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Mesenteric vascular occlusion

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MESENTERIC VASCULAR OCCLUSION

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INTRODUCTION

In writing this paper, it was decided to use a "textbook" form; that is, Definition, Etiology, Pathology, Signs and Symptoms, Differential Diagnosis etc.

The discussion of differential diagnosis was necessarily limited to the diseases considered most often confused with mesenteric vascular occlusion according to the various authors.

It will be noted that incidence and mortality are not discussed. The reason for this is that these are not matters of discussion but consist of facts and statistics and the tables presented will show these statistics and are self explanatory. In the chapters necessitating and presenting statistics, the references have been limited, as far as statistics are concerned, to those presenting a large enough series of cases to furnish reliable statistics.

MESENTERIC VASCULAR OCCLUSION

DEFINITION

Mesenteric vascular occlusion is a condition of obstruction of the mesenteric vessels resulting in either hemorrhage or anemic infarction and resulting in certain pathological changes and manifestations which will be described in this paper.

HISTORY

The first report of mesenteric vascular occlusion was published in 1843 by a German surgeon named Tiedeman. The next published work was by another German named Virchow, who in 1847, reported one case and four years later two more cases. Virchow was the first to recognize and explain embolism and thrombosis and the beginning of our knowledge of these processes, as they occur in the mesenteric vessels, dates from his works. The next and first report of a number of cases was by Litten in 1875, who at that time, summarized twenty cases. (28)

Since 1875, the experimental work initiated by Litten and Faber has been repeated with numerous modifications, but by no means constant results, by Cohnheim, Cohn, Kader, Tansisi, Rydygier, Bolognesi, Zesas, Marek, Bier and many others. During the same period, the number of clinical observations has steadily increased.

Up until 1895, these diagnoses had been made at autopsy and it was not until 1895 that the first intestinal resection was done in the treatment of mesenteric vascular occlusion.

Dr. J. W. Eliot, a Boston surgeon, was first to perform the intestinal resection and he reported two cases. One was with complete recovery following the removal of four feet of jejenum for gangrene due to thrombosis of the superior mesenteric veins; and the other with a fatal outcome following resection of the descending colon for gangrene due to thrombosis of the mesenteric artery. (24)

The next report of any significance was by Watson in 1894, who reported on the diagnosis and surgical treatment for mesenteric vascular occlusion. (28)

Thrombosis of the mesenteric veins following splenectomy was first reported by a Dr. H. B. Delatour of Brooklyn in 1895. (24)

About this time, the disease began to attract attention and Gordon, in 1898, and Tyson, in 1901, came forth with reports of operative recoveries. In 1904, Jackson, Porter and Quinby reviewed the 214 cases that had been recorded up to that time. (28)

In 1913, Trotter presented a monograph based upon his review of 359 cases in the literature and seven personal cases. His work is very complete including work on experimental animals. It is of interest that his findings and ideas as to treatment vary little from present day ideas. (73)

In 1921, Klein made an extensive review of all reported cases, and up to that time, there were reports of only twenty four successful resections. (44)

In 1923, a series of fourteen cases were reported by Brady and three years later, Cokkinis published a comprehensive work based upon

seventy six cases collected from hospitals along with some original work on the mesenteric circulation. (10)

ANATOMY OF MESENTERIC VESSELS

THE ARTERIES

The superior mesenteric artery supplies the whole of the small intestine from the duodeno-jejunal flexure to the ileo-caecal valve, together with the caecum, the ascending and first half of the transverse colon. It assists the inferior mesenteric in the supply of the second or left half of the transverse colon. The superior mesenteric artery arises from the aorta behind the pancreas and at an acute angle immediately below the celiac axis and it's main stem paralleling to some extent, the course of the abdominal aorta and passing forward in front of the third part of the duodenum enters the root of the mesentery. Thus it may be seen that the superior mesenteric artery has a direct connection with the heart and therefore is vulnerable to emboli from above. (11) In this, it passes downward and to the right and ends near the caecum by joining with the descending branch of the ileocolic artery. The superior mesenteric artery gives numerous branches. Ten to sixteen intestinal branches, which are divisible into two groups, jejunal and ileal, pass from it between the layers of the mesentery towards the small intestine. Each divides into two branches which anastomose with those of adjacent arteries to form a series of arcades from which secondary branches are given off. The process of division and union is repeated. In the case of the lower ileal arteries, this

may happen three or four times, so that four or five tiers of arches are formed. In the upper jejunum, only one or two arches are present. From the terminal arcades, straight vessels pass to supply the intestinal wall. These, called vasa recta, will obviously be longer, the higher in the mesentery they are situated. According to Cokkinis, there are no anastomoses between the vasa recta, but of course their areas of supply in the intestinal wall may overlap to some extent. The inferior pancreaticoduodenal artery arises from the proximal part of the superior mesenteric artery or it's first jejunal branch. By joining with the superior pancreaticoduodenal artery, a branch of the gastroduodenal, it forms an obvious channel for the development of a collateral circulation in cases of occlusion of the superior mesenteric artery. (58) The value of this anastomosis is doubted by Trotter, who says that the communications between the mesenteric arteries and others mentioned below should not be reckoned as functionally capable by themselves of supplying enough blood for the vitality of the intestine, though they may become so under certain circumstances which are very rare. (73) The middle colic artery arises from the upper part of the superior mesenteric artery and passes forward in the transverse mesocolon, where, near the bowel it divides into left and right branches which anastomose with the right branch of the left colic and the ascending branch of the right colic arteries respectively. A marginal arcade is thus formed along the transverse colon.

The right colic and ileocolic arteries are also branches of the

superior mesenteric artery. They pass to the right towards the ascending colon. Near the margin of the colon, each divides into two branches. The descending branch of the ileocolic anastomoses with the termination of the superior mesenteric artery. The ascending branch joins the descending branch of the right colic, while the ascending branch of the latter joins with the right branch of the middle colic. The marginal arcade along the colon, formed so far by the anastomoses of the branches of the middle, right and ileocolic arteries, is completed on the left side in similar manner by the branches of the inferior mesenteric artery, which is itself a branch of the middle, right and ileocolic arteries, is completed on the left side in similar manner by the branches of the inferior mesenteric artery, which is itself a branch of the aorta. There is thus formed a marginal arterial arcade all around the colon. From it, vasa recta pass to supply the large bowel. This is the common arrangement of the blood supply of the colon, but a dissection of many bodies shows occasional examples of a pattern of arcades simulating those of the mesentery.

As seen from the above description, there are the following anastomoses between the mesenteric and other arteries:

1. The inferior phrenic with the upper jejunal arteries through the hepatic, gastroduodenal, superior pancreaticoduodenal and inferior pancreaticoduodenal.
2. The lumbar arteries (4 pairs); small branches anastomose with branches from the hepatic and colic arteries to form

the retroperitoneal plexus of Turner.

3. The last dorsal and the ileo-colic.
4. The internal iliac with the inferior mesenteric through the internal pudic, inferior hemorrhoidal and middle hemorrhoidal, the latter itself arising from the anterior division of the internal iliac.

VEINS

According to Brown, there is considerable difference between the arterial and venous systems of the mesenteries and he places special emphasis on the differences between the arterial and venous systems in mesenteric vascular occlusion; but most all of the other authors believe that the distribution of the veins in the mesentery and mesocolon corresponds to a great extent with that of the arteries. (44) (11) The portal system differs from the caval venous systems in that it is situated between two capillary systems--that of it's drainage area and that of the liver. Further, the veins constituting it are devoid of valves. The superior mesenteric drains the whole of the small intestine with the exception of the duodenum, the caecum, the ascending and first half or two thirds of the transverse colon. It joins the splenic to form the portal vein. The inferior mesenteric drains part of the transverse colon, the descending colon and sigmoid, and the upper part of the rectum. It enters the splenic close to the termination of the latter.

In contrast to the arterial distribution, all blood collected in the veins of the abdominal part of the digestive tract, except

the lower part of the rectum, spleen, pancreas and gall bladder passes into the portal vein by which it is filtered through the liver, then through the hepatic veins to the vena cava. Any thrombus arising in the liver may pass on as an embolus into the cephalad channels producing damage where it lodges. Such an embolus could not pass into the caudad channels against the current of venous blood, but a thrombus arising in the liver or below could block off the venous channels resulting in a descending thrombosis extending into part or all of the branches of the superior mesenteric vein. In contra distinction, a thrombus arising in the smaller iodicles of this vein may give rise to an ascending thrombus and thus progressively block off more vessels or become embolic to produce infarction of the liver. The anatomic factors of the venous system predispose to thrombosis more than those same factors do in the arterial system.

The anastomoses between the tributaries of the mesenteries are much freer than those of the branches of the mesenteric arteries. From the point of view of morbid anatomy, as well as of clinical medicine, the anastomoses between the portal and caval systems are of great importance.

Collateral venous circulation to relieve portal obstruction in the liver may be effected by the accesory veins of sappy which normally drain blood into the liver form anastomoses with the systemic circulation. These consist of:

1. Tributaries from the gastrohepatic omentum.

2. Tributaries from the wall of the gall bladder.

3. Tributaries from the Glisson's capsule.

4. Tributaries from the suspensory ligament.

5. Para-umbilical veins: occasionally a patent umbilical vein.

6. Tributaries from the coronary ligament of the liver.

The following communications exist between the portal and superior vena caval systems:

1. Gastro-esophageal anastomoses.

2. Anastomoses between the peritoneal veins and those of the diaphragm and through these with the intercostal veins, the internal mammary vein, and the azygos and hemi-azygos veins.

3. Anastomoses between the para-umbilical or patent umbilical veins and the superior epigastric and through it with the internal mammary vein.

Communications between the portal and inferior vena caval systems are as follows:

1. Venous plexus of Retzius, consisting of

   (a) Anastomoses with hemorrhoidal veins (large and numerous).

   (b) Anastomoses with opposite parts of intestinal canal: duodenum, ascending and descending colon, and rectum. Here the peritoneal parietal intestinal veins (die peritonalen Darmwandvenen) communicate with the veins of the parts not covered by peritoneum, i.e. with

tributaries of the inferior vena cava.

(c) Anastomoses of veins of the descending colon with the left renal vein.

(d) Tributaries from the peritoneum communicate with each other, and drain both into the portal and inferior caval systems. These tributaries correspond with the 'Plexus of Turner'.

2. Anastomoses between the peritoneal venous plexus in the posterior layer of the anterior abdominal wall and the veins of the visceral peritoneum on the one hand (portal system) and on the other with the subcutaneous veins of the anterior abdominal wall. The latter communications take place either through a patent umbilical vein (Baumgarten), or through one or more para-umbilical veins (Sappey): further, through perforating tributaries in the linea alba and recti muscles.

3. Right and left renal-portal anastomoses.

4. Finally abnormal anastomoses; e.g. porto-spermatic, porto-uterine, porto-prostatic, etc.

The above anastomoses of the portal and other venous systems have been given in full as they apply with nearly equal force to the mesenteric. (58) (47) & (73)

ETIOLOGY

The factors which may seemingly lead to mesenteric vascular occlusion are as numerous and may be as vague as those responsible

for thrombosis and embolism and any blood vessels. In reviewing the reported cases and according to the views expressed by the authors of the various articles, there are a number of causes on which all men agree, and then there are isolated cases of unusual causes along with unusual theories which may lead to mesenteric vascular occlusion. In a few cases, the occlusion seems to be without cause. (24)

Even though the etiologic importance of certain diseases and conditions must always be considered and recognized, the fact that occasionally one cannot be identified must not be overlooked and must not lead to the exclusion, positively at least, of mesenteric vascular occlusion in the diagnosis of the acute and subacute abdomen.

Mesenteric vascular occlusion must be divided into arterial and venous occlusion in describing the etiology.

VENOUS OCCLUSION

Various authors disagree somewhat as to the relative percentage of occurrence of arterial and venous occlusion, and the combination of the two. Most men believe that the venous occlusion exceeds arterial occlusion by a considerable margin. According to Bowen, reporting a collective series of 1,142 cases, the arteries were involved in 51.2 per cent; the veins in 43.8 per cent and both in 6 per cent. (7) On the other hand, Trotter reporting a series of 163 cases found 41 per cent were due to venous obstructions; 53 per cent to arterial; and 6 per cent to simultaneous and venous obstruction. Still another authority, Moore, considers venous ob-

struction to be as high as 75 per cent. (58) The other reports fall between these extremes. Venous occlusion is practically always thrombotic.

Most of the etiologic factors discussed can be placed into four major groups.

(1) Infectious

This group includes all cases in which there is a known infection in the regions drained by the mesenteric venous more lymphatic system. Also, in areas from which direct extension of the infection to the mesenteric vascular system would be possible, or in cases in which general circulatory sepsis exist. Thus included in the first group are: thrombophlebitis, pelvic septic progresses, peritonitis, appendicitis and general sepsis.

(2) Hematogenous

This group includes blood diseases or changes which are known to predispose to thrombosis; for example: splenic anemia, polycythemia vera, and leukemia.

(3) Traumatic

This includes thrombosis from any sort of trauma to the mesenteric vessels, tearing of the mesentery, and trauma from abdominal procedures.

(4) Mechanical Causes

This is the largest group consisting of portal stasis and it's various causes, pressure from adhesions or congenital

bands across the mesenteric vessels and pressure from tumors, herniae and volvulus. (24) (10) (75) (50) (55)

All men agree that the two main causes are portal obstruction and sepsis and their various causes; but there are some differences of opinion as to their relative importance. Douglas places them in the following order: 1. Sepsis, especially appendicitis and pelvic disease. 2. Hepatic cirrhosis. 3. Polycythemia vera. (17) Moore, on the other hand, places portal obstruction first and peripheral sepsis second; and he also gives as a source of the sepsis, diverticulitis, ulcerative colitis and gall bladder disease in addition to the more common causes. (58)

Ochsner believes that an insufficiently appreciated cause of thrombosis is that associated with prolonged ingestion of alcohol and reports five cases to prove his point. (17)

According to Green, fatigue and emotional strain may cause mesenteric vascular occlusion, following a study of his cases and the experiments of Cannon, showing that increased adrenalin due to these factors decrease the clotting time of the blood by one half and therefore favoring thrombosis. (14) One of the more hypothetical causes is advanced by Bucura in which he attempts to include various hypothetical toxic substances present in eclampsia, leiomyoma uteri, normal puerperium, etc. Warren and Eberhard definitely denounce this on the basis that until definite evidence can be produced, as it has not been so far, that there are such substances which change the nature of the vessel wall in such a way as to in-

duce thrombosis, they see no reason for assuming the existence of these substances while there are other factors which can equally as well explain the process. (75) (30)

A few authors include retrograde embolus but those discussed and explained on this basis could be explained on the basis of multiple thrombosis. (75)

In 60 per cent of the Mayo series of the cases of mesenteric vascular occlusion, the accident was unrelated and in 40 per cent it was related to previous surgery. In the same series, vascular occlusion in the young was seen more frequently following acute abdominal infection than in relation to any other condition.

According to Trotter, the commonest predisposing cause of portal thrombosis is pressure upon the vein from without. This may be exerted by cicatricial contraction of the liver capsule about the portal fissure as a result of syphilis or alcoholic cirrhosis, by primary or secondary carcinoma of the liver, by metastatic growth outside the liver, by gumma of the liver, by gall stones, by enlarged lymphatic glands about the portal fissure, and finally by fibrotic adhesions in the same region. He also states that thrombosis has been associated in a few cases with definite calcareous in the portal vein.

Donaldson and Stout consider such lesions as intussusception, volvulus, acute and chronic enteritis, important as initiating pathological status in venous thrombosis. (16)

The cases of venous obstruction that give rise to grave

clinical manifestations are almost always those presenting lesions of the superior mesenteric or portal vein. Obliteration of the inferior mesenteric vein alone, without simultaneous involvement of the superior mesenteric or portal vein, practically never leads to infarction, the reason being that the inferior mesenteric vein forms richer anastomoses with the systemic venous system relatively to the amount of blood to be carried away than does the superior, rendering possible the establishment of a more efficient collateral circulation. For this reason, if for no other, thrombosis of the inferior mesenteric vein per se is a condition of incomparably smaller clinical significance than that of the superior mesenteric. (32) (5)

The venous circulation in the intestines can be carried on even if a large part of the mesenteric venous system has undergone thrombosis. So long as tributaries that are free from clot open into the main trunk above the highest limit of the clot, and the anastomosing arcades and their radicles leading from the intestine remain patent, the intestine may remain free from infarction. The great danger in such cases lies in the extension of the clot peripherally into the anastomosing arcades and their radicles: even then the infarct is often limited to the segment corresponding merely with the occluding radicles. (76)

A mesenteric vascular occlusion most commonly involves the superior mesenteric vessels and in Larson's theories of 36 cases, the superior mesenteric vessels were involved in 35, the inferior

in 1. (46) In the Mayo series of 60 cases, the superior vessels were occluded in 57, the inferior in 3. (17)

TABLE I

INCIDENCE OF ARTERIAL AND VENOUS OCCLUSION

The average incidence of arterial occlusion is only slightly higher than venous occlusion.

Series Reported by	No. Cases	Arteries %	Veins %	Arteries and Veins %
BOYCE and McFETRIDGE	13	69.2	15.4	15.4
BRADY	12	33	67	
COKKINIS	76	25	75	
LARSON	36	39	44	17
MILLER	120	61	39	
JACKSON, PORTER and QUIMBY	197	61	39	
REICH	251	47	53	
TROTTER	360	53	41	
WARREN and EBERHARD	11	82	18	
WHITTAKER and PEMBERTON	60	31.7	45	23
TOTAL and AVERAGE per cent	1142	51.2	43.6	6

TABLE I. Comparative Incidence of Arterial and Venous Occlusion in a Collected Series of 1142 Cases.

(7)

In this paper, there will be no attempt to deal with various types of vascular thrombosis other than to mention the two main types. 1. The descending mesenteric thrombosis. This is the more important of the two and usually consists of thrombosis in the portal vein itself or the most central portions of the superior mesenteric vein following which there is a descending or spreading thrombosis from the primary site into the more peripheral branches. 2. Primary mesenteric venous thrombosis. This condition which more commonly occurs from a septic condition, in contradistinction to the descending type which usually has stasis for it's basis, occurs as a primary localized thrombosis in the most distal portions of the venous tree. (19)

ARTERIAL OCCLUSION

The superior mesenteric arterial occlusion may occur either on

(17)

TABLE III

MESENTERIC VASCULAR OCCLUSION

Comparative Involvement of Superior and Inferior Artery

Occlusion of the superior mesenteric artery is about 40 times more common than the inferior artery.

Series Reported by	Superior Artery	Inferior Artery	Both
BROWN	237	6	24
COKKINIS	19	0	0
BRADY	4	0	0
LARSON	14	0	0
BOYCE and McFETRIDGE	9	0	0
ROBEY	12	0	0
TROTTER	121	5	8
WHITTAKER and PEMBERTON	18	0	1
TOTAL	434	11	33

TABLE III. Comparative Involvement of Superior and Inferior Arteries in a collected Series of 434 Cases.

TABLE II

COMPARATIVE INVOLVMENT OF SUPERIOR AND INFERIOR VESSELS

Mesenteric vascular occlusion most commonly involves the Superior Mesenteric vessels.

Series Reported by	Superior Vessels	Inferior Vessels	Both
BOYCE and McFETRIDGE	13	0	0
BRADY	13	1	0
COKKINIS	72	4	0
LARSON	35	1	0
ROBEY	23	0	0
WHITTAKER and PEMBERTON	43	3	2
TOTAL	201	9	2

TABLE II. Comparative Involvement of Superior and Inferior Mesenteric Vessels.

(7)

a thrombotic or embolic basis. Of these, the embolic is the more frequent. According to Whittaker and Pemberton, thrombosis is more frequently associated with pathology predisposing to thrombosis such as splenic anemia and advanced arteriosclerosis and embolism more frequently associated with degenerative heart disease or acute valvular disease. (76) Arterial thrombosis is usually secondary to embolism and according to Cokkinis, primary occlusion of the mesenteric arteries is rare but all men do not agree with this. The causes in primary thrombosis reported in the literature have been found to be aneurysm or atheroma of the abdominal aorta and arteriosclerosis and atheroma or endarteritis of the mesenteric arteries themselves.

In the case of embolic closure, it was noted as early as 1900 that the superior mesenteric artery was involved many times more frequently than inferior mesenteric artery. This is explained on it's earlier exit from the aorta and also on the basis of superior mesenteric artery being more of a direct continuation from the abdominal aorta as well as being a larger vessel. It was pointed out that inferior mesenteric venous occlusion was not nearly so serious a condition as superior mesenteric venous occlusion due to the fact of it's richer anastomosis. This does not hold true for the arteries because all of the arteries are shown to be of the endartery type functionally although not completely so anatomically. (73) (58) (4) (5) (30)

The common sides of the emboli are the left auricle in mitral

stenosis and the aortic or mitral valve in ulcerative endocarditis. Occasionally thrombosis on an atheromatous ulcer in the aorta gives rise to the embolus. Rarely pulmonary vein thrombosis or thrombosis in a patent ductus arteriosus may be the origin. Laufman and Scheinberg also report the superior mesenteric arterial embolism following abdominal surgery. (49) There are also reports of embolism in the mesenteric arteries from the lungs or pulmonary veins and from the right heart via patent foramen ovale. As for arterial occlusion, there seems to be little dissension in the ranks and all of the authors seem to agree that the main causes are emboli from the left auricle usually associated with mitral stenosis and the aortic or mitral valve with ulcerative endocarditis. Thrombosis of the superior mesenteric artery most commonly follows disease or pressure on it's wall. This is in contradiction to Cokkinis. (58) (24) (73)

Arteriosclerosis or atheroma is the common lesion and therefore, it is common to find this an association with diabetes mellitus, and according to Moore, it is not unlikely that localized mesenteric thromboses account for many of the abdominal crises which occur in diabetic subjects. (58) Occasionally an aneurysm of the superior mesenteric artery itself, or of the abdominal aorta near it's exit leads to occlusion or thrombosis.

Thrombo-angiitis obliterans may affect this vessel and lead to thrombosis but this is not a common cause. There are reports of abdominal tumors pressing upon or enveloping this artery with occlusion. Of course all things which predispose to arteriosclerosis

and atherosclerosis will favor the occurrence of mesenteric arterial thrombosis. Sclerosis of the aorta which leads to occlusion is accomplished by the formation of a calcareous plate in the region of the mouth of the mesenteric artery which occludes the artery either by direct overgrowth over the mouth, or by direct spread of the thrombus into the mesenteric artery itself. In the case of aneurysm of the aorta, the obstruction has been brought about either by the spread of the fibrinous lining of the aneurysm into the mouth of the vessel, or more frequently by the gradual constriction of the origin of the arteries as they pass through the wall of the sac. (38) Thrombosis in these cases is probably due to inflammatory changes such as are known to occur about the walls of the aneurysm. Endarteritis is not considered a common cause, yet the dimunition of the blood supply associated with the narrowing of the lumen and the roughening of the intima, no doubt has some effect upon the blood stream increasing the chances of coagulation. If there is the coexistence of myocarditis in any of these conditions, tendency of thrombosis formation would be accentuated due to the associated depressed circulation. (73)

Laufman and Scheinberg found possible etiologic factor which had not been previously reported, nor have I run across a report since. In two of their cases, mesenteric arterial occlusion followed destruction of the lumbar sympathetic chain. Lumbar sympathectomy was performed in one patient while in the other, the lumbar sympathetic ganglia were paralyzed by the injection of

novacaine. They do not attempt to discuss the actual mechanism but suggest that it may be one of reflex vasodilatation or the result of an underlying predisposition to thrombosis. (49)

Intermittent claudication has also been considered a source of occlusion but it seems to be a rare occurrence. (70)

As in the case of arterial embolism, it is generally accepted that the common causes are arteriosclerosis and atherosclerosis of the aorta and the mesenteri

c arteries themselves.

TABLE I.—CAUSES OF VENOUS MESENTERIC THROMBOSIS

	Warren and Eberhard	Larson	Cokkinis	Blackburn	Total
Appendicitis	1	5	11	1	18
Enteritis	2	1		1	4
Thrombophlebitis, iliac and femoral	3			1	4
Endophlebitis obliterans			1		1
Ulcerative colitis			1		1
Diverticulitis			1		1
Abscess	1		1		2
Polycythemia	7				7
Splenic anemia and splenectomy	3				3
Hemophilia				1	1
Previous pelvic operations	6		1		7
Pregnancy					
Abortion	2			1	3
Delivery	1				1
Pressure					
Tuberculous nodes	1				1
Adhesions	1		3	3	7
Ascaris lumbricoides	1				1
Trauma	3		4	1	8
Phlebosclerosis	1				1
Constipation	1				1
Cirrhosis of liver	5				5
Portal thrombosis		4	22		26
Carcinoma of stomach	1				1
Luetic scar of liver	1				1
Unknown	35	6	13		54

(75)

PATHOLOGY AND PHYSIO-PATHOLOGY

PATHOLOGY

The pathology will be taken up under the following headings:

(1) Changes in the vessels and the distribution of the clot.

(2) Changes in the mesentery and glands.

(3) Changes in the walls and contents of the intestine.

(4) Peritoneal changes.

(1) VESSELS (A) VEINS

Thrombosed mesenteric veins are as a rule not merely filled, but distended with blood clot; and if rolled between the fingers give the impression of firm hard cords running through the mesentery. This cord-like consistence is not entirely due to blood clot in the veins, but also in many cases to thickening in the walls and inflammatory thickening in the connective tissue immediately surrounding the veins. Periphlebitis is especially marked if the thrombosis is septic or purulent.

In cases of arterial occlusion, the corresponding veins and vemules are distended with black, fluid blood. (19)

The consistence of the clot depends upon a number of factors, the age and the etiology of the process--whether (a) simple aseptic propagation from a primary clot in some other part of the portal tract, or (b) secondary to phlebitis in the thrombosed vein itself, or (c) purulent.

A simple clot recently formed in a vein is nearly black in color, elastic, non-adherent to the intima, easily withdrawn, and

uniform in consistence. As the clot gets older, it loses it's dark color till it becomes pale grey, firm and brittle, adherent to the vein-wall, and finally it may become organized and even canalised with fresh blood-channels. In some cases where the thrombosis is due to local phlebitis the clot may be stratified, the outer layers being paler than the central core; a thrombus of this description is usually adherent to the intima from the first, and may retain a central channel unobstructed. The changes that take place with increasing age are similar to those in a simple clot.

A purulent thrombus may vary in it's constitution; it may be composed almost entirely of pus transforming the lumen of the vein into an abscess cavity, or of blackish soft gelatinous material containing pus cells, or of semi-fluid blood mixed with pus. (18) (20)

DISTRIBUTION OF THE THROMBUS

Venous thrombosis in the mesentery and intestine may remain strictly localized, or it may spread from one part into practically the whole of the portal tributaries. Again it may take origin from the portal vein, and involve secondarily tributaries in different parts of the mesentery and intestine, producing several infarcts. Finally the process may start in one tributary and involve tributaries at a distance by retrograde venous embolism through the trunk and intervening veins: this is especially the case where thrombosis arises in the appendicular region and spreads into the veins of the jejunum.

As a general rule, the venous radicles draining the intestine

and the anastomosing arcades are thrombosed at a later date than the radiating larger tributaries of the superior mesentery.

If we can depend upon the incidence of infarction of the intestine, it appears that the jejunal and ileal veins are thrombosed with almost equal frequency. The venous thrombosis is generally far more extensive in any given case than can be gathered from the size of the infarcted area of intestine. (4) (18) (20) (76)

(B) ARTERIES

The alteration in the appearance of the mesenteric arteries by thrombosis is not so gross as in the case of the veins, though the arteries become firmer to the touch.

The obstruction may consist of an embolus blocking the mouth of the trunk or one of the main branches, or of a short thrombus, or of a thrombus completely occluding a considerable length of the trunk and branches. In most cases, the artery and branches peripheral to the clot, both in cases of embolism and thrombosis, are quite empty of blood. In cases of venous thrombosis, the arteries are full of blood and the capillaries distended. A recently formed thrombus is of a dull red-brown color, elastic, non-adherent to the intima, and easily withdrawn. It is uniform in consistence and not stratified. As it gets older, it gradually loses it's color, till it assumes a pale reddish grey appearance, becomes adherent to the intima, dry, granular and friable.

Apart from the history of a case, it is often impossible to say whether a clot is a thrombus or an embolus; not uncommonly it

is both, the former being grafted on to the latter. An embolus generally lodges at a bifurcation of the artery, or opposite the offset of a branch partially or completely occluding one or both branches. The injury to the intima produced by the impact may account to some extent for the supervention of thrombosis. (41) (52)

(C) EFFECTS OF BLOOD-STASIS UPON THE CAPILLARIES AND VENULES

Occlusion either of an artery or a vein in the mesentery leads to grave results in the capillaries and venules of the area affected if an adequate collateral circulation is not at once established. If the collateral venous circulation is inadequate, there is produced an engorgement of a stagnant blood with ever increasing tension which soon forms an insuperable barrier to the blood in the capillaries. The arteries of the area continue to bring blood, distending the capillaries and stagnating, it alters the vascular walls and produces an infarct. The effect of an obliteration in an artery is the same, though brought about in a different way. The sudden removal of the force leads to a retrograde venous engorgement, affecting the vessels as far back as the point of obstruction. The immediate consequence of the raised blood pressure combined with stasis and therefore carbonic acid poisoning, is irreparable damage to the delicate walls of the capillaries and venules which consist merely of a single layer of endothelial cells. Extravasation of serum and blood follows, leading, according to the severity of the lesion, to edema, ecchymoses, hematoma, or hemorrhagic infarct and gangrene.

In a few cases, the process has been severe but localized and has led to perforation from complete necrosis of the whole thickness of the wall of the intestine. (59) (71)

(2) MESENTERY AND GLANDS

In the vast majority of cases, gross vascular occlusions in the mesentery give rise to edema in the affected area. This is one of the first changes and one which may advance to an extreme degree producing enormous thickening. The mesentery may measure anything from $\frac{1}{8}$ to $1\frac{1}{8}$ inches in cross section. If the edema is localized, it may simulate a glandular enlargement with periadenitis, the fat and connective tissue being firm, hard and inelastic. With the increase in thickness, there is a corresponding diminution in mobility of the mesentery and this in itself may be a factor in the production of intestinal obstruction which so commonly follows in these cases. (21)

Extravasations of blood are often found in the mesentery in the region of occluded vessels. They vary in size from small petechiae and ecchymoses to large collections of blood capable of being felt through the abdominal wall. In cases of arterial occlusion, the hemorrhages are probably due entirely to venous back-flow and not, as it has been supposed by some authors, to a collateral arterial supply.

The collateral arterial supply in the mesentery is generally very meager, and the point has been emphasized by Taravellier that were the anastomosis responsible for the hemorrhages, one would

expect them to be most marked at the boundaries of the affected area. In actual fact, the hemorrhages are most intense about the obstructed vessels where compensation is least. (53)

As the process of infarction advances, the mesentery becomes darker in color till it may be almost black, the serous coat loses it's gloss and tears easily, and the fat, connective tissue and blood vessels become necrotic. The demarcation of the infarct from the healthy parts on either side may be sharply defined or gradual, whether the casual lesion is arterial or venous, or both arterial and venous together. In Harkin's series he noted abrupt changes in the colon at the proximal end, but gradual changes in shading at the distal end in all his cases. (32) Elliot reports diffuse shading at both ends in his series of cases. (24) In contrast to Harkins, Reich reported abrupt changes at the distal end and gradual shading at the proximal end of the diseased bowel segment. (10)

It might be expected that in cases of arterial occlusion the demarcation would be sharper than in those following venous obliteration, owing to the different capacities for compensation in the two systems, but in view of the uncertainty and scantiness of the data at our disposal, no definite rule can yet be made. In all the cases of anemic infarct, the anemic areas have been sharply defined.

In a few cases of septic venous thrombosis, abscesses have been found in the mesentery. In one case of arterial thrombosis, the mesentery contained gas vesicles along the line of the intestinal

attachment, while in another they were present in this position and also beneath the intestinal mucosa.

The mesenteric lymphatic glands are frequently described as being much enlarged, and they often contain extravasations of blood. At later stages of the disease, they undergo the same necrotic changes as those described above. (1)

(3) INTESTINES AND THEIR CONTENTS

There are two main types of lesions that result in the intestines following mesenteric vascular occlusion:

(1) Hemorrhagic Infarction

(2) Anemic Infarction

HEMORRHAGIC INFARCTION

This may be brought about by closure of an artery, or vein, or both and may involve from a few centimeters to any length of intestine. Jerauld and Washburn report a case involving 580 cm. which is 90 per cent of the small intestine and Munro reports a case following enteric fever involving the whole of the small and long intestines. (40)

The condition of the infarcted bowel closely resembles that of a strangulated hernia and displays all the variability of a strangulated hernia depending upon the degree of vascular occlusion. Therefore, the condition varies from simple edema and engorgement of the intestine, or infarction, or finally gangrene.

In hemorrhagic infarction the affected coil is dark red, purple or blue black and may be mottled with greenish patches.

The bowel wall is usually much thickened from infiltration of it's walls with blood, serum and inflammatory cells and is usually distended from gas in the bowel. The serous coat may retain it's gloss until a late stage but usually becomes dull early. The bowel may be covered with shreds of fibrin and forming adhesions with adjacent structures. The mobility of the affected bowel is either seriously impaired or absent, especially if peritonitis is present. Jackson, Porter, Quinby and Munro report cases lacking these changes in the bowel wall. (38)

The involved mucous membrane is red, hemorrhagic and swollen, not showing ulceration. A few authors report cases with ulceration and these vary with the degree of involvement. Lauer and Glazer, by use of microscopic section, showed their cases exhibited no evidence of ulceration of the mucous membrane even though the other signs were present. (50) When the wall of the infarcted coil is cut through, it is usually much thickened, black and edematous and all of it's layers are engorged with blood. The infarcted intestine generally contains dark, offensive, semi-fluid material which is either streaked with blood, or consists of almost pure blood. The latter may be partly clotted or of tarry consistence, or it may be dark red and liquid. Free blood may be present in the lumen of the intestine in cases where none has been passed per rectum or been seen in the vomited matter; this is probably due to local paralysis of the intestine at the site of the infarct. In the infarcted coil, there is generally an accumulation of gas, which in

the normal state would have been absorbed by the veins. (40) (11)

ANEMIC INFARCTION

Anemic infarction is seldom encountered and is always on an arterial occlusion basis or occasionally a combined occlusion, but even most arterial occlusion includes hemorrhagic infarction. (58) (26) The bowel wall in the affected area is of a white or yellowish grey color and is devoid of blood. The ends of the affected segment are usually demarcated from the healthy intestine by a zone of hyperemia and constriction. The wall is generally very thin but a few cases show an increase in thickness.

The most characteristic thing about the bowel contents is the absence of blood. Some blood may be present if there is much engorgement of the proximal and distal connecting segments.

(4) PERITONEAL CHANGES

The usual finding upon opening the peritoneal cavity in a case of hemorrhagic infarction is a quantity of free blood or bloody peritoneal fluid that has been extravasated from the mesentery and intestine. This will vary from a few cc. to several liters in different cases. In cases of peritonitis, there may be pus mixed with the fluid and the blood may even be clotted.

Peritonitis is frequently found in cases of mesenteric vascular occlusion even when there is no perforation of the bowel, the bacteria passing through the affected and infected bowel wall along with the seepage of blood and not infrequently free gas is found in the peritoneal cavity and is usually described as of a

fetid odor. Perforation is rare but Harkins and Elliot do not consider it so uncommon. (24) (31) (14) (32)

PHYSIO-PATHOLOGY

No matter what the cause of the occlusion, results will depend upon several factors.

(1) Whether the occlusion is arterial or venous.

(2) The caliber of the vessel involved.

(3) The suddeness of the occlusion.

(4) The length of time the occlusion has existed before surgery or death.

(5) The length of intestine affected, or the position in the vascular tree of the occlusion.

The absence or small amounts of peritoneal fluid is usually indicative of a mild degree of occlusion but this is not always true and Harkins cites a case with extreme infarction with less than 250 cc. of fluid. (32)

ARTERIAL COMPARED WITH VENOUS OCCLUSION

As was pointed out in the anatomy, the superior mesenteric artery behaves like an endartery even though it is not strictly such anatomically.

Sudden occlusion of the main artery experimentally results first in violent spastic contractions of the entire small intestine and part of the colon, from the ligament of Treitz to the middle of the transverse colon. This reaction is interpreted as an anoxic response to the sudden cessation of arterial blood flow. The gut is firm, white and acquires a rippled appearance as a result of the

contraction of the circular and longitudinal muscular coats. The intramural vessels are also compressed and the capillary blood is drained off by the veins. As the intestinal musculature becomes fatigued or toxic, it loses it's contractability and gradually relaxes. Some portions of the musculature fatigue more quickly than others, so that after three or four hours the bowel acquires a spotty appearance. Bluish areas appear between areas which remain blanched. After four to eight hours, the entire musculature relaxes and the bowel wall gradually becomes blood soaked. As the musculature of the intestine relaxes the negative pressure created in both the arteries and veins is sufficient to draw blood back into the wall of the intestine. (76)

Sudden occlusion of the superior mesenteric vein experimentally, immediately causes a bluish discoloration of the entire small intestine. The intestine does not become spastic but is relaxed. The course of such an animal is practically identical to that of an animal which is bled to death. The blood pressure slowly drops until death occurs. The blood volume is ordinarily reduced 50 to 60 per cent and death occurs within two to twenty four hours. (1)

Scott and Wangensteen have found that in dogs the average length of life following venous ligation was five and one-half hours; following arterial ligation twenty hours, and following ligation of both the superior mesenteric artery and vein, nineteen hours. (69)

CALIBER OF THE VESSEL INVOLVED

In the dog, occlusion of the vessels of the second mesenteric

arcades causes no circulatory embarrassment of the intestine. The injection experiments of Cokkinis performed on cadavers showed that this is also essentially true in the human. Eisberg has shown that there is a considerable decrease in the margin of safety following ligations of the first arcade or vasa recta. Ligation of the main mesenteric arteries or the colic vessels, or an oblique ligation of the second arcade which includes the terminal arch and vasa recta, has no margin of safety. (22)

SUDDENESS OF THE OCCLUSION

When a thrombus forms very slowly, collateral circulation has an opportunity to carry the blood supply. Warren and Eberhard have pointed out that a very gradual occlusion of the main vessel may, in some instances, result in infarction; in other instances there may be temporary symptoms of pain and intestinal hemorrhage; it may even result in no symptoms at all if it forms slowly enough. (75) Laufman has produced slow occlusion experimentally by placing a loop of cellophane snugly about the superior mesenteric artery in dogs. After about four months the animals died from inanition and anemia. Examination of the bowel revealed a low grade enteritis with erosion of the villi and leukocytic infiltration of the remaining mucosa and submucosa. There was no infarction, but the subserosal vessels contained thrombi. The cellophane had gradually constricted and had produced sufficient fibroplastic reaction to choke off the vessel. The lesion in the bowel was apparently the result of chronic anoxia. (49)

LENGTH OF TIME OCCLUSION PERSISTS BEFORE OPERATION OR DEATH

The onset of gangrene depends upon several factors. For example, if depletion of the blood volume occurs rapidly as in massive venous occlusion, death occurs before gangrene can supervene. When a shorter segment of intestine is involved, the blood loss plays a less important role and the deprived intestinal wall becomes ready ground for bacterial invasion. Thus, gangrene is more apt to be found if a short segment is involved, and death occurs from perforation and peritonitis. It is, therefore, clear that the length of time an occlusion persists before operation or death is not the sole factor in the production of gangrene. On the other hand, if the patient lives sufficiently long after the onset of hemorrhagic infarction, no matter what the cause, gangrene of the intestine will eventually set in.

LENGTH OF INTESTINE AFFECTED BY THE OCCLUSION

In short segments, the circulation is preserved by blood entering at each end through the intramural channels. According to Eisberg, this circulation is effective only for a distance of 5 cm. According to Donaldson, a somewhat longer segment of intestine can survive following venous occlusion alone than following arterial occlusion. (16)

As has been pointed out obstruction of the mesenteric or portal vessels have been shown to be accompanied by an effusion of blood and bloody fluid into the peritoneal cavity, into the tissues supplied by the vessels in question, and into the lumen of the involved gut. These same conditions have been shown ex-

perimentally by various workers. This effusion, in many instances, has been large enough to account for death on a basis of secondary shock, or shock due to loss of plasma or blood from the circulating blood.

Boyce and McFetridge report clinical observations concerning loss of blood or plasma resulting in secondary shock following mesenteric vascular occlusion. They point out the necessity for repeated blood pressure readings and hemoconcentration readings. They state, "The state of shock characteristic of this condition is rarely evident immediately as the duration of the disease increases however, and more and more blood is lost into the mesentery, especially if the occlusion is venous, the blood volume decreases and the typical symptoms of shock appear, a subnormal temperature, falling blood pressure, a fast, thready pulse, clammy perspiration and marked pallor." They point out that mesenteric vascular occlusion is the most fatal of most abdominal emergencies. (50)

Harkins, in reporting his series, also stresses repeated blood pressure and hemoglobin readings. He cites one of his cases in which hemoglobin readings were taken and he reports a hemoglobin reading of 143 per cent and hematocrit of 61 and a blood pressure of 94/70 three hours before death. (32)

Claude Bernard, in 1859, studied vascular occlusion experimentally on dogs but he was unable to explain the resultant death. He believed that death was from some other cause than bloody engorgement of the intestines. Following more experiments in 1877,

he believed that the local accumulation of blood was the cause of death after all. Von Tappeiner, in 1837, performed experiments with results in opposition to the exsanguination theory. (7)

In 1913, Neuhof, also working with dogs, found that death always occurred between 50 and 90 minutes following ligation of the portal vein and at autopsy, there was a bloody peritoneal exudate. The bowel and mesentery were always hemorrhagic and edematous. The spleen was always enlarged and the lumen of the gut contained blood. From this he concluded that death was due to secondary shock. (63)

Elman and Cole reported a series of experiments on ligation of the mesenteric vessels. In these experiments, the entire intestinal tract was removed, following death, and weighed. These weights were compared with the normal control series. The average increase in weights was 5.2 per cent of the body weight. In another series, the increase averaged 3.4 per cent and in a control series, death followed after a 2.7 per cent blood loss following hemorrhage from the femoral artery. (25) (26)

In opposition, Boyce, Lampert and McFetridge present experiments showing the average weight increase following ligation of the portal vein to be 3.5 per cent of the body weight and in the control series, dogs were bled an average of 4.56 per cent of body weight without shock or death. Therefore, they concluded that the loss of blood is insufficient to cause death. Although they believe that the blood loss is insufficient to cause death, they agree that the

blood loss is an important factor. They also report a neurogenic factor, as in primary shock, is the initiating factor and that the blood loss only accentuates and continues the low blood pressure due to the primary shock. (8) (9)

We should remember, in comparing these results and their conclusions as to the theories of the cause of death, that the 3.5 per cent loss of fluid reported by Boyce, Lampert and McFetridge does not represent pure blood but more plasma than cells; and it is well known that it is less serious to lose a certain amount of whole blood than a similar amount of plasma. Johnson and Blalack report the death of dogs after an average loss of 2.5 per cent loss of body weight in blood plasma. (42) Besides the loss of fluid into the peritoneal cavity, bowel and mesenteries, important amounts can be lost by the splenic enlargement and especially by vomiting and diarrhea. (69) (16) The amount of blood and fluid lost into the affected areas have been shown to be sufficient to cause death in the experimental animals and also in clinical cases. However, in many cases it is quite certain the fluid loss is not the cause of death. We must not underestimate, as a result of these reports, the role played by secondary peritonitis and intestinal obstruction in the cause of death. (62) (63) (64)

SYMPTOMS AND SIGNS

Nearly all cases of mesenteric vascular occlusion present an acute onset of symptoms, according to most authors, but a very few report what they term a chronic onset. Arterial occlusion

usually presents a more acute onset than venous occlusion. Venous symptoms, according to Larson, are more likely to be vague and interwoven with symptoms of the preceding disease. (46)

According to some writers, there is a more or less specific symptom pattern for mesenteric vascular occlusion, but this is not universally agreed upon. Ross, Whittaker and Pemberton express the opinion that the symptoms of mesenteric vascular occlusion are those of acute intestinal obstruction, strangulation, rupture of a viscus or peritonitis. (16) (5)

Laufman and Scheinberg agree with these opinions. (49)

In reading through the articles on mesenteric vascular occlusion, it has been my observation that practically all of the writers agree upon about the same symptoms and signs; but that there is a slight difference of opinion as to the degree and importance of the various symptoms and signs. These symptoms and signs in themselves are not diagnostic, and can belong to any one of various diseases.

A complete list of symptoms and signs are as follows:
Pain, vomiting, constipation, diarrhea, shock, fainting, distention, flatus, temperature changes, pulse changes, blood pressure changes, pallor, cold sweat, abdominal tenderness and rigidity, dehydration, palpable mass, ascites, peristaltic sounds, rectal examination findings and facies. These will be discussed individually later.
The average duration of symptoms in Harkin's series was eight days, while in the series reported by Jackson, Porter and Quinby, the

average duration was three days. In Trotter's cases, 76.6 per cent of the arterial cases and 54 per cent of the venous cases were of less than seven days duration.

PAIN

Pain is present in almost every case and is usually the paramount symptom. The disease is usually characterized by sudden onset of central abdominal pain, according to the majority of writers, but some men describe the pain as being slow or late in onset. This is probably more true in venous occlusion. Pain as the first and most constant symptom was found in 57 out of 62 cases of Cokkinis' and in 26 of 33 cases of Blackburn's series and in 85 per cent of Reich's series. (5) Meyers reports severe pain in 100 per cent of his series of cases. (43) As to the character of the pain, there is considerable difference in opinion.

According to Bowen, the pain is usually very intense and agonizing at first and later becoming colicky, and with the further development of peritonitis, the pain becomes constant. (11)

Laufman and Scheinberg and others report that there was no uniformity in the character of the pain and that almost every variety of abdominal pain may be manifested and from this they conclude that the character of the pain is of very little value in diagnosis. They believe that the most constant feature of the pain is it's intensity and constantness and that it usually overshadows the associated vomiting and the pain usually persisted after antispasmodics or morphine were given. They point out that,

on the other hand, the vomiting is the prime symptom in ordinary bowel obstruction. They also report that the variability of the pain was responsible for their incorrect diagnoses of ruptured appendicitis, intestinal obstruction, ruptured peptic ulcer, peritonitis, cholelithiasis and ureterolithiasis. (49)

Whittaker and Pemberton believe that the severe pain is usually preceded by a dull ache which is most frequently constant and intermittent. (76)

Trotter believes that the pain, as well as the other symptoms, is much the same whether of venous or arterial origin. He also points out that the pain is usually acute and of sudden onset, although the conditions which led up to it may have been present for years. (73)

It is frequently pointed out that the severity of the pain is not in proportion to abdominal tenderness and rigidity. (67)

There is nothing characteristic about the location of the pain as it may be localized in any part or parts of the abdomen, or may be diffuse.

VOMITING

Vomiting is probably the second most constant symptom. It is usually preceded or accompanied by pain but may occasionally precede the pain. Vomiting is usually a marked feature from the beginning. (58)

Mallory reports a case in which he resected four feet of intestine without vomiting and in Gambee's case, involving the in-

ferior mesenteric vessels, vomiting did not occur. (54) (27)

Such reports as the above mentioned, represent uncommon and isolated cases, and vomiting in the large majority of writers occurred in from a low of 50 per cent to a high of 100 per cent of their cases.

Cokkinis reported vomiting in 47 out of 62 cases. The Mayo series report vomiting in 75 per cent of arterial cases and 50 per cent of venous cases, and it is the common opinion that vomiting is more frequent in arterial occlusion.

Meyer noted vomiting in 55 per cent of 92 cases of mesenteric vascular occlusion. (57)

Brown, in his attempt to differentiate between arterial and venous occlusion, believes that in a pure venous lesion this symptom should occur less frequently than in arterial occlusion. (11)

Warren and Eberhard place special diagnostical importance on vomiting and according to them, there is a relationship between vomiting and the prognosis. They believe that with less vomiting, the prognosis is better. They point out that when vomiting occurs late, it is likely to indicate a high jejunal lesion and this type is not so successfully treated by surgery. (75)

Boyce and McFetridge found that vomiting usually gets less after six to eight hours, especially when the intestines are emptied. They also found reverse peristalsis above the lesion and the involved area to be inert. (9)

The vomitus at first contains the stomach contents and per-

haps bile, but if continued usually becomes the "intestinal" type and contains blood. "Coffee ground" vomitus is common and if the vomiting is continued further, it may contain fecal material. This may lead to erroneous diagnosis of gastrocolic fistula. (24) (75) (58) (47) (55) (9) (57) (54) (5) (27)

CONSTIPATION AND DIARRHEA

These are probably the next most constant symptoms. Constipation or diarrhea may occur singly or both may be present alternatingly.

Here most writers argue that constipation is more frequent than diarrhea, but Reich reported diarrhea in 41 per cent of all cases of mesenteric vascular occlusion. Laufman and Scheinberg believe that the occurrence of constipation and diarrhea are inconstant and so evaluate them accordingly. Yet, they report 41 per cent showed constipation and 11.3 per cent diarrhea. (49) Trotter's series of 366 cases showed constipation in 209 cases, diarrhea in 87 and alternating constipation and diarrhea in only 19 cases. In contrast, Meyer reported only 10 per cent of his cases showed diarrhea in both arterial and venous mesenteric occlusion. (73) Reich reports a 41 per cent incidence of diarrhea.

Boyce and McFetridge believe that tenesmus is the most usual form of bowel function disturbance.

As for the stools passed, they may be simply blood stained or show large quantities of blood; either clotted or liquid; or black and tarry. A few cases of discharge of almost pure red

blood have been reported. (73) Defecation is commonly very painful and as noted before, associated with tenesmus.

Flatus is common.

Donaldson and Stout found blood present in the stools in 100 per cent of cases and found the stools to be dark and tarry and soft to liquid in consistency. (67) In Laufman and Scheinberg's series, occult blood was also found in 100 per cent of cases but not gross blood. 65 per cent of these cases were mesenteric venous occlusion. (49)

In the Mayo series, blood was also more commonly found in cases of venous occlusion but was found in only 37 per cent of the cases. In arterial occlusion, they reported only 12 per cent with melena. (5) Blackburn reported melena in 14 out of 20 cases, Reich in 26 per cent and Jackson, Porter and Quinby in 41 per cent. (10) (5) (38)

SHOCK

It was hard to decide between constipation and diarrhea, and shock as to their prominence in the symptomatology of mesenteric vascular occlusion. The reports show them to be nearly equal in occurrence but most men placed constipation and diarrhea first.

Some degree of shock is present in nearly all cases with an acute onset, according to Bowen. (7) In the Mayo series, shock was present in 47 per cent of arterial cases, only 2 per cent of the venous cases and 36 per cent of combined cases. (5) In Laufman and Scheinberg's series, shock was present in 45.4 per cent

of all cases and they also showed a higher figure for arterial occlusion and shock was more pronounced than in other abdominal emergencies. (49) Brown, Boyce and McFetridge found that shock was marked, especially in fulminating cases and was proportional to the degree of mesenteric involvement. (11) (9)

DISTENTION

This symptom, according to most authors appears late and is not relieved by defecation or enemata. Brown believes it to be a serious prognostic sign. (11) Again referring to the Mayo series, it was present in 48.3 per cent and was more common in the venous series. (5) Jackson, Porter and Quinby found distention in 78 per cent of his cases, Blackburn in 27 out of 36 cases and Cokkinis in 65 per cent. (5) (38) (5) Laufman and Scheinberg report only 30 per cent of their cases showed distention and of these, 9 per cent were arterial and 23 per cent venous. (49)

As the statistics show, distention is commonly a symptom but the fact that it is a late symptom reduces it's value materially as a diagnostic aid.

RIGIDITY AND TENDERNESS

In the occurrence of rigidity and tenderness, there is considerable difference of opinion.

Trotter, in his series of 366, reports that there is usually extreme tenderness and rigidity of the abdomen. These signs of course depend greatly upon the duration of the disease, it's acuteness and peritonitis. (73)

Jackson, Porter and Quinby reported the complete absence of rigidity and tenderness in 30 per cent of their series. (38)

Laufman and Scheinberg were impressed by the fact that in their series, the degree of distention was almost constantly out of proportion to the severity of the pain, in that the rigidity and tenderness were moderate. (73) Brown agrees with Laufman and Scheinberg that the tenderness and rigidity is much less than would be expected in proportion to the pain. He also points out that rigidity is generally lacking in early cases. (7)

In the articles reporting individual cases, most men believe that rigidity and tenderness are out of proportion to the pain.

In regard to the location of the abdominal rigidity and tenderness, there seems to be no consistency. The tenderness and rigidity may shift or be generalized. The location will depend upon the localization of the diseased area and it's pain to a great extent, and since this is inconstant it results in an inconstant location of the rigidity and tenderness. There is a slight preponderance of rigidity and tenderness in the epigastric and umbilical region, and least of all in the flank. (6) (41) (74) (66)

PALPABLE MASS

This is an infrequent and unreliable sign and seems to be of small value in diagnosis. The mass, when present, is due to the swollen, infarcted coils of intestine and the thickened edematous mesentery.

A palpable mass was present in 4 out of 76 of Cokkinis'

cases and in 11 out of 360 cases in Trotter's series. Laufman and Scheinberg report a palpable mass in 27 per cent of their cases and this was the highest figure encountered.

PERISTALTIC SOUNDS

As a sign in mesenteric vascular occlusion, very few men mention peristaltic sounds but findings of increased visual and auditory peristalsis were reported. These sounds are not as loud and gurgling as in cases of mechanical obstruction. The presence and degree of peristaltic sounds depends upon the extent of bowel affected. The more affected, the less the peristaltic sounds since, as was pointed out before, the affected area is inert. (40) (35)

ASCITES

As for ascites, most writers failed to mention or place much importance on this sign but Cokkinis found it present in 18 out of 22 cases and believes it to be an important clue in diagnosis. Moore and Loop also believe that some degree of free fluid can usually be found. (48) (58)

OTHER SIGNS AND SYMPTOMS

On examination, especially in the acute form of the disease, the general appearance of the patient may be striking. He is usually pale and restless, cold and clammy with a subnormal temperature and a fast, thready pulse, in other words showing signs of the previously mentioned shock. He may be faint and marked dehydration is common, especially with vomiting and diarrhea.

A few cases will show an elevated temperature but most of them will be subnormal. (76) et. all.

Hiccoughing may be seen but is not common. When it is present, it is a source of a great deal of distress and fatigue. It is really a symptom of the onset of peritonitis rather than mesenteric vascular occlusion itself. (65)

LABORATORY FINDINGS

1. Leukocytosis

It was usually found that the leukocytosis was out of proportion to the apparent severity of the disease. In the series of Laufman and Scheinberg, the white blood counts were between 15,000 and 30,000 in most cases, but in considering all of their cases, there was an extremely wide variation from 4,000 to 90,000. The neutrophils numbered over 80 per cent in most cases. They also emphasized the occurrence of not too uncommon cases in which the white blood count never exceeds 9,000 or 10,000; and also the early cases in which the white blood count has not yet begun to rise. Other writers also noted occasional cases showing leukopenia and Hill, Stover, Harris and Feldhym attempt to explain this as a "leukocytic exhaustion" on the basis of absorption of toxic products from the involved intestine. (36) (33)

Several writers point out that the leukocytosis is usually higher in mesenteric vascular occlusion than in other acute abdominal diseases.

Referring back to the Mayo series it was found that in

arterial occlusion the average white blood count was 27,000 with a high of 35,000, while in venous occlusion, the white blood count averaged 20,000. All of these reports, upon the differential counts, were constant and in agreement with Laufman and Scheinberg in that the preponderance of cells were neutrophils.

The use of xray in the diagnosis of mesenteric vascular occlusion has proved to be of little value. In early cases, nothing is noted, but in a small percentage of late cases, the loops of a distended intestine may be shown. In the cases with ascites, a fluid level is usually demonstrable. (64) (3)

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

DIAGNOSIS

Mesenteric vascular occlusion gives rise to lesions that are extremely difficult to recognize and, according to the literature, it is uncommon for this condition to be correctly diagnosed. This is shown by the fact that in Trotter's series, only 4 per cent were correctly diagnosed. This is a very low figure and since that time, higher figures are constantly appearing, but even now the best figures are relatively low. The fact that the earlier figures were extremely low is probably due to the fact that at that time (1913) mesenteric vascular occlusion was a relatively new disease and very uncommonly encountered, and so was very rarely suspected. Since that time, more cases have been reported and discussed, and somewhat of a symptom pattern worked out; also more men are conscious of it's possibility, and place it in the differential diagnosis

of the acute abdomen.

According to the vast majority of writers, the symptoms and signs of mesenteric arterial occlusion and mesenteric venous occlusion are so similar that it is almost always impossible to distinguish clinically between the two lesions. Even though Gerhardt and Kussmaul (quoted by Trotter) laid down certain postulates for diagnosis in cases of mesenteric embolism, it is doubtful if they are even all fulfilled in one given case. These postulates are enumerated as they may form a valuable guide to diagnosis.

1. A source for the embolus (e.g. endocarditis).
2. Copious melena, not accounted for by any portal obstruction (e.g. hepatic cirrhosis) or lesion of the intestinal mucous membrane.
3. Rapid and excessive fall in temperature.
4. Very severe colicky abdominal pains.
5. Later, distention of the abdomen with tympanites and shifting dullness.
6. Earlier or simultaneous embolic phenomena in other parts of the body.
7. Often large hematomata between the layers of the mesentery, felt through the abdominal wall.

Dunphy and Whitfield believe that a typical syndrome does not exist for mesenteric vascular occlusion; but under the following conditions mesenteric vascular occlusion must be considered as the most likely cause.

1. Severe abdominal pain.
2. A scarcity of physical findings so that the picture as a whole is confusing and does not fit any of the usual surgical emergencies.

3. A high leukocytosis and high pulse rate with a relatively low temperature.

4. Abdominal tenderness, more or less generalized.

5. Evidence of a gastro-intestinal disturbance manifested by either vomiting or diarrhea or signs of subacute obstruction. (19) (20) (18)

The above are mentioned very commonly by a high percentage of the other writers and the additions made to these five findings are:

1. Especially when blood is found in the stools.

2. The presence of a possible predisposing disease such as cardiac valvular disease.

3. History of preceding surgery, especially appendectomy, splenectomy, or pelvic surgery.

4. Any of the etiological factors mentioned in the chapter on etiology.

Moore believes that the administration of an enema along with a bloody return is almost diagnostic; and Cokkinis also emphasizes the importance of a diagnostic enema, and believes that the most important single clinical feature of mesenteric vascular occlusion is the presence of intestinal hemorrhage, which because of the superimposed paralytic ileus is not always spontaneously evacuated per rectum, but which an enema will frequently wash down. (58)

Trotter says that if a clinical picture suggesting mesenteric vascular occlusion occurs in an older patient with arteriosclerosis, one should think of mesenteric embolism but it seems as though with arteriosclerosis, thrombosis would be more likely to occur, especially if he had been subject to attacks of intermittent mesenteric claudication or abdominal crises associated with angina pectoris or chronic nephritis. (73)

DIFFERENTIAL DIAGNOSIS

As was mentioned before, the differential diagnosis between arterial and venous mesenteric vascular occlusion is in the vast majority of cases impossible and is relatively unimportant since the treatment in both conditions is the same.

Features noted by several authors are:

(1) Regardless of the duration of the attack, the clinical picture has not been typical of the common surgical emergencies. The localized tenderness of appendicitis, rigidity of the perforated ulcer and the visible peristalsis and early distention of mechanical intestinal obstruction are not present. The patients always appear sick from an abdominal lesion which simulates, but is not typical of obstruction.

(2) Pain is out of proportion to the clinical findings and persists after the administration of the ordinary pain relieving measures.

(3) The only constant finding is deep abdominal tenderness, more or less generalized with rebound tenderness. In the later stages, the signs of generalized peritonitis develop but these should not be considered essential in early diagnosis.

Mesenteric vascular occlusion is most frequently diagnosed as acute intestinal obstruction and this is to be expected since the pathology of the two lesions is similar, that is interference with circulation, congestion, hemorrhagic infiltration and gangrene. As for the specific conditions confused with mesenteric vascular

occlusion, the most common ones are:

1. Intussusception

Intussusception is very liable to be confused with infarction of the intestine, especially when the infarcted coil can be felt through the abdominal wall; there is absolutely no pathognomonic sign whereby intussusception can be excluded. It has been suggested that the variability in the consistence of the tumor on palpation is a point in favor of the latter, but the abdomen is generally too distended or tender in infarction for this sign to be of much value. Although in many cases it is impossible to make a differential diagnosis, yet the following points would be of value as a guide:

(1) The age of the patient. Infarction though it may occur at any age is more prone to affect adults between the ages of 30 and 70; acute intussusception on the other hand is a disease of childhood, though it may affect adults.

(2) A history of one or more previous attacks characterized by marked tenesmus, and the passage of blood stained mucus by the bowel would be in favor of acute intussusception; in infarction the amount of blood and fecal matter should exceed that passed in acute intussusception, except in those very rare cases where obstruction occurs directly after the onset, while the passage of mucus is not common in infarction; tenesmus again is not so constant or prominent a symptom of infarction as of the other disease.

(3) Complete remission of the symptoms between the attacks is

a characteristic of acute intussusception which is not shared by infarction; in the latter disease, after the onset, it is most exceptional for there to be a period of complete freedom from symptoms, though the pain often becomes less severe for a time; the distinction from acute intussusception may become impossible, however in cases of infarction that have been preceded by one or more severe attacks of intermittent mesenteric claudication.

(4) If on abdominal examination, a sausage shaped mass is felt, one end of which is palpable per rectum, intussusception is more probable than infarction. (12) (35) (37) (65) (23) (13) (51) (41) (14) (29)

2. Volvulus

Though the symptoms and signs may be the same in volvulus as in infarction, yet there are certain manifestations of the former disease that differ from those which are more often met with in the latter. Absolute constipation associated with early extreme abdominal distention are signs that would favor a diagnosis of volvulus; for as we have seen, it is extremely rare for absolute constipation to occur at the onset in cases of infarction, and though distention appears early, it does not reach the same degree as it usually does in volvulus till the later stages of the disease. A history of one or more previous attacks would favor a diagnosis of volvulus, but it is important to remember that cases of chronic portal obstruction leading as a terminal event to descending

mesenteric venous thrombosis with infarction may present histories of melena, hematemesis, acute abdominal pains and perhaps absolute constipation.

Several authors have noted that in volvulus of the sigmoid, it is impossible to introduce more than a small amount of fluid per rectum, whereas in infarction, there is no such limitation. (15) (43)

3. Carcinoma of the Colon

This may present a clinical picture very hard to distinguish from infarction, but in obstructive carcinoma, there is generally a more definite history of gastro-intestinal disturbances associated with wasting. On the other hand, cases of arterial thrombosis due to arteriosclerosis and of venous thrombosis secondary to portal obstruction may give histories of recurring or chronic abdominal symptoms. A point which may be of value in the diagnosis is the absence of visible peristalsis in infarction and it's occurrence in chronic intestinal obstruction from any cause.

4. Internal Strangulation

The symptoms and signs of internal strangulation are practically the same as those of infarction. It is only by taking into account coexisting morbid conditions, as for example a valvular cardiac lesion, an infective process in the pelvic organs or appendix region, a chronic arteriosclerosis, a chronic alcoholic cirrhosis of the liver or a syphilitic perihepatitis, that any clue can be obtained as to the nature of the case.

According to some authors, in internal strangulation evacuations per annum end suddenly, and that hemataemesis is extremely rare and for these reasons they believe that there should be little confusion in the differential diagnosis between the two conditions. Most men will not accept this because of the inconstant manifestations of mesenteric vascular occlusion. They point out that hematemesis may be entirely absent and that absolute obstruction may appear at any time in this disease.

Borszeky is of the opinion that a differential diagnosis is in most cases impossible.

5. Acute Pancreatitis

Acute pancreatitis usually is associated with a higher temperature, and vomiting is a much more prominent symptom. The pain of acute pancreatitis is more easily controlled; and according to most authors, signs of early peritonitis or peritoneal irritation are more striking in cases of pancreatitis than in cases of mesenteric vascular occlusion. In acute pancreatitis, the physical signs are more localized in the upper abdomen in most cases. It is pointed out that acute pancreatitis is more commonly found in obese patients with a history of a heavy meal, alcoholic excess, gall stones, recurrent digestive disturbances or chronic cholecystitis. A very important clue as to diagnosis can be found in the blood amylase. In acute pancreatitis, the blood amylase is usually considerably elevated. Many times a tender swelling can be felt in the epigastrium and in cases of acute pancreatitis, bloody stools are not

common.

6. Perforated Ulcer

In cases of perforated ulcer, there is usually a history of gastro-intestinal or "typical ulcer" symptoms. The abdominal signs of perforated ulcer are usually striking. There is usually "board like" rigidity and increased local tenderness. Profuse oral hemorrhage is not common in perforated ulcer. Xray will commonly show the existence of pneumo peritoneum. Bloody diarrhea is uncommon in cases of perforated ulcer.

7. The differential diagnosis of pathological complications which may follow an attack of appendicitis and give rise to acute intestinal obstruction may be extremely difficult. The acute obstruction in such cases is the result of toxemia, peritonitis, adhesions, or infarction of the intestine from venous thrombosis. In the case of the first two complications, the symptoms usually merge into those due to the primary lesion, whereas adhesions or infarction manifest their presence as a rule after an interval of variable duration. The temperature in peritonitis is more often raised than in infarction, though if the peritonitis is generalized, the temperature is often normal or subnormal as in infarction. The pulse is rapid and often irregular in both, but in infarction it tends to be thready on account of the loss of blood. The pain and constitutional symptoms associated with kinking of the intestine from adhesions are generally much less severe than those produced by infarction.

The clinical picture presented by the sudden extension of an abscess arising from a gangrenous appendix sometimes resembles very closely that of mesenteric venous thrombosis originating from the same cause. In a case of the ruptured appendiceal abscess, there is usually a history of appendicitis and if there is vomiting, the vomitus is usually less colored and probably will not contain bright red blood.

8. The distinction between mesenteric venous thrombosis and a bad attack of hemorrhage in hepatic cirrhosis may be difficult, but in the former, the manifestations are much more intense and the general state of the patient much more collapsed. Here also, a history of cirrhosis will probably be present.

Other possible diseases to be thought of in the differential diagnosis are lead colic and typhoid fever.

The diagnosis of intermittent mesenteric claudication is mentioned because of the possible relation to this disease as a predisposing factor to mesenteric arterial thrombosis. When considering a history of recurring attacks of abdominal pain, flatulence, distention, meteorism of the stomach or intestine, and constipation or diarrhea, one should bear in mind the possibility of intermittent mesenteric claudication when the patient presents one of the two following causes that predispose to it: (1) Chronic nephritis in the old, and (2) Syphilis in the young. The symptoms of this disease are always more severe than the abdominal symptoms sometimes associated with angina pectoris; but it is to be remembered

that the two diseases may coexist.

A valuable aid in the diagnosis of intermittent mesenteric claudication from chronic intestinal obstruction is the invariable absence of visible peristalsis. Some men point out that an examination of the vascular system will generally decide the diagnosis, signs of arteriosclerosis being in favor of the former disease. If however, one were to attach much importance to evidence of this nature, the diagnosis of intermittent mesenteric claudication might be made in almost any abdominal complaint affecting a middle aged or syphilitic patient.

TREATMENT

In the beginning, it is universally agreed that mesenteric vascular occlusion is an abdominal emergency and conservative treatment is definitely out. The sooner operative measures are undertaken after the onset of the disease, the better the prognosis. (11) et. all.

Resection of the infarcted bowel and mesentery wall beyond the lesion into healthy tissues is the procedure which statistics show has resulted in the lowest mortality. There is no place for conservative methods in the surgical treatment of mesenteric vascular occlusion, if gangrene is present or imminent. Enterostomy, often of aid in the treatment of intestinal obstruction, is worthless in mesenteric occlusion. In the 24 cases cited by Trotter and Meyer, treated by enterostomy, there was but one recovery. Exploratory laporatomy, without attempting anything else, is useless. Trotter

reported thirty simple explorations with a 100 per cent mortality. Wide resection of the intestine beyond the involved segment, excising the congested as well as the infarcted gut whenever possible, together with the corresponding mesentery, with immediate or secondary anastomosis, is the procedure of choice. In cases of primary anastomosis, post-operative leakage and peritonitis can be avoided by excising well beyond the limit of normal circulation. Resection with drainage of both ends and later secondary anastomosis is indicated when the serious condition of the patient demands a short, rapid, surgical procedure; when the demarcation on either side of the infarction is not sharply defined, and when the large intestine is involved together with the ileum. Exteriorization should be used only in patients in such poor condition that more extensive surgery would result in death and according to Dunphy and Whitfield, exteriorization is also associated with danger because there is a tremendous blood loss into the involved segment of bowel. Also, as soon as the gangrenous segment of bowel is resected, "there is a striking immediate improvement in the patient's conditions, as if there had been absorption of toxic products from the gangrenous bowel." (19) (20) (18) (48)

Radical resection frequently involves the removal of several hundred centimeters of bowel. There might be hesitation in undertaking such massive resections unless there were some idea of the limit of resection compatible with physiological recovery. First, how long is the average human small intestine? The length

varies with each individual. The average length is about 21 feet. Secondly, what is the physiological limit of resection? Experimental work has shown that dogs would survive resection one-third to one-half of the small intestine. Extensive resections in humans have been reported: Haymond analyzed 257 examples of massive resection (over 200 cm.) and found that 143 or over one-half, had resections of 200-300 cm. He makes the following estimate as to the limits of resection: "Discounting the dangers of the operation itself and it's concomitant complications, patient can withstand a massive resection of 33 per cent of the length of the small intestine and expect the digestive tract to return to normal function; 50 per cent removal constitutes the upper limits of safety in extensive enterectomy." (34)

Papaverine hydrochloride, because of it's vasodilator and anti-spasmodic properties has been used and advanced in pulmonary embolism and mesenteric embolism by Allen and Madsen. (2) (5) (3)

Heparin has been successfully used in mesenteric thrombosis by Murray. Heparin was given in six cases of mesenteric occlusion requiring resection of from 45 cm. to 210 cm. with recovery without complications in four cases. The other two patients died of late complications and autopsy revealed no extension of the thrombosis in the mesenteric vessels. Murray also advises the use of heparin following splenectomy and reports it's use in eight splenectomized patients with no incidence of portal or systemic thrombosis. (60) (61) (62) As to preoperative treatment and preparation of

the patient, this must depend upon the condition of the patient and the surgeon's judgment.

TABLE V.—AGE INCIDENCE OF VENOUS MESENTERIC OCCLUSION

Years	Cases
0 to 9	2
10 to 19	4
20 to 29	13
30 to 39	12
40 to 49	14
50 to 59	15
60 to 69	10
Unknown	5

(76)

TABLE IV.—INCIDENCE OF MESENTERIC THROMBOSIS

		W&E	Flynn	Ophuels	Larson	Ceelen	Robey
Venous:	Hospital population	0.0035	0.0025				
	Autopsy population	0.196		0.033	0.044	0.023	
Arterial:	Hospital population	0.0157	0.0025				
	Autopsy population	0.88		0.066	0.055	0.035	
Both	Hospital population	0.019					0.014

TABLE III.—INCIDENCE OF MESENTERIC OCCLUSION

	Warren and Eberhard*	Boston City Hospital (by Robey)**	Baylor Hospital (by Flynn)†	Ophuels	Larson	Ceelen	Cokkinis§	Reich	Brady	L. I. Miller
Admissions	57380		39668							
Surgical admissions	26793	206571	27712							
Medical admissions	30587	162574	11956							
Mesenteric occlusion	11	51	2	10	36	5	77	251	12	120
Mesenteric artery	82%	21	50%	90%	55.5%	60%	25%	47%	50%	60.9%
Mesenteric vein	18%	5	50%	10%	44.5%	40%	75%	53%	50%	39.1%
Surgical specimens	19836									
Autopsies	1019			3000	36000	8521				
Mortality (total)		94%		100%	100%	100%			72.5%	
Mortality (operative)		72.7%					83%	80.2%	62.5%	
Mortality (with resection)							58%			

*Palmer Memorial Hospital—1927–1933 inclusive.
New England Baptist and New England Deaconess Hospitals 1928–1933.
**1900–1932 inclusive—25 cases not classified.
†Five year period.
§1900–1935 inclusive.

(75)

TABLE VI

MESENTERIC VASCULAR OCCLUSION

RECOVERY FOLLOWING MASSIVE RESECTION (OVER 200 cm.)

26 Cases

	Author or Surgeon	Sex	Age	Amount Resected	Type of Anastomosis
1	ADDISON, O. L.	F	42	360 cm.	Jejuno-colostomy
2	BANKE, K.	M	47	435 cm.	Not given
3	BONNOT, E.	F	28	224 cm.	End-to-end
4	BROUGHAM, F. J.	M	27	345 cm.	End-to-end with Murphy button
5	CHILDE, C. P.	F	59	285 cm.	Lateral ileocolostomy
6	DUNPHY & ZOLLINGER	F	45	216 cm.	End-to-end ileocolostomy
7	FALLIS, L. S.	F	25	270 cm.	End-to-end ileocolostomy
8	GOBIET, J.	M	34	320 cm.	Side-to-side ileocolostomy
9	JERAULD & WASHBURN	M	36	579 cm.	Ileocolostomy
10	LINDNER, H.	M	56	260 cm.	Side-to-side
11	McGANNON	F	25	310 cm.	Not given
12	McGUIRE, E. R.	M	20	330 cm.	Lateral
13	McGUIRE, S. M.	F	27	220 cm.	End-to-end
14	MEYER, J. L.	F	62	230 cm.	Lateral
15	MICHAEL, P. R.	F	—	300 cm.	Not given
16	NIKITIN, A.	M	44	504 cm.	Side-to-side
17	ORGEL, D. H.	M	19	225 cm.	End-to-end. Murphy button
18	PARK, R.	–	—	263 cm.	Not given
19	PERRY, A. C.	M	50	330 cm.	Lateral
20	REDER, F.	F	25	215 cm.	Miculicz
21	SJOVALL, S.	F	44	450 cm.	End-to-end
22	SMIDT, G.	M	38	330 cm.	End-to-end
23	THOMASON, G.	M	26	245 cm.	Side-to-side ileocolostomy
24	TURNER, G. G.	F	24	300 cm.	Lateral ileocolostomy
25	WATSON, P.	F	63	228.5 cm.	Lateral entero-enterostomy
26	WULSTEN, J.	M	64	400 cm.	Side-to-side jejuno-cecostomy

TABLE VI. A Collected series of 26 cases that Recovered following resection of more than 200 cm.—up to 579 cm.—Analyzed as to Age, Sex, Amt. Resected, and Type of Anastomosis done.

(7)

TABLE V

MESENTERIC VASCULAR OCCLUSION

RECOVERY FOLLOWING RESECTION (LESS THAN 200 cm.)

90 Cases

	Author or Surgeon	Sex	Age	Amount Resected	Type of Anastomosis
1	ALLEN, A. W.	M	67	120 cm.	End-to-end
2	ASHLEY, K. C.	F	53	50 cm.	Side-to-side
3	ATKINS, H. J. B.	M	56	60 cm.	Exteriorization-later anastomosis
4	BARNETT, J.	M	45	90 cm.	Lateral—Murphy button
5	BARLETT, W.	M	—	150 cm.	End-to-end. Murphy button
6	BIRD, C. E.	M	61	45 cm.	Lateral
7	BLOCK & GOLDBERG	F	45	20 cm.	Lateral
8	BRADY, L.	F	24	100 cm.	Double barrelled ileostomy. Lat. anastomosis 3 wks. late
9	BROWN, W. S.		48	38 cm.	End-to-end. Murphy button
10	BRUNNER, F.	M	62	80 cm.	End-to-end. Murphy button
11	BRUNS	M	11	20 cm.	End-to-end ileocolostomy
12	CAVE, H. W.	M	42	143 cm.	End-to-end
13	CLAGETT & GRAY	F	56	37.5 cm.	Side-to-side
14	CODMAN, E. A.	M	45	110 cm.	End-to-end over Harrington ring
15	COKKINIS, A. J.	F	39	150 cm.	Lateral
16	COKKINIS, A. J.	M	72	30 cm.	Lateral
17	COPE	M	65	118 cm.	Lateral
18	COWLES, A.	F	18	105 cm.	Lateral

19	CURRY & BACKUS	M	60	76 cm.	Lateral entero-anastomosis
20	DAVIS	F	45	45 cm.	Secondary anastomosis
21	DEQUEME & AUTEFAGE	M	39	20 cm.	Not given
22	DESPARD	F	25	Part of ascend. colon & entire transverse	Not given
23	DESPLAS, B.	M	32	60 cm.	Not given
24	DOOLEY	F	—	120 cm.	End-to-end
25	DOUGLAS, J.	M	37	95 cm.	End-to-end
26	DUDLEY, G. S.	M	39	15 cm.	End-to-end. Murphy button
27	ELIOT, E.	M	38	80 cm.	End-to-end
28	ELLIOTT, J. W.	M	22	35 cm.	Double barrelled ileostomy Secondary resection
29	ELLIOTT, J. W.	M	25	120 cm.	Secondary, end-to-end
30	FALQENBURG, Hr.	M	—	—	———
31	FALLIS, L. S.	F	60	135 cm.	End-to-side ileocolostomy
32	FINNEY, M. J. T.	F	45	75 cm.	Lateral
33	FRANK, L.	M	10	60 cm.	End-to-end
34	GHOSE, S. C.	F	54	30 cm.	Lateral
35	GORDON	F	45	60 cm.	End-to-end over bobbin
36	GOSSET	F	50	80 cm.	End-to-end
37	GREEN & ALLEN	F	24	40 cm.	Lateral
38	HAAGEN, T.	F	37	40 cm.	Lateral
39	HARRIS, G. R.	M	33	155 cm.	Lat. ileocolostomy over Murphy button
40	HENRY, J. G.	M	66	45 cm.	End-to-end
41	JAMESON, J. W.	M	22	45 cm.	End-to-end
42	JENNI	M	50	80 cm.	Not given
43	JOHNSON, C. M.	M	53	125 cm.	End-to-end
44	JONES, W. M.	M	9	90 cm.	End-to-end entero-anastomosis
45	JOPSON, J. H.	F	28	Last portion of ileum	Ileocolostomy
46	KOLBING	F	31	50 cm.	Resection and anterior gastro-enterostomy
47	LAMSON, O. F.	F	60	20 cm. ileum	End-to-end
48	LANG, W. H.	F	60	35 cm.	Lateral entero-anastomosis
49	LAPOINTE, A.	F	17	98 cm.	Enterostomy—later resection
50	LOOP, R. C.	F	46	45 cm.	Lateral
51	MASON, J. M.	F	35	163 cm.	Not given
52	MASON, J. M.	F	35	38 cm.	Not given
53	MASTIN, W. M.	M	35	15 cm.	Not given
54	McGUIRE, S. M.	F	46	135 cm.	End-to-end
55	MILLER, H. A.	F	17	125 cm.	End-to-end over Murphy button
56	MacLEOD, C.	F	43	80 cm.	Lateral entero-enterostomy
57	MITCHELL, J. F.	F	20	45 cm.	Lateral
58	MURRAY & MacKENZIE	M	54	135 cm.	End-to-end
59	MURRAY & MacKENZIE	M	49	75 cm.	End-to-end
60	MURRAY & MacKENZIE	M	46	120 cm.	Side-to-side
61	MURRAY & MacKENZIE	M	63	60 cm.	End-to-end
62	NELSON, E. A.	F	43	90 cm.	Not given
63	NEUMANN	F	42	40 cm.	End-to-end over Murphy button
64	NOLAND, L.	F	24	85 cm.	End-to-end

65	PARKER, C. B.	M	62	Not given	Double barrelled colostomy
66	PATAL	M	51	85 cm.	End-to-end over Murphy button
67	PATTERSON, H.	M	22	Not given	Side-to-side ileocolostomy
68	PELTON, C. L.	M	36	190 cm.	End-to-end over Murphy button
69	POLLOK, L. W.	F	49	135 cm.	End-to-end over Murphy button
70	POLLOK, L. W.	M	6	90 cm.	End-to-end Murphy button
71	RAVEN, R. W.	F	68	50 cm.	End-to-end ileocolostomy
72	ROBEY, W. H.		38	15 cm.	Not given
73	SHINKAMA & MORI	M	61	23 cm.	Side-to-side
74	SCHLEY, W. S.	M	42	60 cm.	End-to-end Murphy button
75	SCHOEMAKER, G. E.	M	28	150 cm.	End-to-end
76	SMITH, H. O.	M	10	35 cm.	Side-to-side
77	SMITH, W.	F	32	38 cm.	Double barrelled enterostomy. End-to-end 4 wks. later
78	SPRENGEL	M	38	10 cm.	Double barrelled enterostomy. End-to-end 5 wks. later
79	STULZ & FONTAINE	M	59	60 cm.	Exteriorizaton
80	STULZ & FONTAINE	M	58	120 cm.	Side-to-side
81	THOMASON, G.	F	69	90 cm.	Side-to-side
82	TOSATTI	M	34	30 cm.	End-to-end
83	WAKEFIELD, R. W.	M	60	75 cm.	End-to-end Murphy button
84	WARNSHUIS, F. C.	M	60	32 cm.	Lateral
85	WEIL	F	45	45 cm.	Double barrelled enterostomy. End-to-end 2 wks. later
86	WEIL	F	52	60 cm.	Ileocolostomy
87	WHEELWRIGHT, J. H.	F	57	38 cm.	End-to-end
88	WHITTAKER & PEMBERTON	–	—	———	Miculicz
89	WHITTAKER & PEMBERTON	–	—	———	———
90	WYNNE	F	42	Not given	Double barrelled enterostomy

TABLE V. Collected series of 90 cases which Recovered following Resection of less than 200 cm. of Intestine, analyzed as to Age, Sex. Amt. resected, and type of Anastomosis done.

(7)

TABLE VII.—COMPARATIVE MORTALITY

Author	Period covered	Per cent Mortality
Jackson, Porter, and Quinby...	1874–1904	92
Trotter.....	?1874–1913	78
Cokkinis.....	1900–1926	83
Warren and Eberhard.....	1913–1933	58.8

(75)

MESENTERIC VASCULAR OCCLUSION
Mortality

Series Reported by	No. Cases	Deaths	Total Mortality %	Operated On	Operative Mortality %	Resections	Mortality of Resection %	Not Operated	Non-operative Mortality %
BLACKBURN	35	31	90	23	82.6	18	83	12	100
BOYCE and McFETRIDGE	13	11	84.6	9	88	8	87.5	4	75
BRADY	14	10	72.5	8	50	6	66.6	6	100
COKKINIS	76	69	90	36	83	12	50	40	97.5
DOUGLAS	11	7	63.6	6	33	4	25	5	100
JACKSON, PORTER, and QUIMBY	214	199	93.4	47	92	16	69	167	95
LARSON	36	36	100	3	100	1	100	33	100
MEYER	92	53	57.6	75	56	43	32.6	17	47
ROBEY	51	48	94	11	72	1	100	40	97.5
TROTTER	366	329	90	97	78.3	47	66	263	96
WARREN and EBERHARD	75	44	58.8	55	45.4	38	34.3	20	95
WHITTAKER and PEMBERTON	60	57	95	19	84.2	3	33	41	100
TOTAL and AVERAGE PER CENT	1037	894	86	389	72	197	62.4	648	92

TABLE IV. The Operative, Non-operative, Total Mortality, and Mortality of Resection from an Analysis of 1037 collected cases.

(74)

TABLE VI.—MORTALITY

	Cases	Alive	Dead	Per cent Mortality
Total number of cases	75	31	44	58.8
Operative cases	55	30	25	45.4
Resection cases	38	25	13	34.2
Cases not operated on	20	1	19	95.0

(75)

TABLE VIII.—DURATION OF SYMPTOMS AND MORTALITY

Duration	Cases	Mortality Per cent
0–12 hours	4	25.0
13–24 hours	12	41.6
25–48 hours	9	71.4
3 days	6	33.3
4 days	6	83.3
Above 4 days	16	78.6

(75)

CONCLUSIONS

Mesenteric vascular occlusion has, in the past, been considered a rare disease but at the present time is not considered to be so rare. Since it's first description in 1843, between 700 and 800 cases have been reported. The supposed rarity of the past was due to faulty diagnosis and lack of consideration of the condition. It seemingly still receives inadequate attention and consideration in the differential diagnosis of acute abdominal condition. Both Cecil's and Christopher's textbooks on medicine and surgery dismiss the disease with less than one-half of a page of discussion.

The statistics of various series of cases of mesenteric vascular occlusion show the general incidence to be from 0.003 per cent to 0.007 per cent.

There seems to be no definite symptom complex to separate the condition as a clinical entity, but with careful consideration acute abdominal conditions, especially those resembling acute intestinal obstruction, the diagnosis should be made in 60 to 70 per cent of cases.

The disease may closely resemble the usual acute abdominal conditions; yet a confusingly different picture is present and in these cases mesenteric vascular occlusion should be considered.

The main features of the disease are: (1) Sudden onset; (2) Acute, usually diffuse abdominal pain; (3) Vomiting and diarrhea, both commonly bloody; (4) Perhaps constipation; (5) Shock, usually to a marked degree.

There has been a great improvement in the last few years, both in diagnosis and treatment. The more favorable results from treatment have been the result of earlier diagnosis and therefore early treatment, so important in this emergency condition.

The treatment most successfully used is surgical exploration and resection of the involved bowel and mesentery and primary anastomoses. The prognosis varies proportionally with the duration of symptoms and surgery, being comparatively good in cases operated on during the first twelve hours after acute symptoms appear and extremely bad in cases with symptoms of over 48 hours duration. In all cases, the mortality varies from 50 to 100 per cent with an average of between 80 and 90 per cent.

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