Experimental Mesenteric Venous Occlusion:

III. Diagnosis and Treatment of Induced Mesenteric Venous Thrombosis

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ALTHOUGH revascularization of the acutely and chronically obstructed superior mesenteric artery has been accomplished upon occasion, acute thrombosis of the superior mesenteric vein has not been treated with survival short of massive, disabling intestinal resection. It is the purpose of this report to describe:

1) a simple technic in the dog to simulate mesenteric venous thrombosis in man
2) the anatomic and physiologic derangements attending thrombosis so induced
3) a method for unequivocal diagnosis of mesenteric venous thrombosis
4) a comparison of methods for resuscitation and support of such animals
5) a technic for uniform restoration of patency of the experimentally thrombosed mesenteric veins with preservation of normal intestinal function and architecture

Materials and Methods

Healthy adult mongrel dogs weighing 7 to 25 Kg. were used for these experiments after immunization and worming and a 21-day period of isolation and observation. Anesthesia was induced with intravenous pentobarbital (25 mg./Kg.) and respirations were assisted continuously by a piston respirator (Harvard model 607A) attached to a cuffed endotracheal tube. A femoral artery was cannulated and connected to a Sanborn transducer (267B) and recorder for continuous recording of intra-arterial pressure. Transfemoral catheterization of the inferior vena cava was performed for determination of central venous pressure and as a source of mixed venous blood samples. Patency of catheters in prolonged experiments was maintained with dilute heparin solution, no animal receiving more than 0.25 mg./Kg. body weight. Celiotomy was performed aseptically. The superior mesenteric vein was mobilized immediately proximal to its confluence with the inferior mesenteric vein and doubly ligated with 2-0 silk. 333 N.I.H. units of bovine topical thrombin in 2 ml. saline were injected retrograde through a fine needle into the superior mesenteric vein. The midline incision was closed anatomically with catgut sutures.

The following determinations were performed in the course of each experiment:

1) microhematocrits
2) white blood cell count (cells/mm^3 $\times$ 10^3)
3) blood pH (Beckman pH meter)
4) blood volume (I^{131} tagged serum albumin in the Volemetron) (ml./Kg. body weight) *
5) superior mesenteric arterial flow rates with a non-cannulating electromag-

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* Serum concentrations of albumin and total proteins were normal in each animal.

TABLE 1. *Experimental Mesenteric Venous Thrombosis: Study Groups*

Group	No. Dogs	Treatment	
I	10	None	
II	4	Splenoportography at celiotomy	
III	8	Transfemoral retrograde superior mesenteric arteriography	
IV	4	Normal saline I.V.	75 ml./Kg. in first hour after induced thrombosis, 75 ml./Kg. in next 3 hours.
V	6	Ringer's lactate I.V.	As in Group IV.
VI	4	Kanamycin I.P.	12.5 mg./Kg. in 20 ml. saline immediately after induced thrombosis.
VII	4	Heparin I.V.	3 mg./Kg. immediately after induced thrombosis.
VIII	4	10% dextran (M.W. 40,000) in normal saline I.V.	15 ml./Kg. first hour after induced thrombosis, 15 ml./Kg. in next 3 hours.
IX	4	Tris (hydroxymethyl) aminomethane I.V.	300 mg./Kg. in first hour after induced thrombosis, 350, 400, 450 mg./Kg. in succeeding hours.
X	10	Thrombectomy	As in Group V; thrombectomy 1.5 hours after induced thrombosis.

netic probe and flowmeter (Carolina Medical Electronics) (ml./min./Kg. body weight)

6) biopsy and necropsy tissue specimens stained with hematoxylin and eosin

The experimental groups are shown in Table 1.

Results

Control Group

Eighteen untreated animals died 1 to 4 hours after induced thrombosis of the superior mesenteric vein. The progression of notable visible changes included mesenteric venous dilatation, bleeding into the mesentery, cyanosis of the bowel, intestinal spasm, intestinal congestion, paralytic ileus, and the weeping of serosanguinous fluid from the bowel and mesentery into the peritoneal cavity. Intestinal necrosis and perforation did not occur. Hypovolemia, tachycardia, hypotension, hemoconcentration, leukocytosis, acidosis, and reduced flow through the superior mesenteric artery were uniformly noted. Data in Table 2 describe ten dogs from the control group.

Thrombosis extended from the superior mesenteric vein ligature well into the peripheral tributaries. Wet weight of clots removed at necropsy and thrombectomy ranged from 6 to 15 Gm. Grossly and histologically these clots resembled fresh spontaneous thromboses (Fig. 1). Free injection of bovine topical thrombin into the unoccluded inferior vena cava or portal vein resulted in prompt profound hypotension lasting 5 minutes or less, with spontaneous recovery and without thrombosis of the vein so treated.

A basic abnormality attending interruption of the superior mesenteric vein in the dog, whether by ligature alone or with induced thrombosis, is the translocation of large amounts of extracellular, primarily circulating, fluid into the bowel, mesentery and peritoneal cavity.[20] Such fluid shifts were measured by blood volume determinations, careful serial weights of the abnormal intestine and mesentery and volume of ascitic fluid developing during the course of the experiment. Table 3 compares a group of dogs subjected to ligation of the superior mesenteric vein with those in which thrombosis was also induced. The latter animals experienced a greater blood volume deficit immediately and showed less

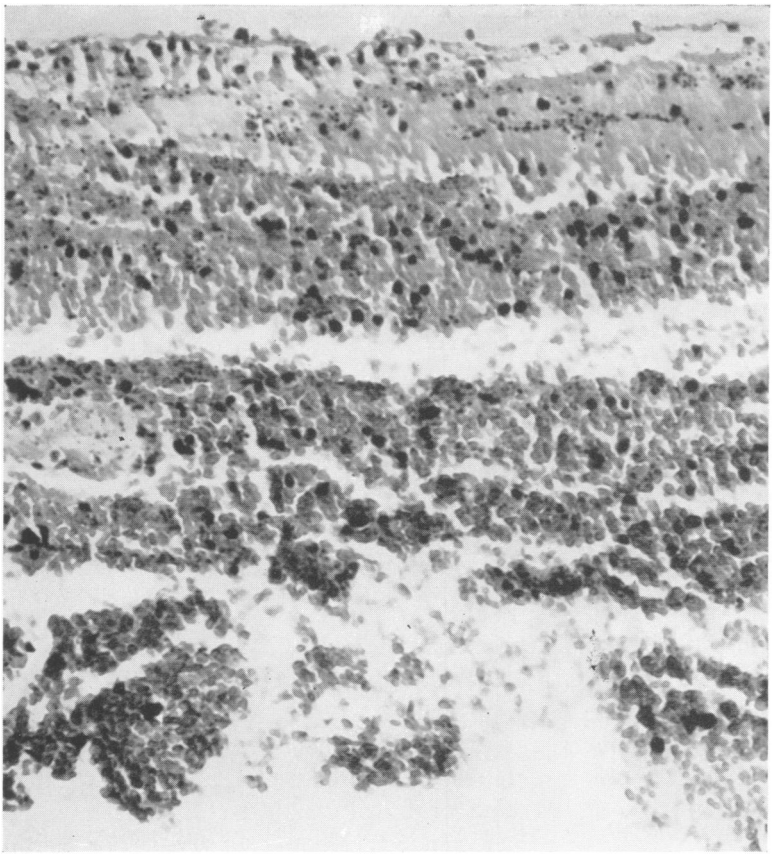


FIG. 1. Photomicrograph of thrombin-induced clot in superior mesenteric vein of control dog dying at 3.25 hours (150 ×).

TABLE 2. *Experimental Thrombosis of the Superior Mesenteric Vein: Untreated*

Parameter	Minutes after Induced Thrombosis					
	0	15	30	60	120	180
Pulse (#/min.)	135 ±21	149 ±27	162 ±19	169 ±24	187 ±23	121 ±30
Systolic B.P. (mm. Hg)	114 ±16	81 ±21	81 ±16	72 ± 8	65 ± 9	46 ±11
Venous pressure (ml. saline)	44 ± 8	47 ±10	39 ± 6	33 ± 9	20 ± 5	22 ± 3
Hematocrit (% r.b.c.)	40 ± 5	51 ± 4	54 ± 7	47 ± 6	46 ± 5	45 ± 4
Leukocytes (10^3 cells/mm.^{3})	7.3 ± 1.5	7.2 ± 2.1	8.7 ± 3.2	10.4 ± 2.5	12.8 ± 2.8	15.3 ± 2.7
Blood pH (units)	7.41± .06	7.40± .11	7.25± .12	7.14± .09	7.07± .12	6.96± .05
Blood volume (ml./Kg.)	93 ± 6	64 ± 8	60 ± 5	57 ± 9	51 ± 6	47 ± 7
S.M.A. flow (ml./min./Kg.)	20.9 ± 3.9	4.6 ± 2.0	4.9 ± 1.5	4.6 ± 2.1	4.0 ± 1.8	3.1 ± 1.9
Survivors	10	10	10	10	5	3

No animal in this group survived four hours.

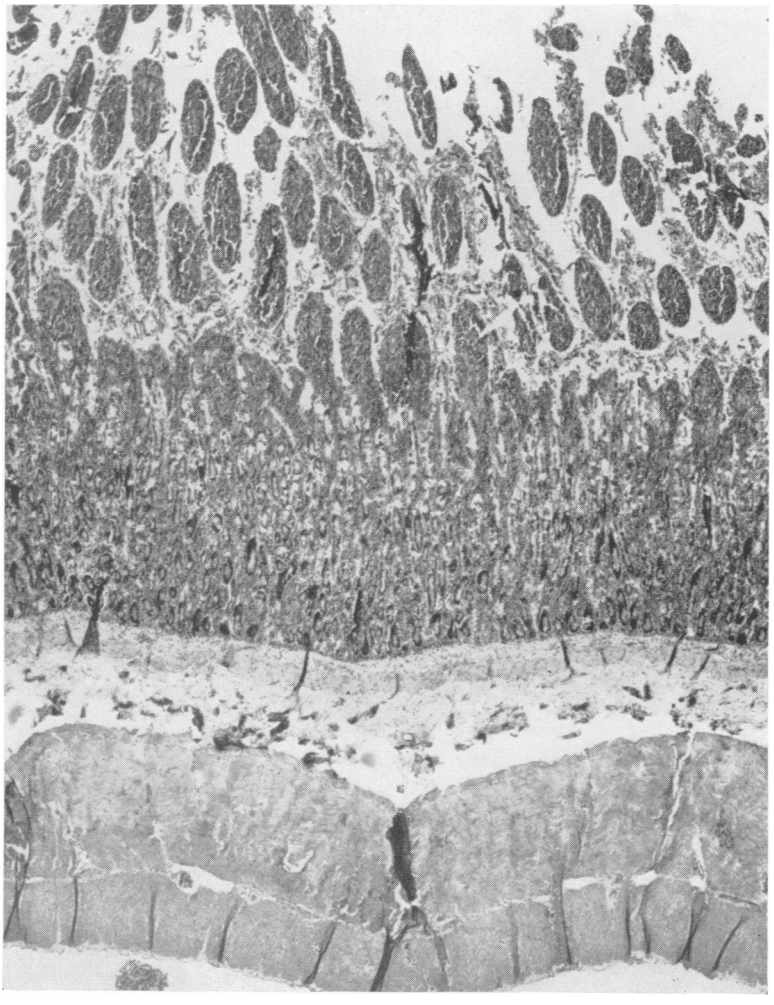


FIG. 2. Hemorrhagic congestion of ileum with separation of muscularis mucosa 3.75 hours after induced thrombosis in control animal. Necrosis is absent (35 ×).

tendency to correct this deficit subsequently than did the dogs undergoing simple ligation of the superior mesenteric vein.

The small bowel histologically is the site of intense hemorrhagic congestion (Fig. 2) unassociated with necrosis or infarct. Examination of liver, heart, kidney, lung, colon, stomach and spleen showed only those changes compatible with prolonged hypotension. Injection of peritoneal fluid into the abdominal cavities of mice did not kill them.

Diagnostic Methods

Belated diagnosis is in large part responsible for the high mortality attending mes-

TABLE 3. *Comparison of Fluid Deficits Following Simple Ligation or Induced Thrombosis of the Canine Superior Mesenteric Vein*

Hours after Interruption	Volume Deficit—ml./Kg. body weight			
	Ligation		Thrombosis	
	B.V.I.*	B.V.II**	B.V.I.*	B.V.II**
½	86^{10}	80^{2}	90^{10}	88^{2}
1	76^{12}	83^{5}	92^{10}	89^{2}
2	68^{10}	64^{4}	80^{5}	90^{5}
3	62^{7}	60^{3}	81^{3}	92^{2}
4	51^{5}	54^{5}	72^{3}	94^{3}
6	47^{3}	49^{4}	—	—
12	12^{3}	28^{2}	—	—

Values used are the mean of the number of observations shown by the superscript.

* Blood volume determined by I^{131}-human serum albumin (Volemetron).[30]

** Computed from intestinal weights and ascitic fluid, measured at necropsy or by the method of Turner.[26]

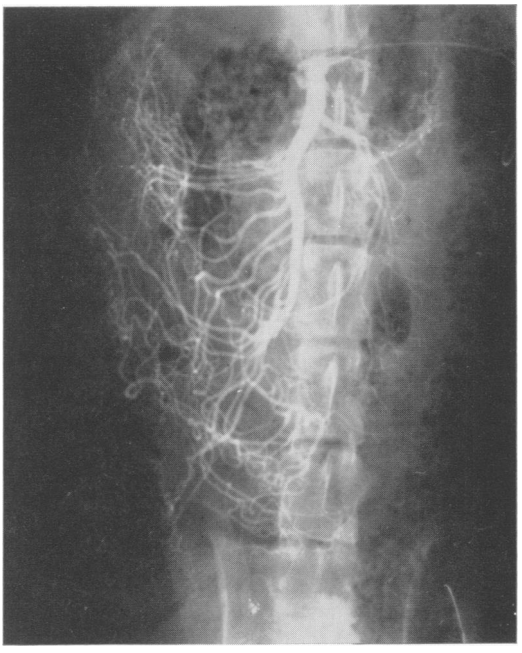


FIG. 3. Arterial phase of control superior mesenteric arteriogram four seconds after injection of 30 ml. contrast material.

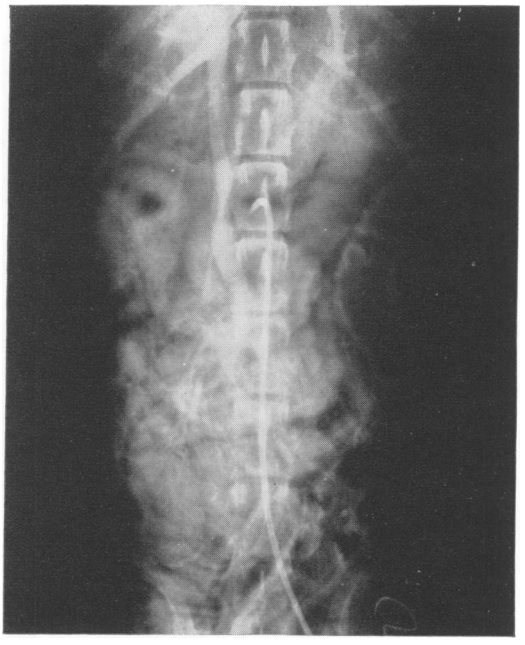


FIG. 4. Venous phase of control superior mesenteric arteriogram 10 seconds after injection showing normal superior mesenteric and portal veins.

enteric venous thrombosis in man. The clinical situation, hematologic changes, and fluid redistribution are by no means diagnostic.[3, 11, 18, 29] By the time such signs are indicative of mesenteric venous occlusion it is invariably too late for revascularization and reversal of intestinal changes. Seeking diagnostic methods of high reliability, splenoportography and retrograde percutaneous superior mesenteric arteriography were performed in dogs subjected to mesenteric venous thrombosis.

Splenoportography. At celiotomy the spleen was mobilized and 15 ml. 60 per cent methylglucamine diatrizoate was injected into the parenchyma of the organ. After control angiograms were obtained, the superior mesenteric vein was thrombosed and repeat splenoportograms were obtained in the same manner 1 hour later.

Absence of the dilutional effect of normal superior mesenteric venous influx was noted in the venograms of all four animals. Similar observations were also present in two control venograms. One one occasion sufficient retrograde filling occurred to outline the thrombus in the superior mesenteric vein. In view of the likelihood of failure to demonstrate such dilutional effect in normal superior mesenteric veins, no further evaluation of this technic was carried out.

Mesenteric Arteriography. A curved-tip Odman catheter was introduced into the left femoral artery and subsequently manipulated at celiotomy 1 cm. into the superior mesenteric artery. A control arteriogram was then obtained in each animal with injection of 30 ml. of 60 per cent methylglucamine diatrizoate in 2 seconds. Subsequently, the superior mesenteric vein was thrombosed by injection of topical thrombin dissolved in 2 ml. of 75 per cent sodium diatrizoate and repeat superior mesenteric arteriograms were obtained at 5 minutes and again at 30 to 60 minutes.

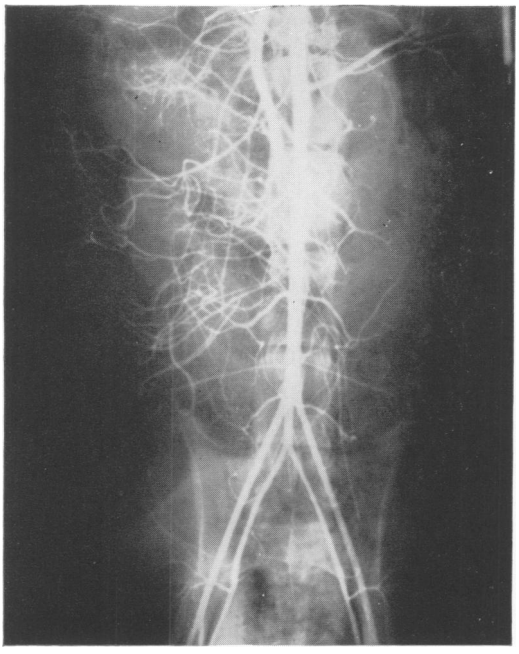


FIG. 5. Superior mesenteric arteriogram five minutes after induced venous thrombosis—eight seconds after injection.

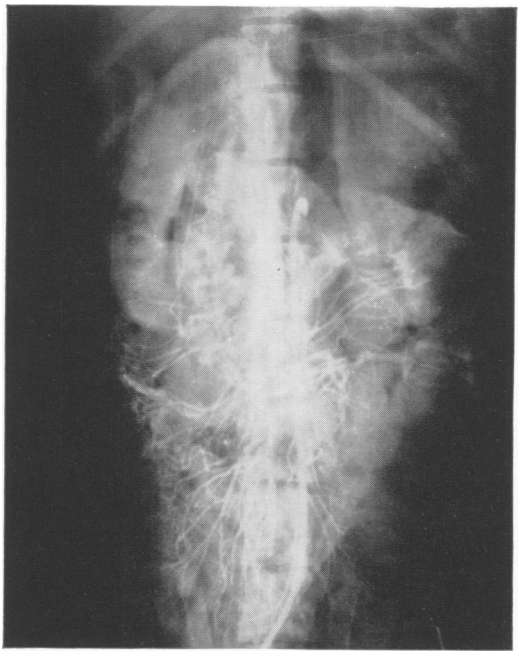


FIG. 6. Superior mesenteric arteriogram five minutes after induced thrombosis—40 seconds post injection. Opacity overlying L2 and L3 represents thrombus (see text).

The radiographic technical factors are described elsewhere.[8]

This technic proved unequivocally diagnostic of mesenteric venous thrombosis in each of eight dogs studied. False positive examinations were not seen in prolonged simple intestinal obstruction, in closed loop obstruction or in segmental strangulated obstruction.

The control superior mesenteric arteriogram showed reflux of contrast material into the aorta in only two animals. Intestinal arteries were numerous, large, and tapered gradually (Fig. 3). Contrast material flowed rapidly through arteries and reached the intestinal wall within 2 seconds. Duration of opacification of the vasae recti did not exceed 6 seconds. Intense filling of the superior mesenteric vein and portal system occurred within 8 seconds (Fig. 4). The bowel wall did not appear thickened nor was intraluminal contrast material seen. The angiographic findings following mesenteric venous thrombosis include:

1) reflux of dye into the aorta in each animal (Fig. 5)
2) spasm of the superior mesenteric artery and its major branches which was more marked after 30 minutes than at 5 minutes (Fig. 5)
3) opacification of few peripheral arterial branches of small caliber (Fig. 5)
4) prolongation of the entire arterial phase with dye remaining in the arterial system for as long as 40 seconds (Fig. 6)
5) intense opacification of the thickened bowel wall (Fig. 7)
6) intraluminal contrast material was noted occasionally
7) failure to opacify the mesenteric venous-portal system despite the lapse of 40 seconds between injection of the contrast medium and the exposure of the roentgenogram

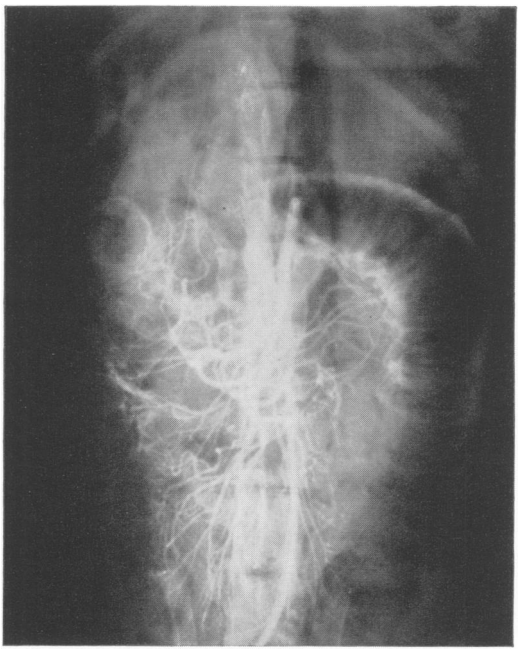


FIG. 7. Superior mesenteric arteriogram 30 minutes after induced thrombosis—21 seconds post injection. Opacity overlying L3 represents thrombus (see text).

Restoration of Fluid Losses

Attempts to restore the plasma volume were carried out as follows.[20] Amounts of electrolyte solutions equivalent to 15 per cent of the body weight (about twice the total blood volume) were administered intravenously, half the total being infused in the first hour following venous occlusion and the remainder in the next 3 hours. The results of such therapy is presented in Tables 4 and 5 and Figure 8.

Normal saline proved briefly effective in restoring blood volume with improvement in pulse, blood pressure, venous pressure and superior mesenteric arterial flow rates. Although the hematocrit decreased uniformly in animals given intravenous saline, the pH of the blood, magnitude of leukocytosis and mortality of the treated group of dogs did not differ from the animals not treated.

Lactated Ringer's solution (pH 8.5) was more beneficial than normal saline in equivalent amounts. All parameters except leukocytosis showed a change toward more normal values. Although all animals died, survival times were better among the animals treated with lactated Ringer's solution than among the animals of the other experimental groups with the exception of those treated by thrombectomy.

Intraperitoneal Antibiotic Therapy

The benefits attending the use of antimicrobial therapy in clinical and experimental forms of strangulated intestinal ob-

TABLE 4. *Comparison of Supportive Treatment in Experimental Mesenteric Venous Thrombosis (Mean Values One Hour After Thrombosis)*

	Study Groups						
Parameter*	I Control	IV Isotonic Saline	V Ringer's Lactate	VI Kanamycin I.P.	VII Heparin	VIII Dextran**	IX T.H.A.M.
Pulse	169	150	138	168	174	145	161
Systolic B.P.	72	86	98	94	70	96	76
Venous P.	33	39	42	30	30	43	37
Hematocrit	47	44	44	46	41	36	47
Leukocytes	10.4	11.8	9.6	5.8	10.9	11.1	9.9
Blood pH	7.14	7.10	7.28	7.15	7.18	7.20	7.34
Blood vol.	57	67	78	56	59	71	63
S.M.A. flow	4.6	5.8	7.2	4.7	4.1	4.8	3.9
Survivors	10/10	4/4	6/6	4/4	3/4	4/4	3/4

* Units as noted in Table 2.

* Molecular weight 40,000 in saline.

struction prompted a trial of intraperitoneal kanamycin.[6, 7] Although the leukocytosis was uniformly aborted survival was not improved nor were the other parameters studied affected. No prolongation of survival times could be attributed to this form of therapy when combined with other methods.[21]

Anticoagulation

Intravenous infusion of heparin either before or after induction of thrombosis, in amounts sufficient to prolong clotting times to 20 minutes, did not affect survival time. Indeed, in one animal intestinal hemorrhage was pronounced and contributed to the death. In a related study[21] three of six dogs so treated with heparin developed significant intestinal bleeding without improved survival.

Dextran in Saline

Dextran (molecular weight 40,000)* in normal saline was infused systemically following induced mesenteric venous thrombosis in an effort to offset the profound stasis of flow in the intestinal microcirculation. Mesenteric capillary perfusion and integrity appeared improved when examined with the dissecting microscope. Moderate improvements were noted in pulse, blood pressure, hemoconcentration and blood volume. However, the blood pH and superior mesenteric artery blood flow were not significantly altered. These dogs did not survive longer than the control animals did (Fig. 8). On the other hand, dextran (m.w. 40,000) proved to be a valuable adjunct to thrombectomy in hypotensive animals not treated with lactated Ringer's solution.[21]

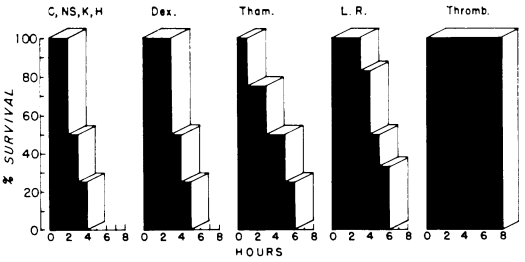


FIG. 8. Graphic representation of results of treating experimental thrombosis of the superior mesenteric vein. Efficacy of method is proportional to the area in black.

Correction of Acidosis

Tris-hydroxy-aminomethane** was infused to correct the profound acidosis ac-

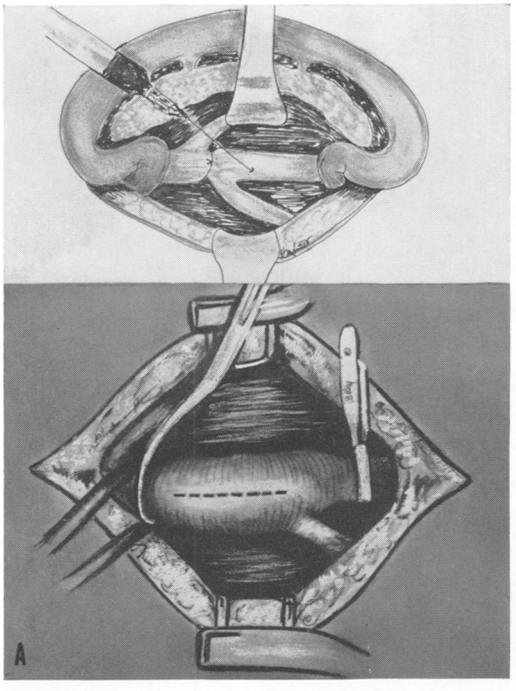
* Supplied as Rheomacrodex, 10 per cent in normal saline, by Pharmacia Laboratories Inc., New Market, New Jersey.

** Supplied as Talatrol by Abbott Laboratories, North Chicago, Illinois.

TABLE 5. *Comparison of Supportive Treatment in Experimental Mesenteric Venous Thrombosis (Mean Values Two Hours After Thrombosis)*

	Study Groups						
Parameter*	I Control	IV Isotonic Saline	V Ringer's Lactate	VI Kanamycin I.P.	VII Heparin	VIII Dextran**	IX T.H.A.M.
Pulse	187	161	149	184	184	148	190
Systolic B.P.	65	69	83	70	60	71	66
Venous P.	20	22	29	24	18	31	25
Hematocrit	46	38	34	45	45	33	47
Leukocytes	12.8	13.6	11.7	6.1	13.3	13.9	10.9
Blood pH	7.07	7.02	7.18	6.10	7.06	7.11	7.30
Blood vol.	51	53	59	52	47	61	49
S.M.A. flow	4.0	3.8	5.3	4.1	3.3	4.8	3.7
Survivors	5/10	2/4	6/6	2/4	2/4	4/4	3/4

* Units as noted in Table 2.
* Molecular weight 40,000 in saline.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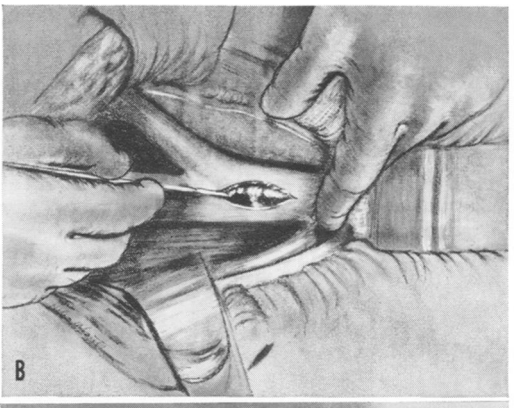
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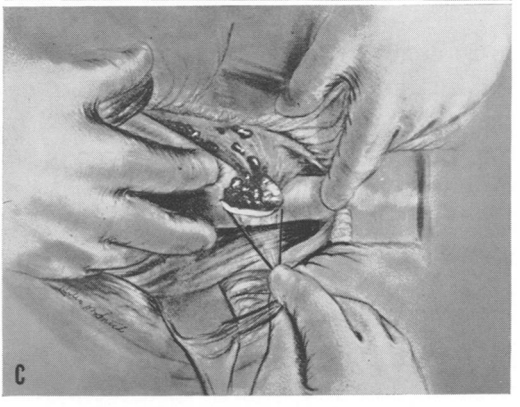
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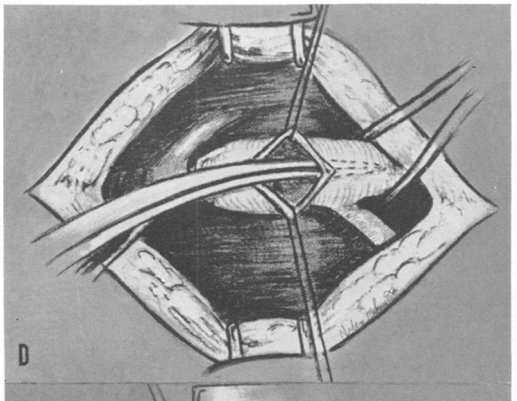
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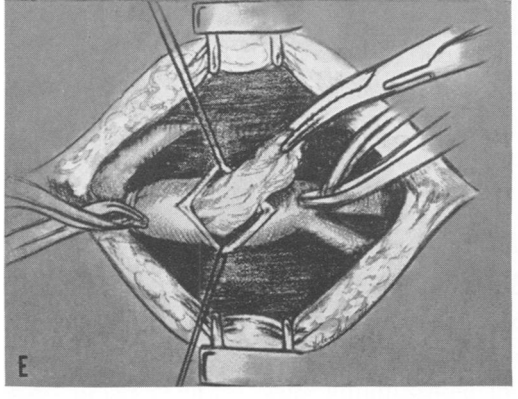
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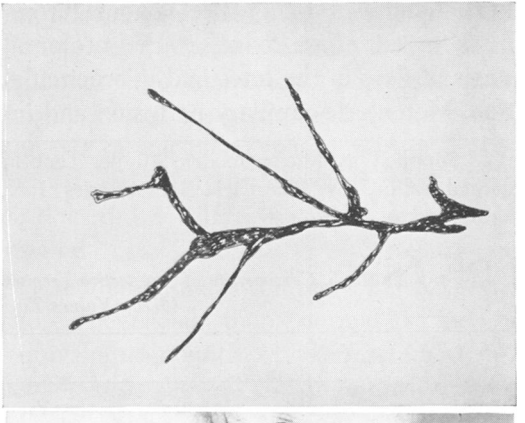

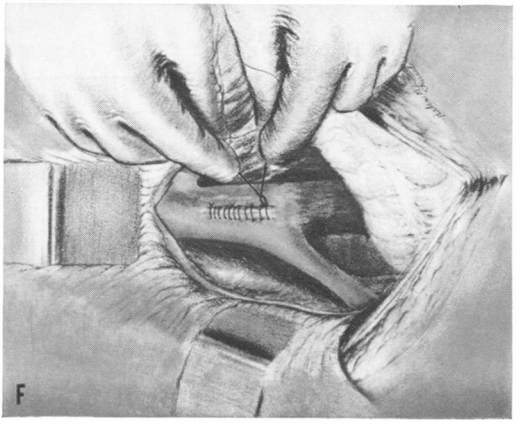
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TABLE 6. *Patterns of Recovery after Thrombectomy (Mean Values in 5 Dogs)*

Parameter*	Control	Thromb.	Hours Post-thrombectomy		
			1	4	24
Pulse	128	153	166	141	130
Syst. B.P.	120	100	84	106	112
Venous P.	60	43	63	77	64
Hematocrit	45	44	51	46	41
Leukocytes	7.1	11.8	15.7	20.6	18.1
Blood pH	7.44	7.17	7.07	7.28	7.38
Blood volume	92	61	78	104	93
S.M.A. flow	19.1	6.1	21.6	16.7	20.8

* Units as noted in Table 2.

companying mesenteric venous thrombosis. More nearly normal blood pH values were maintained initially, but the animal became progressively more acidotic, requiring larger amounts of the buffer. Survival times were not significantly improved, nor were the other measurements appreciably altered (Tables 4 and 5, Fig. 8).

Thrombectomy

In contrast to the lack of benefit of the previously described agents, uniform long-term survival and venous patency could be achieved by deligation and thorough operative removal of the induced thrombus and its propagations. Essential features of the procedure are shown in Figure 9. Proximal control of the portal vein is mandatory to prevent embolization of hepatic radicles. A venotomy of 1.5–2.0 cm. is advisable to allow the easy introduction of instruments to extract the thrombus. Control of the peripheral branches of the vein is essential to limit the loss of blood at the time of thrombectomy. Vigorous bleeding from the venotomy may follow completion of thrombectomy; this is poorly tolerated by animals already hypotensive and acidotic. Repair of the venotomy and restoration of blood flow through the intestine is often followed by more hypotension and acidosis. Fluctuation of the several parameters studied in five dogs treated by thrombectomy is shown in Table 6. Each of ten such animals were permanent survivors. Venous patency was confirmed by phlebography or sacrifice and necroscopy.

The primary defect of this technic in the experimental animal was the inability to obtain permanent survival in dogs in which thrombectomy was delayed more than 2 hours following induced thrombosis. Although removal of the thrombus and the venous ligature resulted in a patent vein in each animal, early death followed. The cause is unknown. Further study of this aspect of the experimental system may permit prolongation of the period in which thrombectomy will insure survival.

Discussion

Mesenteric venous thrombosis occurs in man most often under circumstances of altered blood coagulation, portal hypertension and intraperitoneal sepsis.[3, 18] It occurs almost as frequently as arterial occlusion.[11, 29] The paucity of survivors from this catastrophe and the debility resulting

FIG. 9. Technic of thrombectomy. Upper inset shows method of thrombosis. A. Proximal and distal control obtained. B. Venotomy. C. Thrombus expressed by exerting gentle pressure on the small intestine and mesentery. D. Extraction of residual adherent thrombus. E. Removal of proximal extent of thrombus with retrograde flush of portal vein. F. Repair of venotomy. Lower inset is a photograph of a typical extracted thrombus.

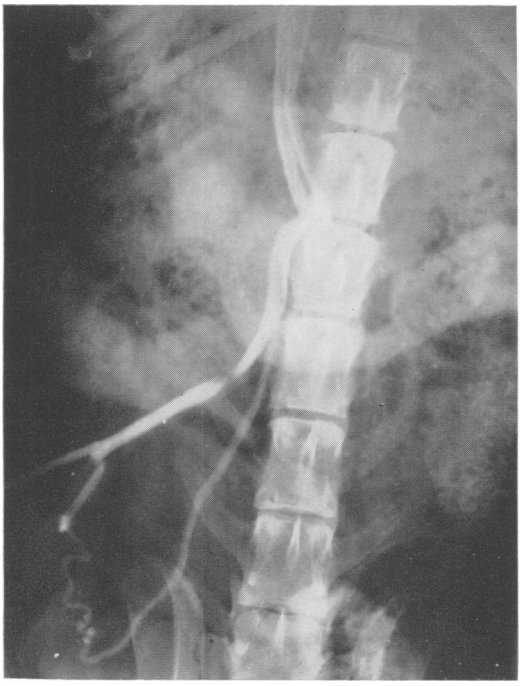

FIG. 10. Mesenteric phlebogram five months after thrombectomy confirms mesenteric-portal venous patency. Venotomy site is opposite L3-L4 interspace.

from massive intestinal resection are potent stimuli for the experimental study of this problem.

The experimental system has proved useful in itself. Contrary to the studies of Nelson and Kremen [19] and of Lepley, Mani and Ellison [12] survival of the untreated animal following simple ligation of the superior mesenteric vein is not unusual.[20, 23] Ligation or thrombosis of the mesenteric veins in this and other laboratories is to be differentiated from other experimental intestinal strangulations associated with obstruction.[22] The method of induced thrombosis, suggested by Holden [10] has proved simple, inexpensive and uniformly effective. Other technics [27, 28] were briefly tried and discarded. This experimental system has much in common with experimental hemorrhagic [15] and endotoxin shock,[1, 14] particularly the sequestration of a significant portion of the entire extracellular fluid within the small intestine and its mesentery. Certainly the magnitude of the fluid losses into a slowly or noncirculating compartment provides a thorough test of substances which may restore depleted intravascular volume. This deficit, developing primarily in the first half hour following induced mesenteric venous thrombosis, represents more than 6 L. in a 70-Kg. man. Such measurements do not account for the more slowly altered intracellular water and electrolyte disturbances. The work of Shires, Williams and Brown [24] suggests that deficits of similar magnitude, although incurred more slowly, may follow standard operative procedures under certain circumstances. Furthermore, clinical experience following infusion of massive volumes of lactated Ringer's solution in individuals sustaining large burns [16] or undergoing extensive pelvic surgery in this hospital has been most satisfactory. Based upon this information, amounts of electrolyte solutions equivalent to the entire blood volume infused in the first hour of the experiment and an equal amount distributed over the next 3 hours proved superior to more timid protocols. Pulmonary edema did not occur in this study. Such therapy tends to increase the extravasation of fluid into the intestine slightly but to increase the measurable blood volume by a relatively greater amount. The superiority of lactated Ringer's solution to normal saline may be attributed largely to its alkalinity. Data obtained in standard hemorrhagic shock experiments indicate that lactated Ringer's solution prepared in the Barnes Hospital (pH 8.5) is superior to lactated Ringer's commercially available (pH 6.5).[5]

Blood flow through the superior mesenteric artery correlate well with that reported by several groups using a variety of technics.[2, 9, 25] The reduction in mesenteric arterial flow encountered after venous thrombosis is almost identical to changes in portal blood flow in experimental hemorrhagic shock.[4, 17]

The mere suspicion of mesenteric arterial or venous occlusion is usually sufficient to warrant immediate operation. However, un-

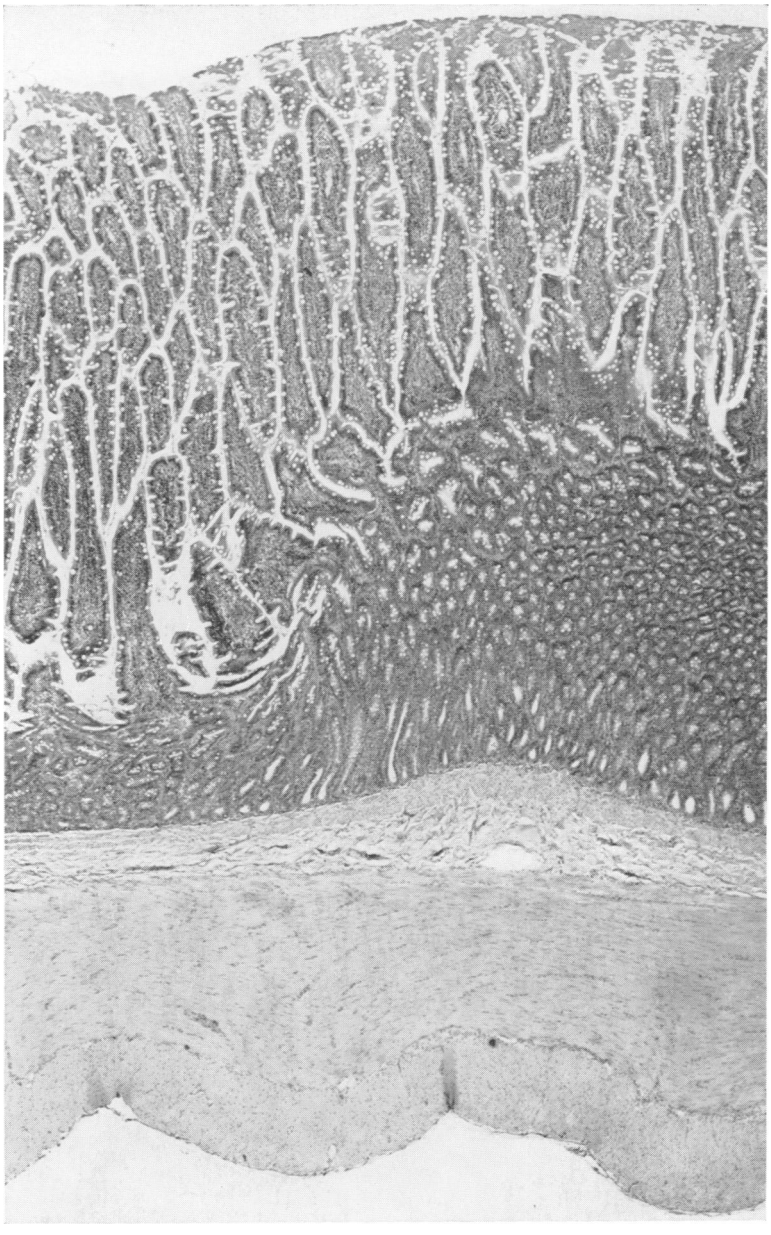

FIG. 11. Normal small bowel architecture three months after thrombectomy (30 ×).

der circumstances in which the diagnosis is equivocal and other factors increase the risk of celiotomy, transfemoral retrograde mesenteric arteriography should be performed. Roentgenographic diagnoses of arterial or venous obstruction is highly reliable with this technic. Similarly, when mesenteric venous thrombosis is encountered with the intestine even equivocally viable, thrombectomy should be performed. Dramatic improvement always follows successful thrombectomy in the experimental animal. Should revascularization be less than perfect, the extent of intestinal resection may be reduced.

Summary

Thrombosis of the superior mesenteric vein in the experimental animal is followed uniformly by tachycardia, hypoten-

sion, hemoconcentration, leukocytosis, acidosis, hypovolemia and reduced mesenteric arterial blood flow.

Retrograde transfemoral superior mesenteric arteriography is an effective method for the diagnosis of mesenteric venous occlusions. Diagnostic radiographic signs in the dog are illustrated.

Agents designed to alter the abnormalities induced by mesenteric venous thrombosis have been evaluated and found ineffective. Survival after mesenteric venous thrombosis occurred only when thrombectomy was performed.

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