946 [2] vein lesions amounted to 39,6 per cent of all blood vessel injuries. According to *Huntington*, out of 106 cases of vein injury, 86, i. e. 81.1 per cent, died.

Injuries to the great veins are usually treated by the following methods:

- a) ligation ;
- b) application of a lateral or circular suture ;
- c) application of an indwelling clamp ;
- d) wrapping the injured vein in a cuff of gelatine or fibrine foam.

In case of linear injury without loss of tissue, suturing narrows the calibre of the vein. If, however, in the vein wall a defect of the extent of only a few square millimeters has been caused, the suture will result in narrowing and subsequent occlusion.

When treating their injuries, certain differences must be observed between the systems of the inferior *vena cava* and that of the *portal vein*. In the infrarenal part, the *inferior vena cava* may be ligated or even divided without endangering life. On the other hand, a ligation applied above the renal vein is invariably fatal, leading to death within 2 to 3 days.

Injury to the *portal vein* is an unfrequent occurrence. This may account for the scarcity of reports on the subject. Treatment presents a difficult surgical problem.

The injury may be brought about under various circumstances. *Ferreire* and *Caricchio* [15] observed a girl 12 years of age who had developed symptoms of internal haemorrhage after a fall. On opening the abdomen a linear injury of 8 mm length was found on the anteromedial surface of the portal vein. The same authors collected 19 further cases from the literature. In 4 cases the

haemorrhage from the portal vein had been due to a blow with a blunt object, while in 3 cases to penetrating injury. In 12 cases the injury had been caused during operation. In *Jansen's* case [21], fatal haemorrhage resulted from gall-stone arrosion. During operation the portal vein is easily vulnerable, owing to its identical anatomical relation to the hepatic duct and the choledochus. When investigating into the possible causes of injury during operation, it must be taken into consideration that not only pathological processes such as inflammation, adhesion, tumourous infiltration, etc. may render preparation in the operative area difficult, but also the frequent variations in the anatomy of the portal vein and the extrahepatic bile ducts (*Douglas*, [14], *Griffilian* [18], *Bendle* [1]). These facts account for the statement of *Ferreire* and *Caricchio* [15] that out of 19 cases of portal vein injury 12, i. e. 63 per cent, had arisen in the course of operation. Nevertheless, there are very few reports on operative lesions in the literature. Experiments have been performed in order to clear the problems of portal vein injury and for studying the possibilities of an operative solution.

Ligation of the portal vein in man for a period longer than 20 minutes is fatal, except in case of hepatic cirrhosis. Death ensues with symptoms of blood pressure fall and collapse; venous stasis and haemorrhagic infarcts are found in the stomach, intestines and spleen (*Thöle* [45]). In the dog, ligation of the portal vein leads to death within 2 hours in every case. It is impossible to save the animal either by blood transfusion or by a temporary porto-caval anastomosis (*Mallet-Guy, Device, Gangolphe* [32]). Sudden occlusion of the portal vein results in an abrupt fall with 50 per cent in the blood pressure. If the ligature is taken off within 25 minutes, the blood pressure returns to normal (*Kumata* [29]). Compression of the common mesenteric vein leads to sequelae reversible within 25 minutes. If after ligating the portal vein physiological saline is continuously injected towards the liver, the blood pressure remains at a constant level and the animal may be kept alive for a time (*Borszéký and Baron* [6]). In case of liver cirrhosis the portal vein may be compressed for a longer period; it is even possible to divide the vessel and to ligate the proximal stump. This is the method employed by *Blakemore* [4] for termino-lateral porto-caval-anastomosis, which may be performed if collaterals have developed beforehand. *Torcigliani* [46] carries out two successive operations on dogs. First the vein is narrowed to one of its original lumen, and a month later, when collaterals have already been developed, is the portal vein completely ligated. Part of the collateral veins, the so-called hepatopetal collaterals, conveys the blood from the portal vein to the liver. The most important of these vessels is the pyloric vein, which may dilatate to the size of the portal vein (*Sappey* [40]). The other collaterals, the hepatofugal ones, carry the blood of the portal vein into the area of the inferior and superior venae cavae, to the oesophageal, periumbilical, epigastric, haemorrhoidal veins and the retroperitoneal anasto-

moses (*Josselyn de Jong* [28]). The portal vein usually divides into two branches just before entering the liver. Ligature of one of these is of no fatal consequence considering that in this case blood is still conveyed to the liver by the other branch, as it has been described by *Hedri* [19] in connection with extirpation of the left lobe of the liver in man. Hyperplasia of connective tissue and atrophy of the parenchyma develops on the occluded side while on the undamaged side compensatory hypertrophy develops (*Oehlecker* [35]). Blood from the stomach and the spleen is carried mostly in the left, blood from the intestines into the right lobe of the liver, they being mixed with another only to a slight degree in the portal vein (*G. Moore* [33]). Therefore the first task in case of a lesion to the portal vein consists obviously in restoring the circulation. It is vitally important that this be accomplished within 20 minutes. The measures to be taken depend on the extent of the injury. Linear lesions are sutured (*Ferreire and Caricchio* [15]). Suturing of injuries with tissue defect results in stenosis. A rapidly evolving stenosis of this kind is fatal, although the lethal effect takes a longer time to develop than following an abrupt total occlusion (*Mallet-Guy* [32]). In case of lesions with tissue loss, the defect must be immediately covered in order to restore the vitally important circulation of the portal vein within 20 minutes, without causing stenosis. In the literature available we could find no method recommended for covering defects of the portal vein.

A new procedure has therefore been developed for restituting the wall of the great abdominal veins in the dog. The method consists in covering the area injured with the serous coat of the small intestine, by suturing this to the gap. Experiments were performed in the infrarenal and suprarenal sections of the inferior vena cava as well as on the portal vein.

Experiments and observations

a) *Experiments on the infrarenal part of the inferior vena cava*

The operative area was the section of the inferior vena cava between the meeting point of the iliac veins and the junction of the renal veins. The operations were performed in intratracheal anaesthesia, this being the best method of ensuring an even and easily registrable narcosis (*Csillag—Novák* [11]).

After performing a median laparotomy, in order to prevent shock, the perivascular area was infiltrated with a 0.25 per cent solution of novocaine, with *Vishnievsky's* technique. Then the section of the vena cava inferior between the junction of the iliac veins and the renal veins was laid free. The veins from the lumbar region were ligated and the right urether passing through the area of operation, was retracted. After the inferior vena cava had

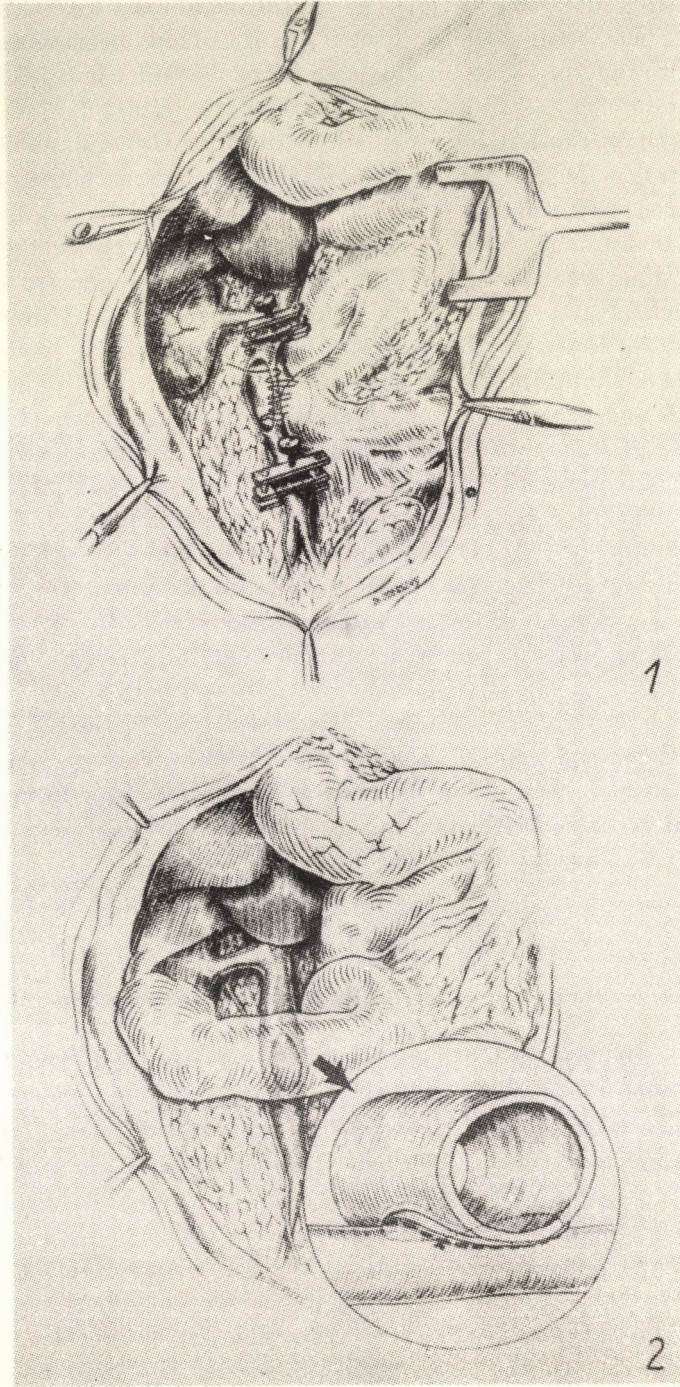


Fig. 1, Fig. 2

been laid free, a clamp was applied proximally from the junction of the iliac veins and another clamp below the orifice of the renal vein. After application of the clamps the respiration became gradually accelerated. On the section between the two clamps, an oval opening 20 mm long and 10 mm broad, was made on the anterior wall of the inferior vena cava, thus producing a defect embracing about half the circumference of the vein. The gap was covered with small intestine, fixing the first loop of the jejunum by means of a continuous everting suture to the inferior vena cava, thus supplementing the defect in the wall of the vein (Fig. 1). In the beginning, atraumatic needles were employed for the operation, but after some time it was discovered that fine serous membrane needles were equally suitable for suturing, which was made with paraffined silk dyed black with India ink. Extreme care must be taken that the defect of the vein should be covered by a piece of serous membrane of sufficient size: one single inaccurately applied stitch may constrict the vessel to such an extent as to produce thrombosis. After having accomplished the suture of the serous membrane, the clamps were carefully removed, taking off first the proximal, then the distal one. The lumina of the intestine and the vein are separated from one another by the intestinal wall (Fig. No. 2). haemorrhage occurred in the sutured area. Anticoagulants have not been used.

Observations

The method described has been tested in two successive series of experiments, first in 11, subsequently in 8 dogs. The 11 dogs operated on in the first series survived the intervention without exception. Post-operative haemorrhage did not occur in any of the cases.

First series

Six of the operated dogs died several days after the operation from inter-current disease or due to technical faults. Five dogs were sacrificed.

Data of death of the six dogs

The first dog died on the 3rd post-operative day from diffuse peritonitis.

The second dog died on the 10th day, due to canine distemper.

The third dog, which a week before had undergone a cranial operation, died on the second day from an unidentified cause.

The fourth dog died on the second day; the urether had been injured during operation.

The fifth dog died on the second day from intestinal obstruction, because at the end of the operation the intestines had been put back into the abdomen with the mesenterium twisted. The chylous cystern had been also injured.

The sixth dog died on the sixth post-operative day from intestinal invagination.

In these six dogs the inferior vena cava had not been narrowed in consequence of the intervention and no obstruction of any kind was found at autopsy.

Data of the five sacrificed dogs

The seventh dog was autopsied 17 days after the operation. Total occlusion and obliteration of the inferior vena cava had occurred in consequence of constriction due to an unsatisfactory suturing technique.

In the eighth dog, examined on the 5th post-operative day, there was no trace of constriction of the vena cava but a small thrombus was discovered in the opening.

The ninth dog exhibited on the 5th post-operative day a nut-sized abscess in the operated area. The novocaine had been contaminated.

The tenth dog was autopsied on the 8th day. The vena cava had retained its original lumen, no trace of constriction was found.

The eleventh dog was examined on the 22nd day. The inferior vena cava proved unobstructed; there was no narrowing. The opening had been replaced by a smooth, shiny membrane.

Second series

In the second series the method was tested upon 8 dogs. All the dogs survived, and no haemorrhage occurred. Three of the eight dogs were killed on the 43rd day after operation. In these dogs no constriction of the vena cava was found, the vein was smooth and shiny at the site of the fenestration. The remaining five operated dogs were all alive at the time this report has been compiled 71 days after the operation.

b) Experiments on the suprarenal section of the vena cava

In two dogs of the second group the operation had been performed on the suprarenal section of the vena cava. In these animals the renal arteries and veins were temporarily ligated for the period needed for covering the injury, 15 to 30 minutes.

c) Experiments upon the portal vein

The following technique was applied for correcting defects of the portal vein. A median laparotomy was performed in intratracheal narcosis. The perivascular space in the area around the portal vein, the vagus nerve and the superior and inferior mesenteric arteries were infiltrated with a 0.25 per cent solution of novocaine (*Vishnievsky*, [48]). In order to decrease venous stasis, the inferior and superior mesenteric arteries were occluded with clamps (*Mallet-Guy*, [32]). The portal vein was laid free and between two clamps an oval

defect 20 mm long, embracing half of the circumference, was made in its anterior wall. Subsequently the defect was covered with the duodenum, fixing its serous coat to the intima of the vein by means of a continuous seromuscular suture (*Csillag—Novák* [9]). After removal of the clamps no haemorrhage was found, cyanosis of the intestines disappeared and mesenteric pulsation was resumed.

The cases were controlled by means of portography (*Dr Zsebők*), which revealed no changes. Tests of liver function performed (by *Dr Mihály Földi*) two months after the operation gave normal results.

Observations

The operations were performed in two series. *In the first series* six dogs were operated upon. During operation only the portal vein was compressed, the superior and inferior mesenteric arteries were left free. All the animals died within a few hours. At autopsy, haemorrhages were revealed in the intestinal wall.

In the second series the operation was performed on ten dogs. Both the portal vein and the inferior and superior mesenteric arteries were occluded for the time of the operation, taking a minimum of 15, and a maximum of 22, minutes. All ten dogs survived. In two cases post-operative haemorrhage occurred because the continuous suture did not hold. On applying a serous suture the haemorrhage ceased. Eight dogs were killed between the 1st and 42nd day. Two were kept alive. Occlusion of the portal vein was not observed in any of the cases.

In some cases the method described may prove life-saving. Stored veins are not always available; preparation of one of the patient's own veins for restoring the gap takes time and the precise suture of the portal vein has to be accomplished rapidly. For all these procedures the short period of 20 minutes at disposal is hardly sufficient.

In our procedure the tissue used for restoring the defect is immediately at hand and the suture is performable within 15 to 20 minutes.

Morbid Anatomy

a) Gross examination

After penetrating into the abdomen next to the scar, the site of the suture was located, eventual adhesions liberated and the sutured section of the intestine severed proximally and distally from the suture. Subsequently, the vein in question (*cava, porta*) was laid free above the section operated upon, eventual lateral branches of the vein were divided and the vein with the intestine attached to it was removed. When the experiment had been performed on the suprarenal

section, the vein was removed in connection with the kidneys, while in portal vein experiments together with the liver.

After having removed the intestine-vein-complex, the intestine was opened and examined on the side opposite the suture.

Two different methods were used for examining the vein. In animals killed a short time, 10 to 12 days, after the operation, the vein was cut open on the side opposite to the injury and the area operated on was inspected. In these cases a thrombus more or less recent, according to the time elapsed, was discovered, filling the slight protuberation formed between the everted border of the defect and the intestine. The lumen of the vein presented no narrowing whatever.

In cases autopsied after some time had elapsed since the operation the vein was opened only in special cases. In these the thrombus had already disappeared from the protrusion, the defect has also disappeared and its site was recognizable only from a more whitish shade of the endothelium. In another group of older cases the vein was not opened; passability was tested by injecting water into the vessel.

The preparations obtained were fixed in formaldehyde. In the vein a transversal plane cut, in the intestine a cut in the longitudinal plane was used as a histological embedding surface.

b) *Microscopical observations*

The fate of the defect and the development of the new vein-wall will be demonstrated in a series of microphotographs showing histological preparations made at different intervals after the operation.

Fig. 3. exhibits a dog killed immediately after operation on the portal vein. The picture illustrates the anatomical conditions produced by the intervention: a) intestinal mucous membrane; b) submucous layer; c) muscular layer of the intestine; d) the U-shaped vein-wall lying next to the intestinal muscle layer; e) the exerted parts of the U-shaped section are the borders of the defect, close upon the serous coat of the intestine. The intima is everted. The space between vein and intestine is an artefact which resulted from shrinking. The defect in the muscle layer on the left side of the picture is the place of a suture knot.

This figure demonstrates, how the gap in the wall of the vein is temporarily substituted by the serous coat of the intestine, closing and encircling the entire lumen of the vein.

As to the subsequent changes occurring in the defect, high power microphotographs of the everted borders of the gap and the serous and muscle layers (traced parts of microphotograph) are demonstrated.

Fig. 4 caval vein defect after five days: *a)* intestinal mucous membrane; *b)* submucous layer; *c)* muscular layer of the intestine. Traces of suture are clearly discernible (*d*). Part of the intestinal muscle layer demonstrated in *Fig. 3, e*, with everted portion of vein (*e*). The injured part is easily discernible within the everted section of the vessel (*f-f₁*). In the small protrusion situated between the everted segment of the vein-wall and the intestinal wall a newly

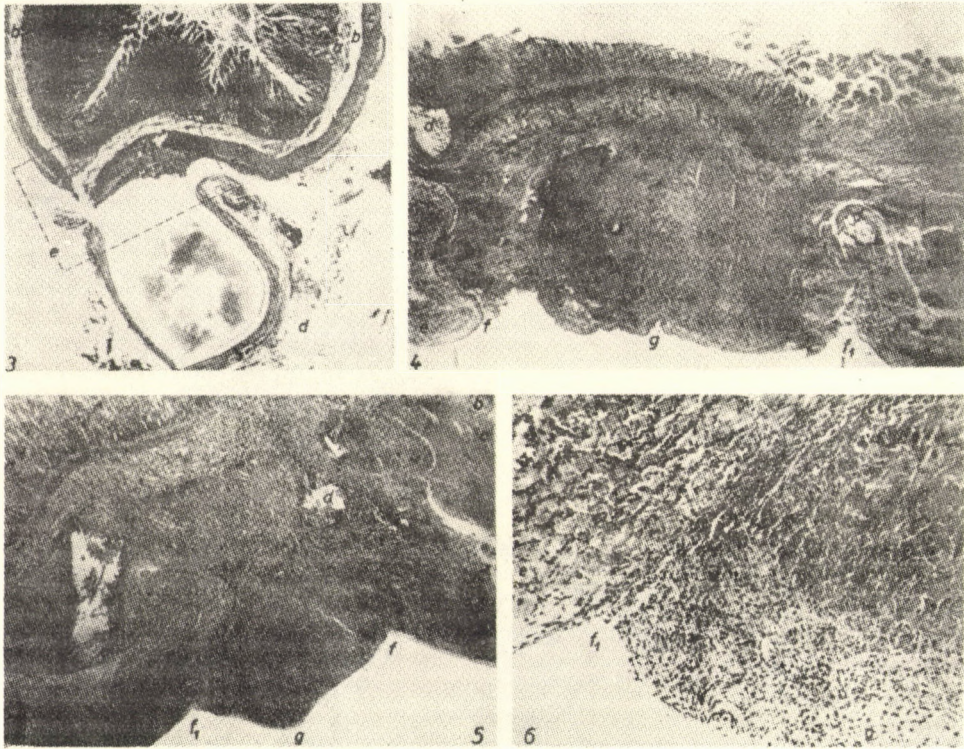


Fig. 3. Animal killed after operation. Haematoxylin-eosin stain. *Fig. 4.* 5 days old case. Haematoxylin-eosin stain. *Fig. 5.* 10 days old case. Haematoxylin-eosin stain. *Fig. 6.* Detail *f₁* of *Fig. 5*, under high power

formed thrombus is visible (*g*). In this picture the lumen of the vein points downwards.

Fig. 5 represents a caval vein defect after 10 days: *a)* mucous membrane of the intestine; *b)* submucous layer; *c)* muscle layer; *d)* suture knots with foreign body granulation formed around them. *e)* Between the sites of foreign body granulation the everted intima of the vein joins the muscle layer of the intestine. Between the two foreign bodies (*f-f₁*) the defect is seen, containing a slightly protruding thrombus (lower part of the picture) *g*). This thrombus is somewhat older than the one shown in *Fig. 4*.

Fig. 6., the same as *Fig. 5. f₁*, under high power. *e)* fenestrated section of vein, *g)* surface of thrombus touching the border of the gap. The picture demonstrates that on the 10th day after operation, the thrombus and its surface

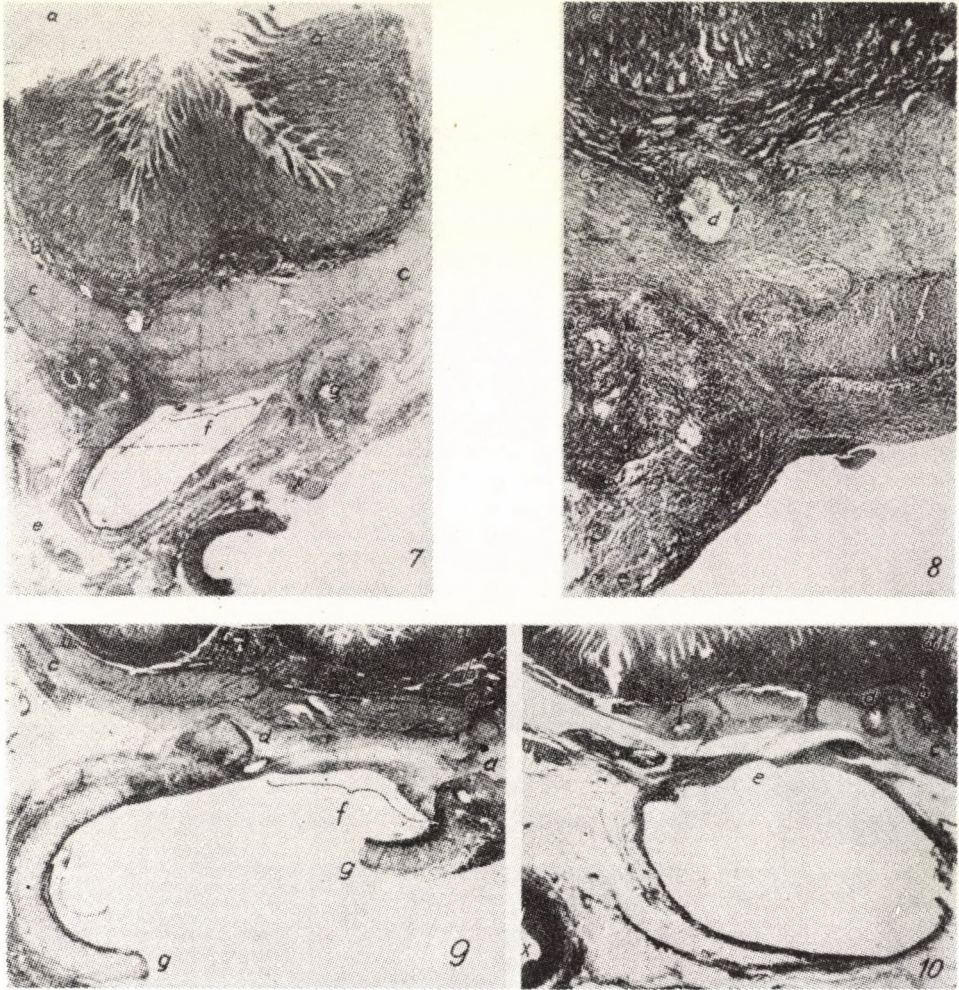


Fig. 7. 14. days old case. Resorcin-fuchsin — van Gieson's stain

Fig. 8. Magnified detail of enclosed part of *Fig. 7.* Resorcin-fuchsin — van Gieson's stain

Fig. 9. 21 days old case. Resorcin-fuchsin — van Gieson's stain

Fig. 10. 43 days old case. X- aorta. Resorcin-fuchsin — van Gieson's stain

have been covered with fresh granulation tissue from the intima and exhibit symptoms of organization. The original thrombus is discernible only in the area corresponding to (*h*). The organization of the thrombus is rapidly progressing at this period.

Fig. 7 represents a portal defect 14 days old: *a*) mucous membrane of the intestine, *b*) submucous layer, *c*) muscle layer, *e*) the vein, *f*) injured section where the gap had been, *g*) foreign bodies surrounded with granulation

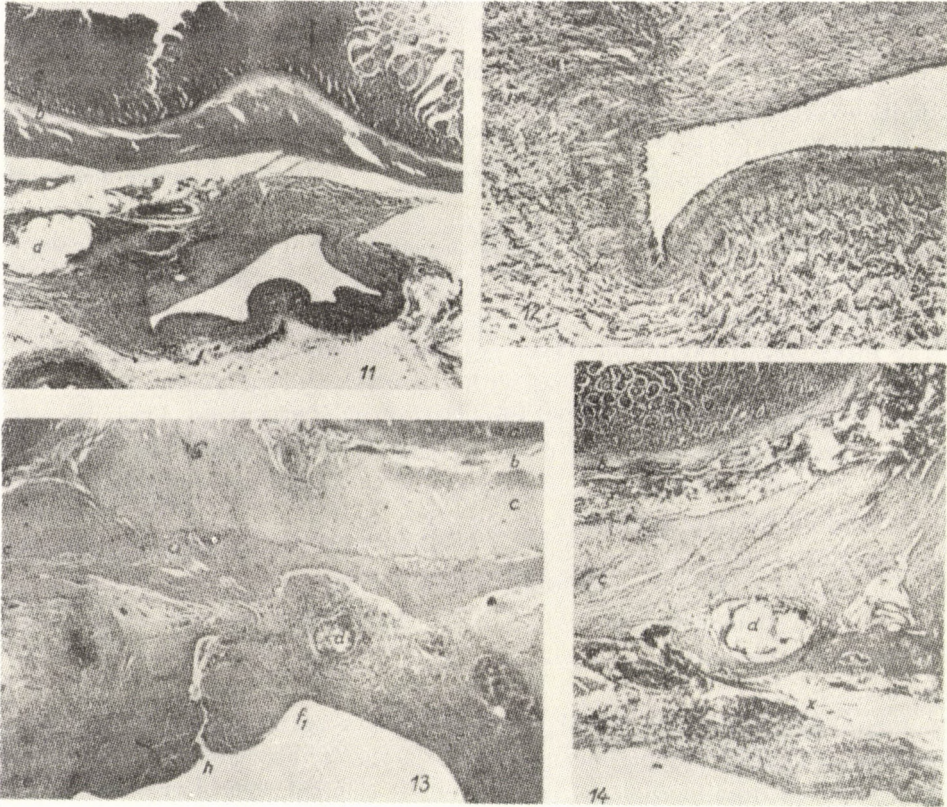


Fig. 11. 43 days old case. *a*- mucous membrane; *b*- submucous layer; *c*- muscular layer. *d*- foreign body; urether. Haematoxylin-eosin stain.

Fig. 12. Magnified detail of part «*a*₁» of Fig. 11.

Fig. 13. 22 days old case. Haematoxylin-eosin stain. *a*- mucous membrane; *b*- submucous layer; *c*- muscular layer; *d*- foreign body granulation; *e*- vein-wall; (*f*-*f*₁). defect. Recanalization of the organized thrombus discernible in the opening (*h*)

Fig. 14. 43 days old case. Resorcin-fuchsin—van Gieson's stain. *a*- mucous membrane *b*- submucous layer; *c*- muscle layer; *d*- foreign body granulation in the muscle; *e*- original wall of the vein; *f*- section of the newly formed vein-wall. Transition of *f*- into *e*- is clearly visible at *X*-; the darkly stained elastic fibres are distinctly seen in the part of original wall but not visible in new wall

tissue. The picture demonstrates that at *f*) a new vein-wall distinctly separated from the intestine has been developed.

Fig. 8 represents a magnified detail of the encircled part of Fig. 7 *e*) showing sections of original wall of the vein, with its intima everted, *a*) mucous membrane of the intestine, *b*) submucous layer, *c*) muscle layer containing

at *d*) a foreign body with granulation around it; *f*) the new wall of the vein consisting at this period already of vascularized fibrous tissue. The surface covered with endothelium, *g*) the adventitial layer of the intestine. The picture shows clearly that by this time the new wall has become entirely separated from the intestine.

Fig. 9 portal defect after 21 days. *a*) mucous membrane, *b*) submucous layer, *c*) muscle layer of the intestine with the vein lying next to it. The gap with its everted borders is seen at *d*) with foreign body granulation. In the section between the two (*f*) the newly formed vein wall, which, when compared to its condition shown in earlier pictures, is seen to be changing gradually from fibrous tissue into collagenous tissue, *g*) the two cut surfaces of the vein opened opposite the injury. It is clearly discernible that in the newly developed section of the vein subintimal elastic network of the original wall is missing.

Fig. 10 represents a 43 days old caval defect. *a*) mucous membrane of the intestine, *b*) submucous layer, *c*) muscle layer of the intestine, *d*) two foreign bodies in the muscular coat with granulation tissue around them. Close to the intestine the uninterrupted circular lumen of the vein. At *e*), between the two foreign bodies surrounded by granulation tissue, where the defect had been, is the newly-formed part of the vein. The darkly staining elastic fibres of the original wall are missing in the newly-formed part, which consists chiefly of collagenous tissue. The space between the vein and the intestine is an artefact due to shrinking during embedding.

Fig. 11 demonstrates another caval defect after 43 days. The vein is a perfect circular unity, as in *Fig. 10*. The defect has been replaced by a new vein-wall.

Fig. 12 shows the detail *a*₁ of the former picture, under high power. Border between the original and the new sections of the vein. The shape of the lumen resulted from fixation. *a*) represent the original wall of the vein. *b*) The undulating elastic fibrous structure discontinues and its transition into the new section, composed of collagenous tissue containing few cells and no elastic fibres, is seen. Both the new and the original sections of the vein are covered with endothelium.

Evaluation of results

The survey of our material and the above described photographs of the histological sections obtained, clearly illustrate the formative process of the new vein-wall. An artificial defect has been produced in the vein. After joining the injured vein to the serous surface of the intestine, in the lumen of the vein a small protuberation is produced and in this a thrombus is being formed because of the lack of endothelium at the site of the injury and owing to the vortex caused by the protuberation. The reason why the thrombus rises scarcely above

the level of the convexity in which it had developed and does not occlude the entire lumen of the vein is explained by the fact that since the lumen of the vein has not at all or only to a slight degree been narrowed, circulation remains unchanged in the operated area and, consequently, no vortex arises and no thrombus is formed except at the immediate site of the injury.

Borst and *Enderlen* ([5], 1909) had discovered the same phenomenon in the course of vein transplantation experiments, when they observed that corresponding to the suture, within the lumen of the vein, a small circular sulcus is formed, in which fibrin is precipitated. This results in a thrombus which later becomes covered with endothelial cells.

Spontaneous healing of small arterial lesions starts equally with thrombus-formation. (*Pirogov* [37], *Küttner* [30], *Prokunin* [36], *Dobrovol'skaya* [12], *Petrovsky* [36], *Lexner*, [31], etc.) In these cases blood escapes through the gap into the area surrounding the defect, and a so-called »button«-shaped thrombus is formed. The process of organization begins in the intimal and adventitial coats, the media takes a lesser part in the process. The scar, which had been named »vessel-callus« by *Stitch* [41], develops in the above-described manner.

In our cases organization of the thrombus started mostly from granulation of the intima, but was promoted by the proliferative process of the serous coat.

Essentially, healing of the vein injury, as well as the conditions of the process, depend on the formation of a thrombus.

After the process of organization, the thrombus undergoes further changes. The vessels and sinuses of the fibrous granulation tissue are merged and this process may even involve the lumen of the vein (Fig. 13, h). In consequence, the volume of the original granulation tissue, viz. of the original thrombus is decreased and at the same time the vein is increased in size and thus the process of recanalization furthered. In Fig. 5, the thrombus rising slightly above the surface of the gap may have caused a narrowing of the lumen. The flattening of the thrombus somewhat protruding into the lumen of the vein is due to the fibrous transformation of the granulation tissue, which, as commonly known, results in shrinking. Wall thrombi of different origin are being transformed into flat hardly perceptible scar-tissue pads by the same mechanism.

At this stage the newly developed section of the vein has already been covered with endothelium. This observation is in agreement with the literary data. *Borst* had observed the presence of endothelium on the 20th, *Stitch* around the 15th day, in their above-mentioned cases. In experiments, in which plastic materials had been employed for restoring the defect, *Jenkins* [27] observed endothelium on the surface of the inlay on the 27th day.

As the granulation tissue becomes older, collagenous fibres begin to predominate. At this stage, the lumen of the vein is already circular and in the fenestrated area a new vein-wall distinctly separated from the wall of the intestine

has been developed, as demonstrated in our 43 days old case. The original defect has been completely filled by the new vein-wall developed from the wall thrombus formed after the operation.

The new vein-wall consists essentially of scar-tissue. Consequently, the new wall is less resistant than the original one had been, owing to the peculiarities of its structure, especially to its deficiency in elastic fibres. (Figs. 12 to 14). The border between the original and the new parts of the vein remains clearly discernible, the patterns of the collagenous fibres in the newly formed section being different. (Fig. 12). Compared with the undulating, thick fibres of the original vein, the fibres in the recently formed part are straight and fragile. The phenomenon is probably due to the fact that in consequence of the fixation of the vein-wall dynamic influences have no effect on it and this factor influencing the development of fibre differentiation is absent. (*Rumyantzev and Suntzova*, [38], *Suntzova*, [42], *Doljaňsky and Roulet*, [13], *Szinai and Jellinek*, [44], *Vanyo*, [47]). Moreover, 43 days is a term relatively short, though in their transplantation experiments *Borst* and *Enderlen* observed perfect histological adaptation in the new area after the same period. Their observations were, however, made on arteries and it must be taken into consideration that in these structures the formative effect of dynamic factors is greater, resulting in a more rapid structural adaptation of the newly-formed tissue.

A further series of experiments has been designed for observing the histological changes occurring in veins separated from the intestine and in case of aorta-intestinal sutures. As a preliminary result, Fig. 15, exhibiting a vena cava separated from the intestine 70 days after the operation, may be demonstrated. The animal was killed 40 days after the vein had been separated from the intestinal wall. In this case it is almost impossible to differentiate the new section of the vein from the original one. Only the foreign-body granulation and a slight thickening of the intima indicate the area that had been injured. Further experiments are, however, required in order to arrive at definite conclusions.

It must, however, be emphasized that the method described above, i. e. suturing the intestine upon the fenestrated area of the vein is not equivalent to substituting the vessel-wall. The intestine takes part in filling the gap only so far, as the organization of the thrombus starts also from the serous coat. It is a newly formed vein-wall by which the defect in the vein is replaced and only the development of the new tissue is ensured by the wall of the intestine.

Different methods of restituting defects in both arteries and veins have been described in the literature. The procedures recommended range from homo-hetero-transplantation, substitution with muscle, fascia, peritoneum etc., to the employment of plastic materials. (*Bier*, [3]; *Borst* and *Enderlen*, [5], *Braytzev*, [7], *Bogoraz*, [56], *Carrel*, [8], *Fischer* and *Schmieden*, [36],

Jassinovsky [22], *Morozova* [34], *Petrovsky* [36], *Frantze* [16, 17], *Jenkins* [23—27], *Swensen and Gross* [43], *Vanyo* [47]).

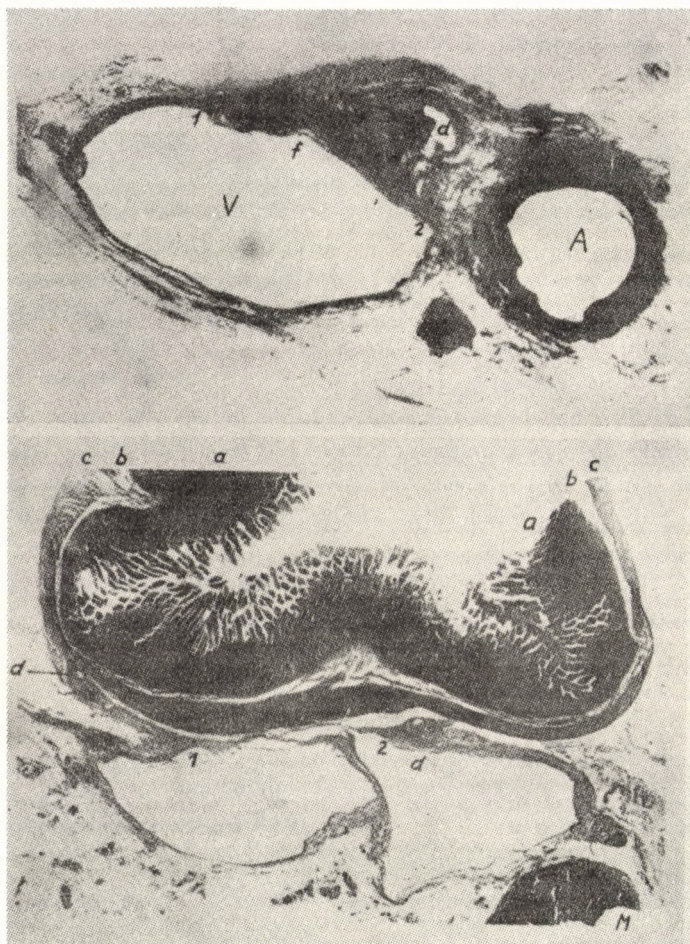


Fig. 15. Dissected vena cava. Resorcin-fuchsin — van Gieson's stain. *A*- aorta; *V*- inferior vena cava; *f*- The defect was situated between parts 1 and 2; *d*- foreign body granulation, at 1, formation of small intima pad

Fig. 16. Picture showing the area below the meeting point of the inferior vena cava and the renal vein. (Resorcin-fuchsin — van Gieson's stain.) *a*- mucous membrane; *b*- submucous layer; *c*- muscle layer. Foreign body granulations are observable at *d*. Opening of the vein had extended from 1 to 2. *M*- Suprarenal gland

Borst and Enderlen have supplied the most detailed experiments on vein and artery transplantation. Their percentage of successful cases was not favourable. Histological examination of the transplanted tissue showed regressive changes, new tissue growth occurring only in the original vein, where it started

in the adventitial and intimal layers gradually to grow over the transplanted tissue.

Experiments for restituting vein injuries with muscle, fascia, and peritoneum came also short of expectation. The transplanted tissue became necrotic, resulting in further necrosis of the vein.

In trials using plastic materials for restoring vessel defects it has been discovered that the plastic materials applied were replaced by scar-tissue, and the gap in the wall of the vein was gradually filled by the latter. From within, the scar became covered with endothelium.

In contrast with other procedures, our procedure presents the great advantage that the material used for restoring the defect, i. e. the serous surface of the intestine, is more physiological, as far as it retains its original function, as well as its blood and nerve-supply to a much higher degree, than any other kind of transplanted tissue, pediculated flaps not excepted. The danger of other methods, the possibility of necrosis, does not occur in this case. To our best knowledge, this has been the first instance when an organ transplanted in order to fulfill some new function could in addition retain its original function.

Another danger in the treatment of vessel injuries consists in the development of an obliterating thrombus. The material observed by us is sufficiently large for allowing the conclusion that with the method employed, there is no probability of that occurrence. According to the histological observations, the process of healing follows the same course as during spontaneous recovery, i. e. the defect becomes covered by a thrombus and the organization of the latter presents the essential basis for healing. In cases with extensive loss of tissue, as it has been demonstrated in our experiments, there is no possibility for that mechanism, in that instance, however, the function of the serous coat of the intestine consists in ensuring the formation of a wall thrombus, allowing the restitution of the continuity of the vein-wall. The functional continuity of the vein has also been reestablished in a satisfactory manner, the lumen of the vein exhibiting no constriction or substantial narrowing whatever at gross examination.

It has to be mentioned as a point of some interest that, when the injury had been inflicted on the suprarenal section of the vein above the collateral branches, the missing part of the septum that had divided the vessel, has also been newly developed. The defect extending to both branches of the vein has been completely regenerated (Fig. 16).

No histological changes were found to occur in either the kidney or the liver.

Summary

Defects in the great abdominal veins in dogs were covered with small intestine. Experiments were made on (I) the inferior vena cava and on (II) the portal vein.

I

An artificial defect was produced on the inferior vena cava making an oval gap 20 mm long and 10 mm broad on the anterior wall of the vein. The defect was then covered with the first loop of the small intestine, suturing the serous surface of the intestine to the vein. Two series of operation were performed. In the first series the experiment was performed on 11 dogs, all of which survived the intervention. Haemorrhage did not occur in any of the cases. 6 dogs died from intercurrent disease or in consequence of an incorrect technique. 5 dogs were sacrificed after the operation. In 9 cases out of 11 the inferior vena cava proved unobstructed. In the second series, the method had been tested on 8 dogs, all of which survived the operation. No haemorrhage occurred. 3 dogs were killed on the 43rd day after operation. The inferior vena cava proved unobstructed in all animals. 5 of the operated dogs were still alive 71 days after the operation.

II

Ligature of the portal vein causes death after 20 minutes. The defect inflicted on the portal vein of dogs was covered with small intestine. An artificial tissue defect was made by excising a section 20 mm long and 10 mm in diameter from the anterior wall of the portal vein. The gap was covered with the first loop of the small intestine, suturing the serous coat of the intestine to the vein. 2 series of operations were performed. 6 dogs were operated upon the first series, in which only the portal vein was compressed for the time of the operation, without compressing the superior and inferior mesenteric arteries. All 6 dogs died. In the second series the method was tested in 10 dogs with the modification that both the superior and inferior mesenteric arteries were clamped for the time of the operation. All 10 dogs survived the intervention. No thrombus was developed, the portal vein remained unobstructed. In the literature available no data pertaining to the subject were found and up to now it was not possible to restore the defect of the portal vein.

III

It could be established by histological analysis of the experiments that temporary covering of the abdominal vein defect with small intestine which retains its function will promote the development of a new vein-wall. In place of the defect, in the protuberance between the serous coat of the intestine and the borders of the gap, a thrombus is formed by the organization of which the defect becomes gradually filled and a new vein-wall is developed. The new section of the vein is essentially a scar being entirely composed of collagenous fibres and completely lacking elastic fibres.

No case of obliterating thrombosis occurred in the experiments and it could be established that the method recommended is well suited for restoring injuries situated in a branching section of the vein. In one case it could be demonstrated that after the new vein-wall had developed, it became functionally independent of the serous coat of the intestine used for covering the defect.

In the course of the experiments it became possible to induce the organism, by establishing a new environment, to replace a defective tissue from its reserve cells.

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НОВЫЙ МЕТОД ПРИКРЫТИЯ ПРОБЕЛОВ БОЛЬШИХ БРЮШНЫХ ВЕН

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Резюме

Мы прикрывали пробелы больших брюшных вен у собак тонкой кишкой. Опыты продились 1) на нижней полой вене, 2) на воротной вене.

1.

На нижней полой вене мы подготовили искусственный пробел путем вырезки участка сосудистой стенки длиной 20 мм и шириной 10 мм из передней стенки вены. Этот пробел или окно мы прикрывали первой петлей тонкой кишки, которая фиксировалась сосудисто — серозными швами. Эта операция была исполнена в двух сериях.

В первой серии опыты производились над 11 собаками. Кровотечение не произошло ни в одном случае. 6 собак через известный срок погибли из-за интеркуррентных заболеваний и технических промахов. 5 собак было нами убито. В 9 случаях из 11-и нижняя полая вена явилась совершенно проходимой.

Во втором ряде опытов мы испытывали свой метод над собаками. Все 8 собак пережили операцию. Кровотечение не произошло ни в одном случае. 3 собаки мы убили на 43 м дне после операции. Нижняя полая вена оказалась полностью проходимой 5 оперированных собак до сих пор живет. Со дня операции прошел 71 день.

2.

Лигатура воротной вены после 20 минут ведет к смерти. Мы прикрывали травматический пробел воротной вены у собак помощью тонкой кишки. Мы подготовили искусственный пробел путем вырезки участка сосудистой стенки длиной 20 мм, шириной 10 мм из передней стенки сосуда. Этот пробел или окно мы прикрывали первой петлей тонкой кишки, и фиксировали сосудисто-серозными швами. Операция проводилась в двух сериях. В первой серии мы проводили опыты над 6 собаками. Во время операции мы зажимали только воротную вену, но не зажимали верхнюю и нижнюю брыжеечную артерию. Все 6 собак погибли. Во второй серии опытов мы испытывали свой метод над 10 собаками. Во время операции мы предварительно зажимали и верхнюю и нижнюю брыжеечную артерию. Все 10 собак пережили операцию. Тромб не образовался. Воротная вена явилась полностью проходимой. В имеющейся к нашему распоряжению литературе нет данных о подобных опытах. Пробел воротной вены до сих пор ничем не удалось прикрыть.

3.

На основе патогистологического исследования подопытных животных мы установили, что временное покрытие пробелов больших брюшных вен с помощью кишки, сохраняющей свою функцию, ведет к дополнению пробела путем организации тромба, образовавшегося в выпячивании, возникающем между серозной оболочкой кишки и окном в сосудистой стенке. Таким образом возникает новый участок сосудистой стенки. Если сопоставлять этот участок со старой стенкой сосуда, его можно считать рубцом стенки, поскольку в нем не встречаются эластические, а только коллагенные волокна. Опыты доказывают и то, что данный метод не ведет к образованию тромбов, запирающих просвет сосуда, и его можно применять и в тех случаях, когда пробел сосудистой стенки расположен над разветвлением.

В одном случае мы в ходе опыта снимали кишки. Этот опыт показал, что вновь образованная стенка сосуда через некоторое время функционально отделяется от серозной оболочки кишки, первоначально покрывающей пробел. Таким образом в наших опытах нам удалось путем создания новой обстановки принуждать организм к замене тканевого пробела из резервных клеток.