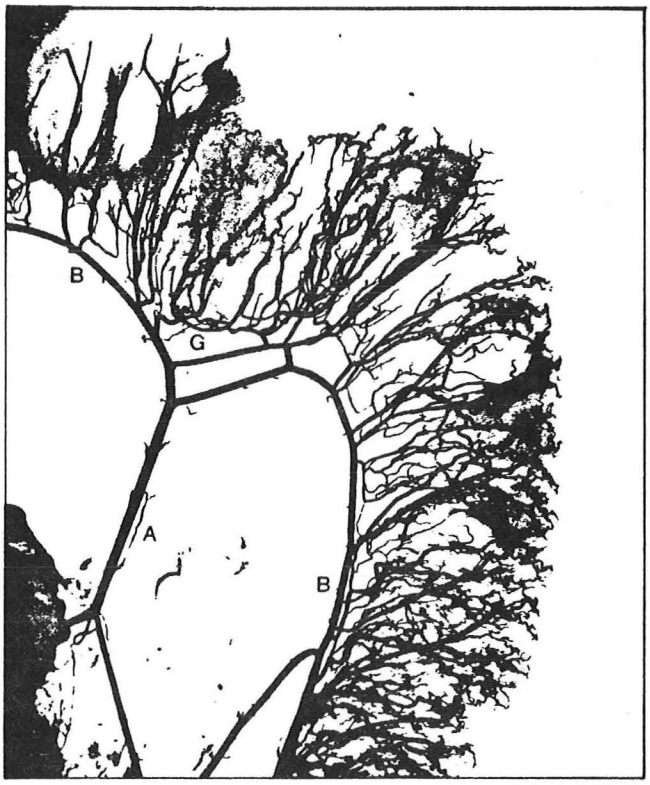
GI

# INTESTINAL ISCHEMIA




## MEDICAL GRAND ROUNDS

**University of Texas**
**Health Science Center**
**at Dallas**

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Intestinal infarction as a result of ischemic injury has been recognized as a disease entity for more than a century. In the past the most characteristic feature of intestinal infarction has been a mortality rate close to 100%. The few survivors often required extensive intestinal resections, and were left with a short-bowel syndrome.

Over the last decade the clinical spectrum of intestinal ischemia has been better defined mainly due to the introduction of angiography, and several distinct clinical syndromes have been identified. Also, early recognition and more aggressive management subsequent to the development of improved vascular surgical techniques have led to a considerable improvement in survival rates.

It is the purpose of these Grand Rounds to review the pathophysiology, symptomatology, and management of the syndromes of acute and chronic intestinal ischemia.

## The Splanchnic Circulation

The intraabdominal organs are vascularly supplied by three major vessels, the celiac artery and the superior and inferior mesenteric artery, all originating from the abdominal aorta (Fig. 1).

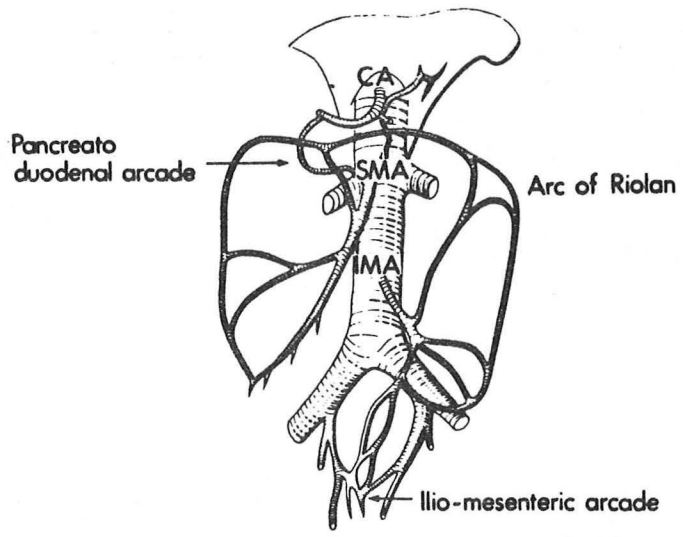



Fig. 1: Origin of the three major splanchnic arteries from the abdominal aorta and the connecting arcade systems

The celiac artery or axis emerges as a trunk at the level of the first lumbar vertebra and divides into three major branches, the splenic, hepatic and left gastric arteries, which supply the liver, spleen, stomach, duodenum,

and pancreas. The arterial supply by the celiac axis is claimed to be the most diversified in the body with multiple collateral pathways, which is illustrated by the fact that there are at least 26 possible collateral routes to the liver alone. Also, there are important collaterals, which connect the celiac axis and the SMA, namely the pancreaticoduodenal arteries through the pancreatic arcade system.

The superior mesenteric artery (SMA) arises at a level between the first and second lumbar vertebra about 1 cm below the celiac axis (Fig. 2).

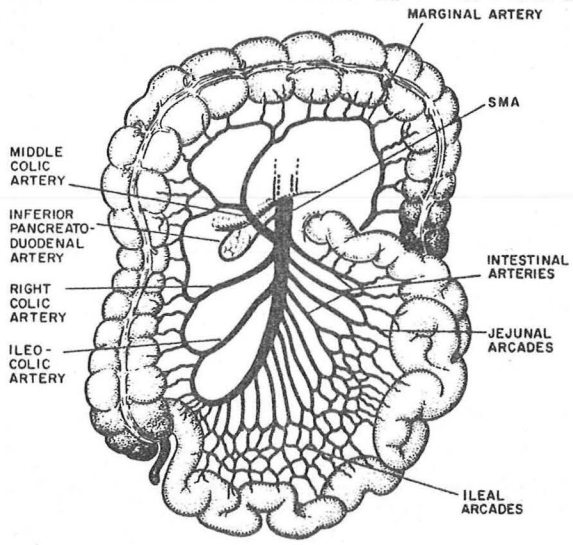



Fig. 2: The superior mesenteric artery distribution and the intestinal arcades

This artery supplies the small intestine, ascending and transverse colon passing through the mesentery in a series of arcades. From the terminal arcade vasa recta transverse into the intestinal wall at the mesenterial border. The cecum, ascending and transverse colon is supplied by the ileocolic, right and middle colic artery. The final arcade formed by these three arteries is called the marginal artery of Drummond, which runs along the medial aspect of the entire colon. From the splenic flexure to the rectum the marginal artery is formed by branches from the left colic artery which originates from the inferior mesenteric artery (IMA). Thus, an important anastomosis between the SMA and IMA is established at the splenic flexure (Griffiths' point). In an angiographic study of 58 post mortem colon specimens, however, it was found that this anastomosis was well developed in only 48%, tenuous in 9%, and absent in 43%. Thus, absence of vascular links between the SMA and IMA is not uncommon and accounts for the fact that the splenic flexure is the most vulnerable area to ischemic injury of the colon.

The inferior mesenteric artery originates at a level of the fourth lumbar vertebra and gives off the left colic, the sigmoid and the superior hemorrhoidal arteries (Fig. 3).

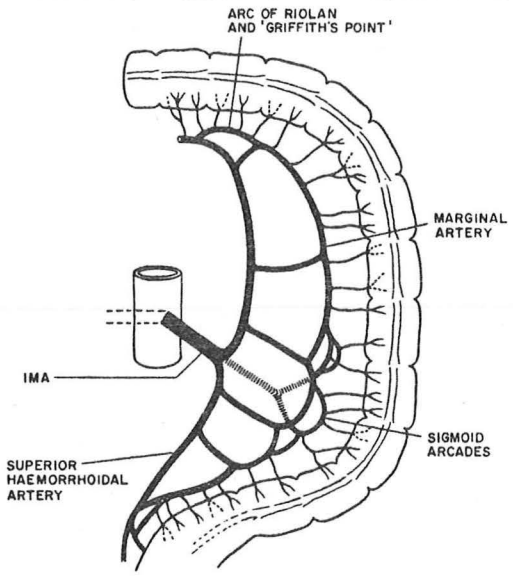



Fig. 3: The inferior mesenteric artery distribution

The ascending branch of the left colic artery may anastomose with the middle colic artery from the SMA, forming the so-called arc of Riolan. Also, the superior rectal artery forms collaterals with the middle and inferior rectal arteries, which originate from the hypogastric artery.

Thus, the three major splanchnic arteries are intimately interconnected by a diversified collateral system, which is of importance not only from a functional and surgical point of view, but also in the understanding of the various syndromes of intestinal ischemia. The collateral circulation may expand considerably in case of occlusion of one or more of the main splanchnic arteries

## Microcirculation

The vasa recta entering the intestinal wall form a rich network of anastomosing vessels in four layers as shown in Figure 4.

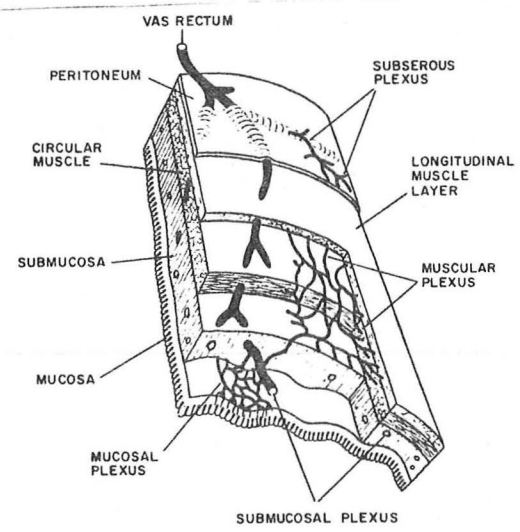



Fig. 4: The arrangement of vascular plexus in four layers of the intestinal wall

The arrangement in four parallel layers will allow blood flow to vary independently in the different layers depending on demand. The arteries that supply the villus originate from the submucosal plexus. Each villus is supplied by one or two central arterioles, which terminate at the villus tip and form a dense capillary network which descends in a fountain-like arrangement towards the base of the villus (Fig. 5).

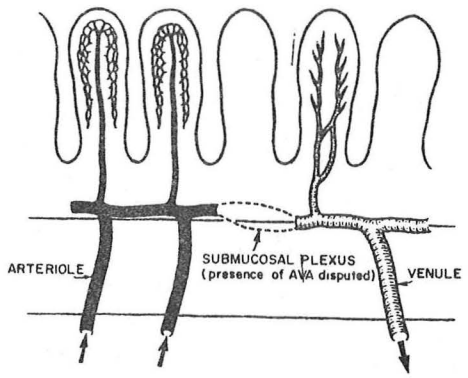



Fig. 5: Vascular supply of the villus in the small intestine

The capillaries drain into a central vein. The close approximation between the arteriole and central vein (< 20 μm) has led some investigators to propose that a countercurrent mechanism exists in the villus with exchange of oxygen and other nutrients between arteriole and vein circumventing the capillary system at the tip of the villus. However, the concept of a villus countercurrent mechanism is not generally accepted.

## Splanchnic Blood Flow

Most of the information on splanchnic blood flow has been obtained in animal experiments with a variety of different techniques, such as microsphere distribution, indicator-dilution, and washout or absorption of highly diffusible inert gasses ($^{133}Xe$ and $^{85}Kr$). A considerable discrepancy has been observed in the amount of blood flow to the splanchnic organs when individual methods are compared in the same animal species presumably both due to the complexity of the splanchnic circulation and the insensitivity of some of the methods.

The number of studies of splanchnic blood flow in man are necessarily limited, and most of the human studies have been performed with the indocyanine green method. Indocyanine green is a dye that is only extracted by the liver and the study requires catheterization of an artery for constant infusion of the dye and of a hepatic vein for continuous sampling and measurement of dye concentration. This method measures total hepatic blood flow which is equivalent to total splanchnic blood flow and thus includes contribution from nonintestinal sources (splenic vein and hepatic artery). It has been estimated that human splanchnic blood flow is about 1800 ml/min and that the splanchnic organs receive 25% of the cardiac output at rest. Since the splanchnic organs only constitute 5% of the total body mass these organs are richly perfused (Fig. 6).

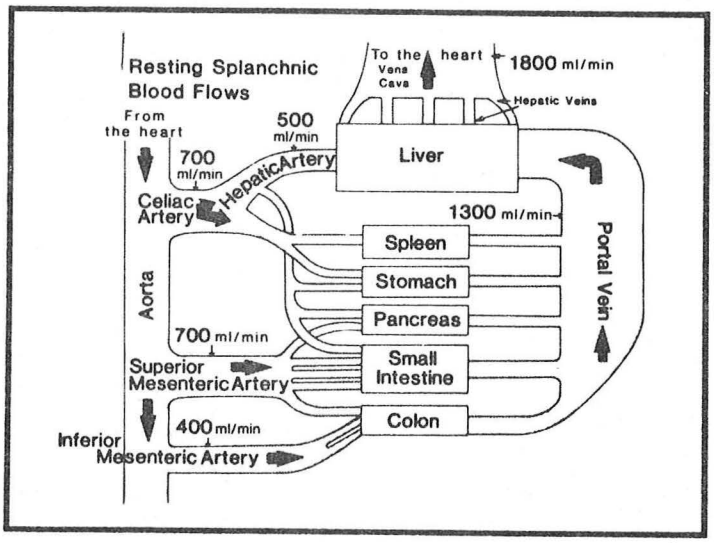



Fig. 6: Splanchnic blood flow at rest.

The regulation of splanchnic blood flow is complex and poorly understood. The major resistance to splanchnic blood flow is exerted by the resistance vessels in the intestinal wall.

Splanchnic circulation at any given time is regulated by at least five different factors (Fig. 7).

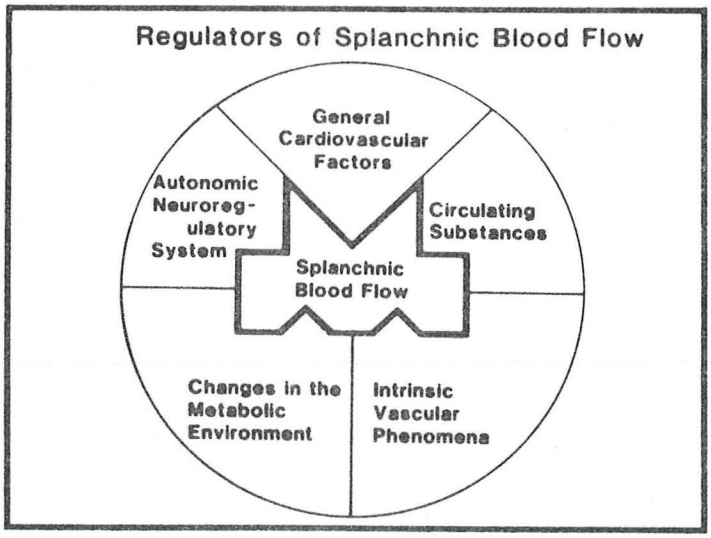



Fig. 7: Regulation of splanchnic blood flow

First, cardiac output is necessarily a determinant factor, and a decrease in cardiac output invariably results in decreased splanchnic perfusion as the gut in these instances is of minor importance as compared to more vital organs. Second, the resistance vessels are innervated by the autonomic nervous system. Increased sympathetic activity results in vasoconstriction and is probably the most important modulator of the resistance vessels. Cholinergic stimulation increase intestinal motility, which secondarily may result in vasodilation and increased intestinal perfusion, but a primary effect of cholinergic nerves on the resistance vessels has not been recognized. It has recently been recognized that the autonomic nervous fibers only constitute a fraction of the total number of nervous fibers in the intestine. VIPergic, serotoninergic and purinergic nerve fibres have been identified by immunofluorescence techniques and whether any of these are involved in the regulation of the resistance vessels are presently unknown. Third, circulating factors released by meals such as GI-hormones, histamine, serotonin, and prostaglandins have been implicated as causative factors in increased splanchnic blood flow during meals. Each of these substances have been found to increase splanchnic perfusion in animals when given in pharmacological amounts, but their precise role in regulation of splanchnic blood flow when fluctuating within the physiological range remains to be defined. Fourth, intrinsic vascular phenomena, such as autoregulation and vascular escape, are also observed in the mesenteric circulation and primarily function to keep blood flow constant

when blood pressure or peripheral resistance vary. The autoregulatory phenomenon is only operative over a limited range of blood pressures as shown in Figure 8.

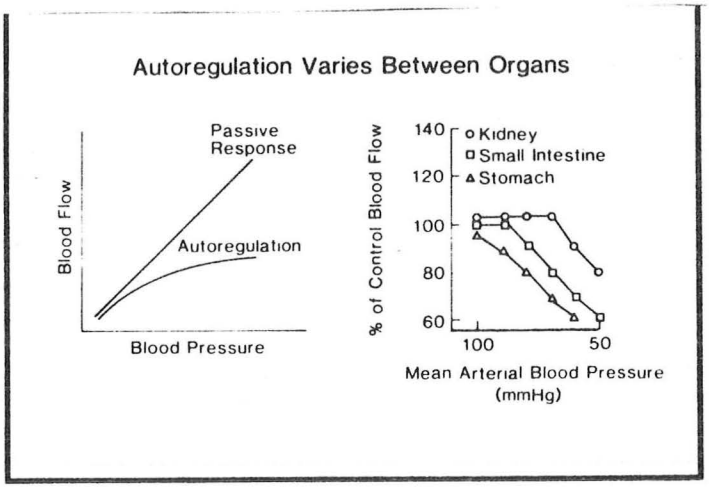



Fig. 8: Autoregulation of blood flow in stomach, small intestine and kidney.

If a vasoconstrictor such as norepinephrine is infused intraarterially, mesenterial blood flow decreases but with time returns to control value, so-called escape, despite continued infusion of norepinephrine (Fig. 9).

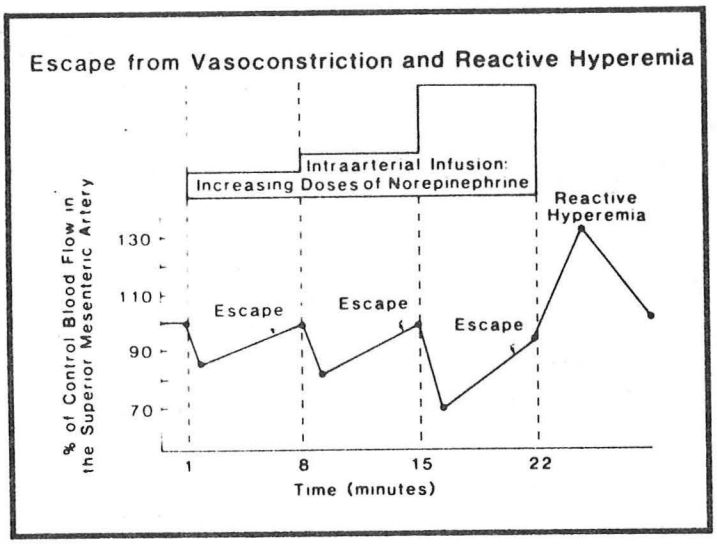



Fig. 9: The escape phenomenon.

The mediator that overrides the norepinephrine vasoconstriction is unknown. Fifth, the intraluminal pressure in the bowel may affect at least mucosal blood flow as ischemic injuries have been observed in patients with intestinal

obstruction. Boley has studied splanchnic blood flow in dogs as a function of increasing intraluminal pressure, as shown in Figure 10.

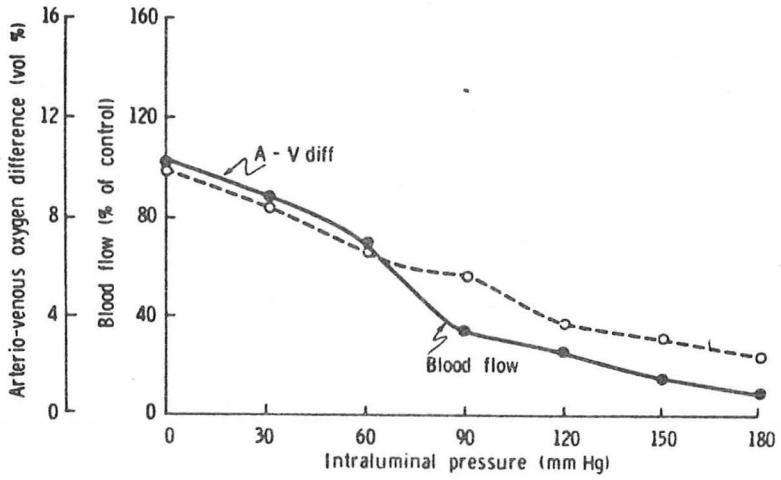



Fig. 10: Splanchnic blood flow in dogs as a function of intraluminal pressure

Splanchnic blood flow decreased progressively as intraluminal pressure was elevated to pressures far beyond a physiological range.

Only a few studies have addressed the effect of ingestion of a meal on splanchnic blood flow in man. A Swedish group measured superior mesenteric artery blood flow in five cancer patients before and after a meal. The mean blood flow before the meal was 708 ml/min and increased to 1558 ml/min sixty minutes after the meal - a mean increase of 121%. As shown in Figure 11

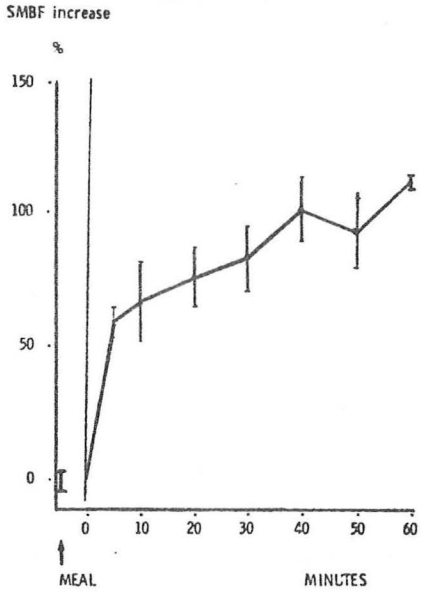



Fig. 11: The average increase in superior mesenteric artery blood flow (SMBF) after a meal in man (Norryd, 1975)

the increase in blood flow commenced five minutes after ingestion of the meal and continued to increase up to 60 minutes. Again, the mediators which initiates meal-induced increase in splanchnic circulation are not identified.

## The Ischemic Lesions

### Acute ischemia

The effects of intestinal ischemia has been studied in several experimental animal models starting more than a century ago, when Litten first reported on the effect of SMA ligation in dogs. He found that the dogs developed vomiting, bloody diarrhea, and fever, and invariably died within 12-48 hours. On a macroscopic level a characteristic series of events ensue after SMA ligation. First, all pulsation necessarily ceases within the SMA distribution and the bowel wall turns blue-white and has increased peristaltic activity. Second, after a few hours contractions are abolished, and the intestinal loops relax and become distended. Third, after 8-10 hours intestinal hemorrhage is observed, and finally at autopsy the bowel wall is dark red with bloody edema. The serosa is raised in blebs, hemorrhage is observed in the muscle layers, and the mucosa is edematous with massive secretion of sero-sanguinous fluid into the intestinal lumen. The veins are dilated without evidence of venous thrombosis. Litten's original findings have been reproduced by several investigators in other animal models, and furthermore, these changes are also seen in humans with acute SMA thrombosis or emboli. It was also noticed in these experimental studies that there was a sharp demarcation between healthy and infarcted bowel at the duodenojejunal flexure and at the splenic flexure of the colon corresponding to the part of the intestinal tract supplied by SMA. The animals often die before frank perforation of the gangrenous bowel wall has occurred. A common feature is the presence of a moderate amount of a turbid, foulsmelling peritoneal fluid.

On the microscopic level, the earliest changes are seen in the mucosa with formation of a fluid filled space just above the basement membrane. Next, the cells at tips of the villi begin to slough and inflammatory cells and bacteria accumulate in the mucosa. The edema progresses and hemorrhagic necrosis extends into the submucosal area, and eventually all mucosal detail disappears and is sloughed off into the lumen. The hemorrhagic necrosis progresses towards the serosal surface and the bowel becomes more thin-walled. At this stage, perforation of the intestinal wall may occur.

### Systemic Effects of Acute SMA Occlusion

The occlusion of SMA has profound systemic effects, and it has repeatedly been observed that the animals (or patients) often die before total gangrene of the intestine has occurred. Moreover, the animals with experimentally occluded SMA will die more quickly if the occlusion is released, than if the occlusion is maintained.

The major systemic effects of SMA occlusion can be summarized as follows:

First, there is loss of blood and plasma proteins into the bowel wall and lumen, due to the hemorrhagic necrosis, and consequently a decrease of circulating blood volume, which alone may be of a fatal consequence, or have a detrimental effect on the perfusion of vital organs. Marston found that total blood volume decreased on the average by 34% over the period of time from SMA ligation to the death of the dogs.

Granger has recently proposed that the increased capillary permeability is a result of the generation of oxygen radicals in the ischemic tissue. During tissue hypoxia ATP is catabolized to hypoxanthine and xanthine dehydrogenase is converted to xanthine oxidase which catalyzes the reaction:

$$\text{HYPOXANTHINE} + O_2 \longrightarrow \text{XANTHINE} + O_2^-$$

and results in the release of superoxide radicals which are highly toxic. Xanthine oxidase is inhibited by allopurinol and the superoxide radicals can be converted to hydrogen peroxide by the enzyme superoxide dismutase (SOD) In an experimental animal model Granger observed that treatment with either allopurinol or superoxide dismutase largely prevented mucosal injury produced by three hours of intestinal ischemia.

Second, interruption of the blood flow in SMA invariably results in a metabolic acidosis due to the production of massive amounts of lactic acid in the nonperfused bowel. Furthermore, the ischemia leads to release of vasoactive mediators, which cause increase in systemic vascular resistance with a concomitant fall in blood flow to vital organs. The exact chemical nature of these vasoactive substances has not been identified. Also, on the basis of early electrocardiographic changes, it has been postulated that a myocardial depressant factor is released from the ischemic intestine.

Third, the necrotic bowel wall creates a favorable environment for bacterial invasion and spread through the venous circulation to the portal system and systemic circulation with resultant septicemia.

These effects may act singularly or in unison to produce severe cardiovascular changes which will further compromise an already delicate balance, as most patients with acute SMA thrombosis or embolus have significant preexisting cardiovascular disease. It is, therefore, not surprising that these disease states continue to carry a very high mortality rate.

Boley has conducted an extensive series of experiments on the effect of acute intestinal ischemia in dogs and one of the important results he obtained through these studies was the observation that acute intestinal ischemia is often associated with mesenterial vasoconstriction. In these studies the blood flow in the SMA was decreased by 50% and maintained at this level while cardiac output, systemic and mesenterial arterial pressures and blood flow through other splanchnic arteries were monitored. Immediately after the acute reduction in SMA blood flow by 50%, the mean arterial mesenteric pressure in

the peripheral bed decreased by 48%, and blood flow in the celiac artery increased (Fig. 12).

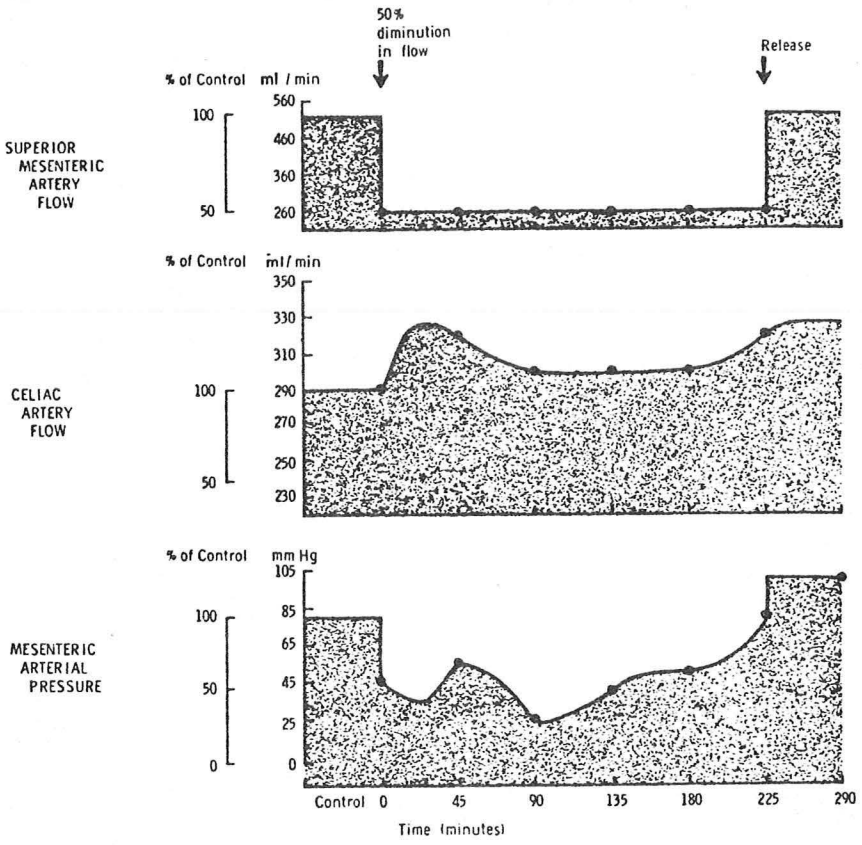



Fig: 12 Effect of prolonged 50% reduction of SMA blood flow on celiac artery flow and mesenteric arterial pressure.

The blood flow in the SMA was maintained at 50%, and within a variable time period (1 to 6 hours), the mesenterial arterial pressure and blood flow in the celiac artery returned to control values. If the occlusion of the SMA was released at this point, the blood flow in the SMA promptly returned to control values. However, if the occlusion of SMA was maintained for a variable time period ($\frac{1}{2}$ to 1 hours) after mesenterial arterial pressure had returned to

control value, then the blood flow in the SMA remained at 30-35% of the preocclusion flow for up to five hours (Fig. 13).

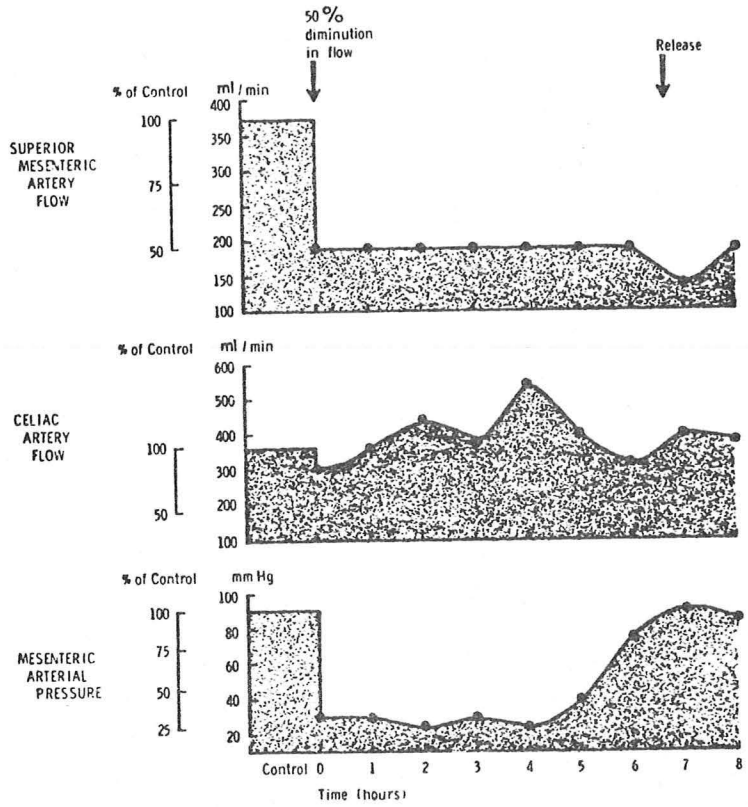



Fig. 13: Effect of prolonged 50% reduction of SMA blood flow on celiac artery flow and mesenteric arterial pressure.

The results of these studies may be interpreted as follows: A 50% reduction in SMA blood flow results in mesenterial vasoconstriction, which is time dependent (variable) and may persist after the occlusion on the SMA has been removed. The mediator(s) of the vasoconstriction (increased sympathetic activity? - unidentified vasoconstrictor substance?) is unknown. However, another important observation in Boley's studies was that simultaneous papaverine infusion (1 mg/min) in the SMA during the occlusion would prevent any increase in mesenterial arterial pressure, and SMA blood would return to

normal with the release of the occlusion (Fig. 14).

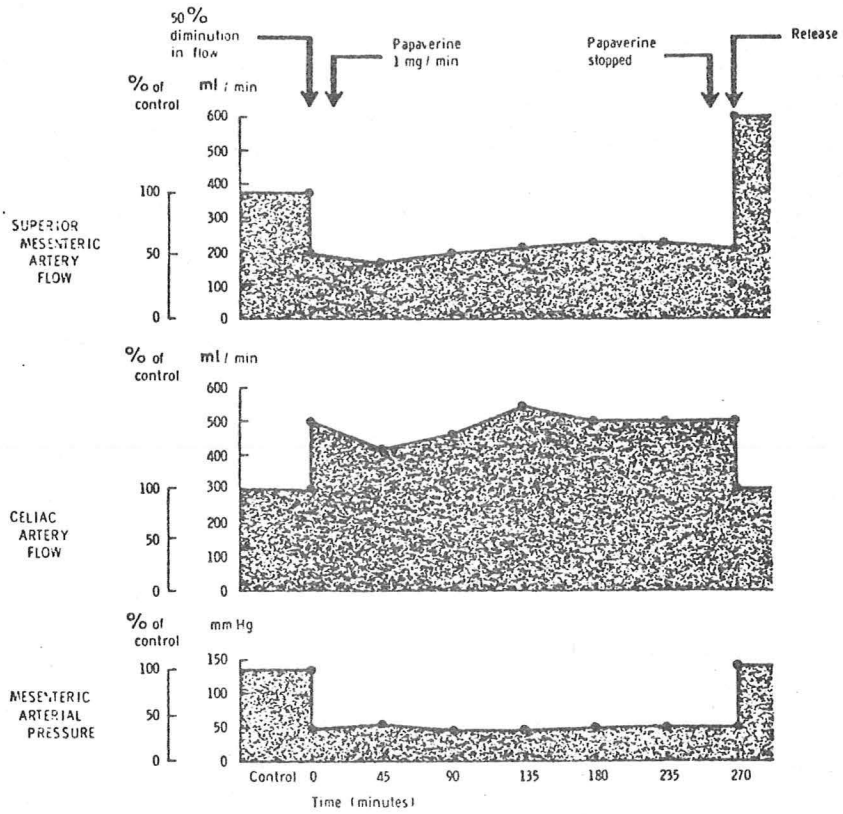



Fig. 14: Effect of papaverine infusion into the SMA during 50% reduction of SMA blood flow.

Furthermore, infusion of papaverine into dogs who had developed vasoconstriction would alleviate the vasoconstriction and result in an increase in SMA blood flow to control value.

Thus, the initial vascular responses to a reduction in SMA blood flow are: increased flow in celiac artery and IMA, and reduced mesenterial artery pressure, which tend to maintain adequate intestinal blood flow. If, however, a low flow state persists, vasoconstriction may develop and continue beyond the point, where the occlusion of SMA has been removed. This series of events are thought to play a major role in the development of nonocclusive mesenterial ischemia.

Gradual Occlusion of SMA

In chronic intestinal ischemia the three major splanchnic arteries are gradually occluded by atheromatous processes at the origin of the vessels - a process that presumably develops over a time span of several years, which allows for development of a collateral circulation through splanchnic or extrasplanchnic arteries, and which generally does not result in acute intestinal necrosis, unless another disease state supervenes (embolus, low-flow state).

Marston has studied the effect of gradual occlusion of the SMA in dogs. In these experiments the collaterals between SMA and the celiac artery and IMA were divided, and the SMA was gradually compressed by a plastic cylinder until the lumen was totally occluded after three weeks. The dogs developed a transient GI disturbance after about one week with vomiting and diarrhea but otherwise stayed healthy without weight loss or signs of malabsorption. At laparotomy after 8 to 10 weeks, the small bowel was pale without pulsations in the intestinal arcades. Aortography at this time showed that the small bowel was supplied by a fine network of extrasplanchnic arteries. Small bowel biopsies of the intestinal mucosa taken at intervals throughout this study remained normal.

The conclusion of this study was that the small intestine can, indeed, survive interruption of the blood flow in the SMA if the occlusion is produced gradually so that a collateral circulation can develop. This observation has also been substantiated in man. There are several case reports of patients, who have total occlusion of all three splanchnic arteries, but do not have any gastrointestinal symptoms. In these patients the splanchnic circulation is maintained by collaterals from the middle and inferior rectal arteries or from extrasplanchnic arteries originating from the retroperitoneal plexus of Turner.

Occlusion of Segmental Arteries

Interruption of blood flow in a segmental artery at the level of the intestinal arcades in the mesentery generally is tolerated very well, and no obvious macroscopic or microscopic lesion is observed as a rich collateral network exists between the arcades.

Similarly, ligation of IMA in experimental animals is not associated with development of ischemic changes, because blood supply to the left colon will be maintained by the marginal artery of Drummond and derive the blood supply from the SMA and the middle rectal arteries.

If, however, both IMA and the marginal artery are ligated in dogs, a series of events occur which appear to reproduce the pattern seen in ischemic colitis in man. First, on day one after the ligation, circumferential sloughing of the mucosa is observed at the rectosigmoid junction on sigmoidoscopy, and the mucosa on both sides of this lesion is heavily congested. Second, after two weeks discrete ulcers are seen in the same area, and finally after six weeks, a fibrous stricture develops.

Obstruction of the blood flow to the colon in dogs by gradual compression of the colic arteries does not result in ischemic changes but when blood flow in the SMA is suddenly reduced in these dogs by controlled hypotension or reduction in blood volume, ischemic colitis develops.

This experimental manipulation may be extended to the clinical situation when a patient with occlusion of the IMA develops ischemic colitis in association with a hypotensive episode.

## The Syndromes of Intestinal Ischemia

The intestinal ischemic syndromes are conventionally divided into acute and chronic ischemia depending on the duration of the symptoms (acute: hours to days; chronic: months to years). Chronic ischemia may at any time reverse to an acute ischemic catastrophe.

In larger series the cause of acute intestinal ischemia is due to SMA occlusion in about 50% of cases, where thrombosis or embolus of the SMA occur with equal frequency. In another 25% the acute ischemia is seen with IMA occlusion, mesenterial vein thrombosis or vasculitis. In the last 25% of cases there is no obvious occlusive disease and the syndrome has been named nonocclusive mesenterial ischemia.

Table I Acute Intestinal Ischemia

| | |
|---|---|
| SMA occlusion (thrombosis or embolus) | 50% |
| IMA occlusion, mesenterial vein thrombosis, vasculitis | 25% |
| Nonocclusive mesenterial ischemia | 25% |

Data compiled from 3 recent large series of acute intestinal ischemia (Boley, 1981, Haglind, 1979 and Ottinger, 1978) and comprise 237 patients.

The true incidence of acute intestinal ischemia is unknown as this entity is still underdiagnosed and often not recognized. In England the reported number of deaths due to acute intestinal ischemia has remained around 1000 per year (incidence: 2/100.000) which is probably a minimum number. In 1975, Dr. Goyal reported on the local experience of acute intestinal ischemia at Parkland Memorial Hospital - 76 cases of confirmed intestinal ischemia were seen over a four-year period. It was Dr. Goyal's personal opinion that this number was a gross underestimate, and that probably 10 times as many patients have significant intestinal ischemia.

## Pathogenetic Considerations

### Arteriosclerosis/thrombosis

It is, of course, not surprising that thrombosis of the SMA accounts for a high proportion of both acute and chronic intestinal ischemia, as these diseases most often occur in older persons with established arteriosclerotic disease (arteriosclerotic heart disease, peripheral vascular disease, aortic aneurysm). The origin of the SMA is most commonly involved by deposition of atheromatous plaques and subsequent narrowing of the arterial lumen, but both the celiac artery and the IMA may be involved as well. The lumen may be occluded gradually or acutely by subsequent thrombus formation. Ottinger

divided the proximal portion of the SMA into four regions (Figure 15)

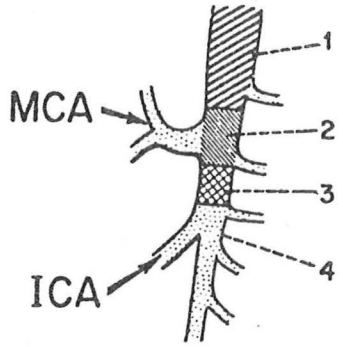



Fig. 15: Division of the origin of the SMA into four regions (Ottinger 1978)

and could accurately analyze the site of occlusion (region 1 to 4) and the extent of intestinal infarction in 100 patients. Generally, there was a good correlation between the site of occlusion and the extent of infarction, i.e. more extensive infarction with proximal lesions.

It should be stressed that the angiographic demonstration of total occlusion of all three major splanchnic arteries does not necessarily signify intestinal ischemia, as the splanchnic blood flow may be maintained by extrasplanchnic arteries. These patients, however, may be particularly prone to develop acute intestinal ischemia if, for any reason, systemic blood flow is acutely decreased.

Arterial Emboli

Embolism of the spanchnic arteries is most frequently observed in the SMA which is thought to be due to the oblique take-off of SMA from aorta where the celiac artery and IMA both originate in the horizontal plane. The emboli may be thrown from a mural thrombus in the left ventricle, or from atheromatous plaques in the aortic wall. Hence, SMA emboli are often associated with severe organic heart disease, such as post-myocardial infarction, atrial fibrillation, ventricular or aortic aneurysm, and there are often synchronous emboli to other arteries. The emboli tend to lodge at a site of normal anatomic narrowing usually at the origin of a major branch. In a series by Ottinger of 54 cases of SMA embolism, the majority of the emboli (80%) were localized in regions 2 and 3.

Mesenterial Vein Thrombosis

Thrombosis of the mesenterial vein accounts for about 8-10% of all cases of acute intestinal ischemia and is most often idiopathic. It has been observed in a variety of clinical conditions, such as hyperviscosity syndromes, hypercoagulability, tumor compression of the portal vein, and in association with the use of contraceptive pills. Whenever mesenterial vein thrombosis occurs, there are often multiple emboli in the smaller veins in the mesentery.

## Vasculitis

The small vessels in the intestinal arcades or in the bowel wall may be involved in a variety of connective tissue diseases, such as SLE, polyarteritis nodosa, and rheumatoid arthritis. The damage to the small vessels occurs insidiously and often with only segmental involvement. Therefore, massive intestinal ischemia is rare in these disorders, but segmental infarction may result with mucosal ulcerations and later stricture development.

## Nonocclusive Mesenteric Ischemia

Nonocclusive mesenteric ischemia has emerged as an important cause of acute mesenterial infarction. The existence of this disease entity was speculated upon in the 60's, when intestinal ischemia was encountered in the presence of nonoccluded splanchnic arteries, but it was not definitely proven to exist until angiography became available. This syndrome usually develops in patients who have partial occlusion of the splanchnic arteries and severe cardiac disease. The splanchnic blood flow is presumed to be reduced but not to a level that causes symptoms of intestinal ischemia. If these patients, however, sustain an episode of decreased cardiac output or hypotension, mesenteric blood flow may be reduced to a critical level and ischemic injury may ensue. The important feature of nonocclusive mesenteric ischemia is the development of mesenteric vasoconstriction - in an effort to maintain blood flow to more vital organs than the gut or due to vasoconstrictors released by the ischemic tissue - and that vasoconstriction may persist after systemic blood flow has been restored analogous to the experimental model with 50% SMA occlusion in the dog.

A contributing factor to vasoconstriction can be drug induced. Many of these patients are treated with digoxin, and it has been shown experimentally in several studies that digoxin causes mesenterial vasoconstriction. Since mesenterial vasoconstriction in the setting of nonocclusive mesenteric ischemia is probably multifactorial, it is difficult to evaluate if digoxin adds synergestically to the persistent vasoconstriction, but a general advice at present is that digoxin should be used with caution in these patients. Vasopressin is also a potent mesenterial vasoconstrictor at the dosages usually used to treat bleeding esophageal varices and acute intestinal ischemia has been described in this setting. Also, if hypotension is the precipitating event, $\alpha$-adrenergic agonists should be avoided in the treatment of hypotension, as these drugs increase mesenterial resistance by vasoconstriction. Dopamine, however, does not constrict mesenterial arterioles and should be the drug of choice to relieve the hypotension.

## Symptomatology

Generally, acute intestinal ischemia is a disease most often seen in the older population (> 50 years), and is usually associated with severe cardiac

disease (congestive heart failure, valvular heart disease, cardiac arrhythmias or recent myocardial infarction) or hypovolemia/hypotension (Fig. 16).

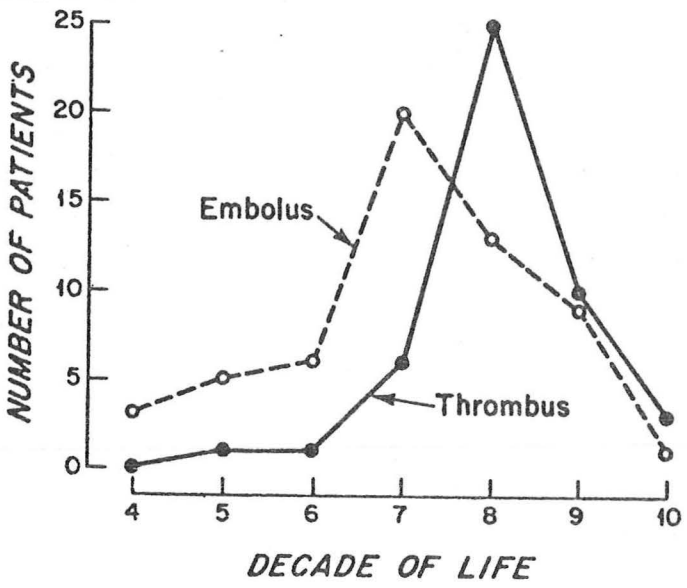



Fig. 16: Age distribution of acute SMA occlusion by emboli or thrombi in 103 patients (Ottinger, 1978)

The typical symptoms of acute intestinal ischemia are abdominal pain, nausea, vomiting, and diarrhea. The pain is present in 75 to 95% of patients but may vary considerably in severity, location and character.

In up to 25% of patients acute ischemia develops without pain but the patients present with an ileus-like picture with abdominal distension and/or diarrhea. This occurs especially in patients with nonocclusive intestinal ischemia. Diarrhea is present in 30 to 50% of cases but bloody diarrhea is rare. Occult blood, however, may be positive in up to 75%.

The physical findings on the examination of the abdomen are of limited value. The initial findings early in the course may only be abdominal distension with diffuse tenderness and active bowel sounds. The presence or absence of bruits over the abdominal vessels is not of diagnostic value. It should be noted, however, that the pain when present is often out of proportion to the physical findings, and this should raise the suspicion of an abdominal vascular catastrophe. It is of utmost importance that the threshhold for suspecting intestinal ischemia should be kept at a low level so intervention can be undertaken as early as possible, as survival is critically related to the duration of symptoms.

Later in the course when intestinal infarction and/or perforation has occurred the physical findings are more prominent with absent bowel sounds and rebound tenderness.

Laboratory findings, unfortunately, are also of limited value. Leucocytosis is often observed, but is unspecific. It has been claimed that s-phosphate and alkaline phosphatase may be elevated early in the course, but these findings have not been substantiated in a prospective manner. Intestinal ischemia may cause a severe metabolic acidosis but again, this finding is often not helpful in these severely ill patients.

What is truly lacking is a specific intestinal enzyme elevation to indicate mucosal damage analogous to CPK-MB in acute myocardial infarction.

Management

The first steps in the initial management of patients with suspected acute intestinal ischemia are directed against the presumed precipitating cause, such as congestive heart failure, arrhythmia or hypotension with appropriate corrective measures. The question whether digoxin should be discontinued is still a matter of dispute, and it may not be possible. Patients with systemic effects of intestinal infarction, such as hypovolemia and/or bacteremia are treated with intravenous fluids, gastrointestinal decompression and antibiotics.

When the patient has been resuscitated and is stable, a plain radiograph of the abdomen should be obtained. The main purpose of this examination is to rule out other causes of a surgical abdomen such as a perforated viscus. A normal KUB does not exclude acute intestinal ischemia. Rather, the lack of abnormal findings in a paient with abdominal pain and associated risk factors of acute ischemia should raise the suspicion of preinfarction ischemia. When positive findings are seen on KUB (dilated loops of bowel with air-fluid levels) the patients often have positive physical findings indicative of intestinal infarction and peritonitis and the outcome is dismal with mortality rates about 90%. It has been estimated that the latency period between the onset of compromised bowel perfusion and positive signs of ischemia is in the area of 12 hours in cases of thrombosis or embolus, and up to several days in nonocclusive mesenteric ischemia.

At this stage angiography should be contemplated. According to Boley all patients with suspected acute intestinal ischemia should undergo angiography, irrespective of the findings on physical examination or KUB. Another group of vascular surgeons would reserve angiography to patients with preinfarction (negative physical examination and negative KUB) and recommend immediate surgery for patients with positive findings, as most of these patients will require intestinal resection and/or revascularization.

Only angiography can differentiate between occlusive and nonocclusive mesenteric ischemia. Angiography serves several purposes: 1) it deletes the type and severity of intestinal ischemia, 2) it can localize the site of occlusion, 3) the splanchnic circulation including collateral contributaries can be evaluated, and 4) it can provide a means of local injection of vasodilators.

Initially, a flush aortogram is obtained to visualize the aorta (aneurysms, dissections), occlusions of the major splanchnic arteries (thrombosis or emboli) and evaluate the collateral circulation. Thereafter,

selective angiography of the SMA is performed to further delineate occlusions or signs of vasoconstriction. The usual signs of vasoconstriction are narrowing at the origin of the major branches of the SMA and intermittent narrowing and dilation of the arteries in the intestinal arcades (string of sausages) as shown in Figure 17.

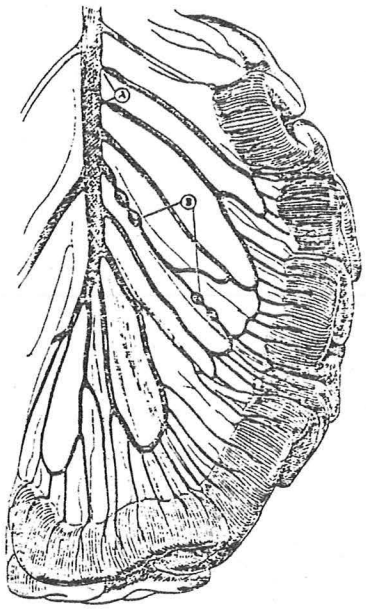


Fig. 17: Typical angiographic findings in nonocclusive mesenteric ischemia (Boley, 1981)

The findings on angiography will decide the further treatment. Essentially there are four possible findings as outlined in the scheme below and the treatment plan, as proposed by Boley, will vary accordingly (Fig. 18).

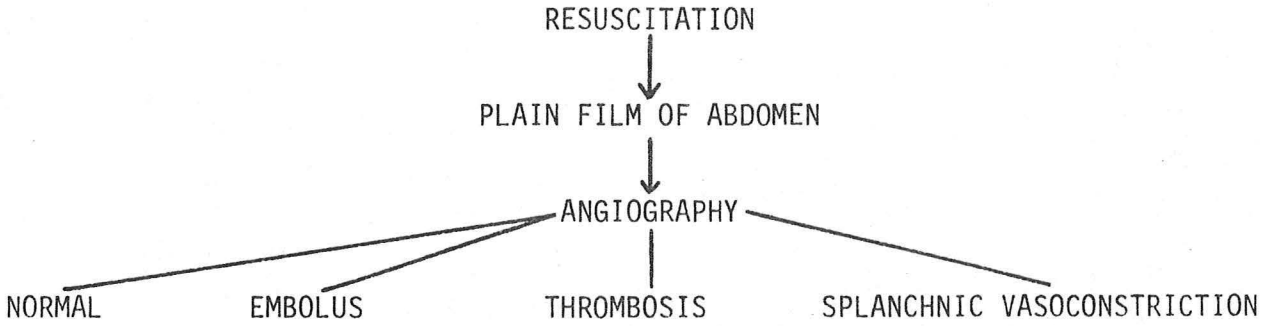



Fig. 18: The initial steps in the management of patients with suspected acute intestinal ischemia.

If angiography is normal and the patient does not have peritoneal signs then continued observation is recommended. In the presence of positive peritoneal findings, one should proceed to laparotomy as the patient may have a disease other than acute ischemia as a cause of peritonitis.

If an embolus in the SMA is observed there may be a total occlusion, but more often the SMA is only partially obstructed and there is usually signs of vasoconstriction in the arteries above and below the embolus. The management of patients with acute SMA emboli according to Boley's proposals is outlined in Figure 19.

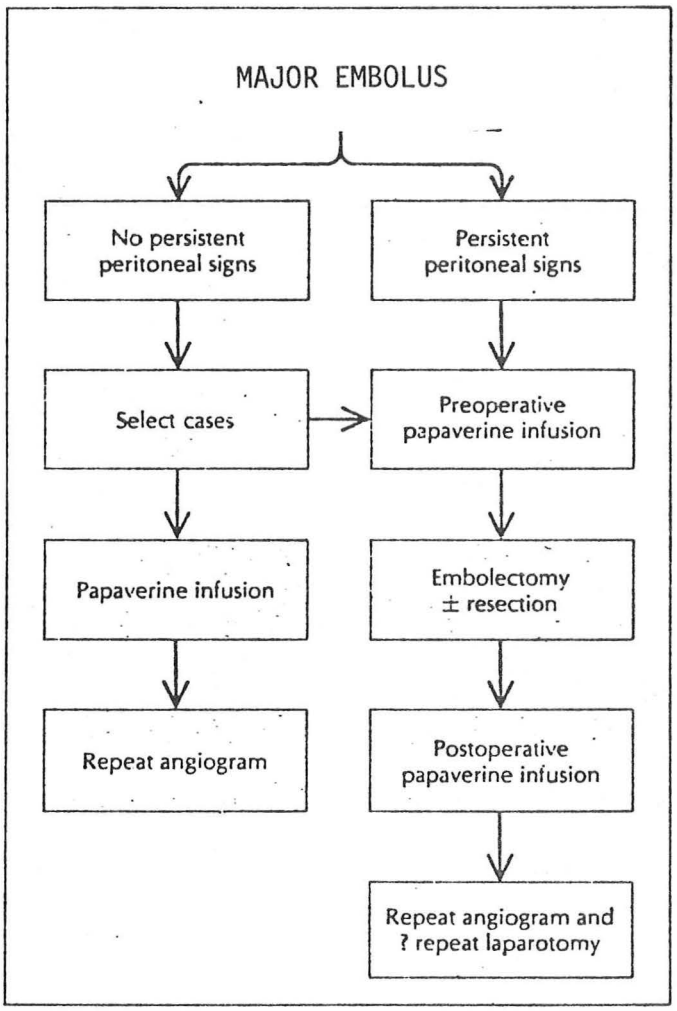



Fig. 19: Management plan for acute SMA embolism (Boley, 1981).

In SMA embolism, Boley recommends an initial bolus infusion of 25 mg Tolazoline ($\alpha$-adrenergic blocker) into the SMA followed by a repeat angiogram in order to obtain a better visualization of the peripheral mesenterial circulation and to provide information about the potential response to papaverine infusion. At this point a papaverine infusion into the SMA is started irrespective of the physical findings. Selected cases (minor, more distal emboli with apparent good mesenterial perfusion after tolazoline) may be managed by papaverine infusion alone. Patients with positive physical findings undergo laparotomy, while papaverine infusion is continued, and embolectomy is performed and care is taken to remove residual clots. When the blood flow in the SMA has been reestablished, intestinal viability is assessed and obvious nonviable intestine is resected. The papaverine infusion is continued for 12 to 24 hours and a repeat angiogram is then obtained. If it has been decided to perform a "second look" operation, the papaverine infusion is continued until the operation has been completed. Anticoagulation is started 48 hours after the operation.

Boley recently surveyed the results in 47 patients with SMA emboli seen at the Montefiori Hospital between 1967 and 1978. Twenty-five patients were managed according to traditional methods, and 20 were managed according to the above mentioned scheme (high degree of suspicion, early angiography and papaverine infusion). Two patients died before treatment was initiated. The overall mortality in the first group was 80%, whereas it was reduced to 45% in the group treated with the more aggressive approach.

Acute SMA thrombosis angiographically appears as a total occlusion of the proximal portion of SMA. These patients often have evidence of arteriosclerotic disease (coronary, cerebral, peripheral) and may have had symptoms of chronic intestinal ischemia for several months prior to the acute thrombosis, which develops over an ulcerated plaque in an already arteriosclerotic narrowed SMA. The peripheral mesenterial branches of SMA may still be visualized due to perfusion through collaterals. A difficult problem sometimes arises in the differentation between acute and chronic intestinal ischemia in patients with abdominal pain, normal physical findings, and total occlusion of the SMA on angiography. In these instances it is important to observe the collateral circulation. If it is well developed this argues for a more long-standing occlusion and the patients are treated expectantly. On the other hand, if the collaterals are minimal or absent, acute intervention is

indicated. The suggested plan for the management of acute SMA thrombosis according to Boley is shown in Fig. 20.

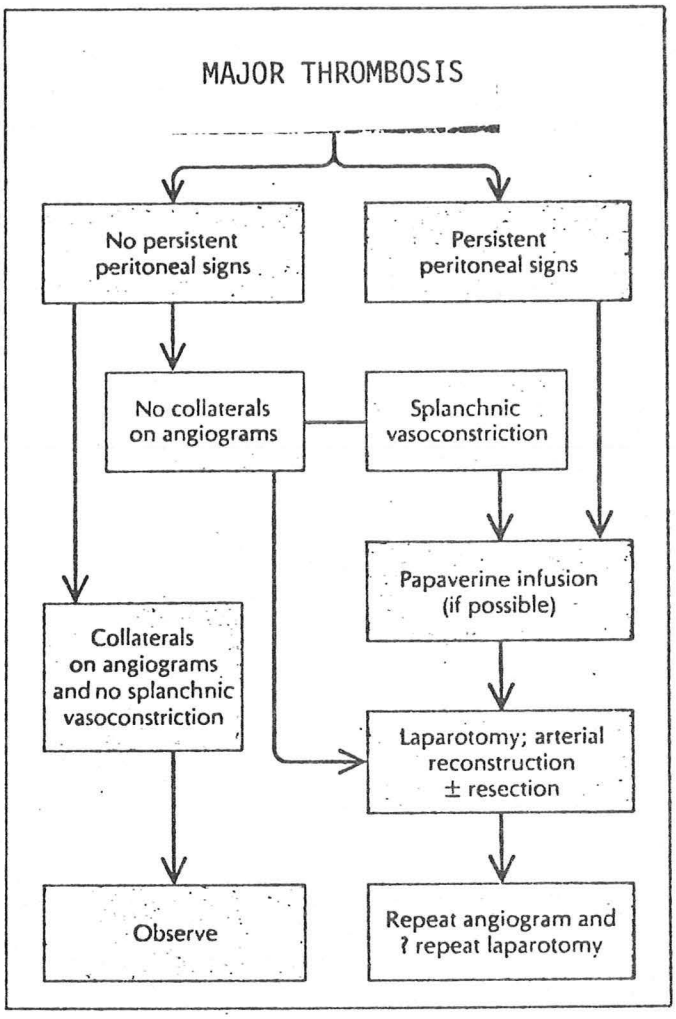



Fig. 20: Management plan in acute SMA thrombosis (Boley, 1981).

Again, in this situation Boley favors preoperative papaverine infusion which, however, often is impossible when total SMA occlusion is present, and the patients are brought to the operating room. The recommended approach to treatment of SMA thrombosis is now insertion of a bypass graft (saphenous vein or dacron) between the aorta and to a point on the SMA distal to the obstruction. Previously, thromboendarterectomy was often employed, but has proven to be technically more difficult. If a bypass graft is impossible to insert due to severe arteriosclerotic damage of the abdominal aorta, a more distal bypass may be created by anastomosing the ileocolic artery to the right common iliac artery.

The prognosis of acute SMA thrombosis is still dismal. In Ottinger's series of 103 patients with acute SMA occlusion 46 had acute thrombosis and the mortality rate was 96%.

The diagnosis of nonocclusive mesenteric ischemia is based upon a nonoccluded SMA and signs of mesenterial vasoconstriction as previously described in patients, who are not hypotensive or on vasopressors at the time of angiography, but have severe underlying disease (patients in intensive care units). These patients are also started on papaverine infusion irrespective of physical findings. Boley's proposal for the management of nonocclusive mesenteric ischemia is outlined in Fig. 21.

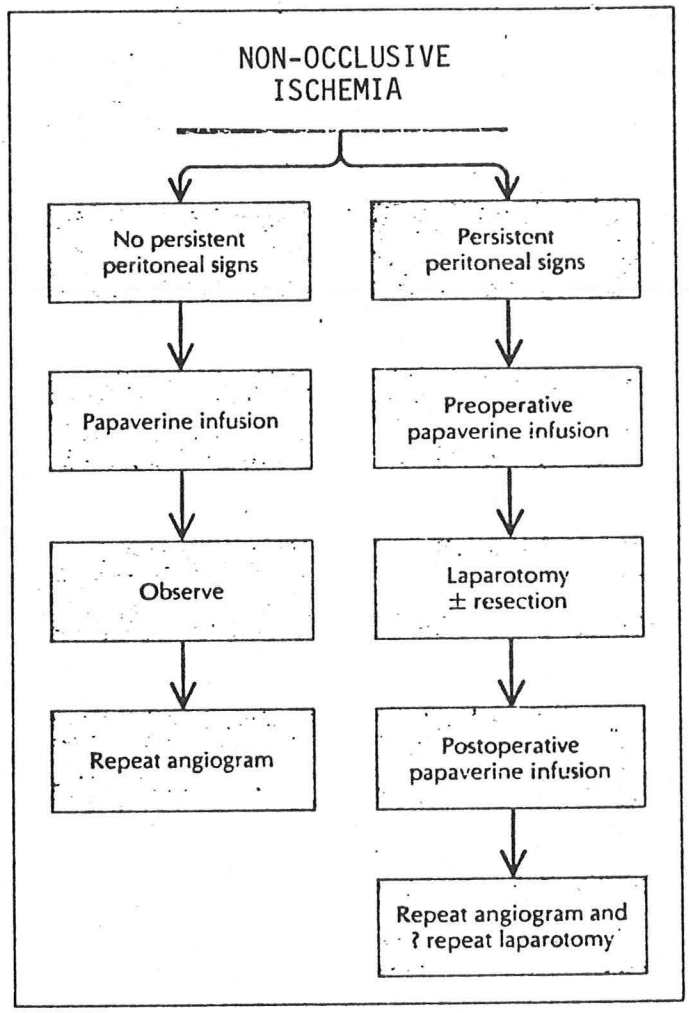



Fig. 21: Scheme for the management of nonocclusive mesenteric ischemia (Boley, 1981).

If overt peritoneal signs are absent the infusion is maintained for 24 hours and angiography is repeated. Patients with positive physical findings undergo laparotomy while papaverine infusion is continued and obvious infarcted intestine is resected. A "second look" operation may be necessary.

In most previous series the mortality rate of nonocclusive mesenteric ischemia approached 100% but with the approach outlined above, Boley had a mortality rate of 40%, albeit in a limited number of patients (n = 15).

Acute mesenteric venous thrombosis is now a rare condition accounting for only 5-10% of all cases of acute intestinal ischemia. Prior to angiography it was thought to be the most frequent cause of intestinal ischemia, as nonocclusive mesenteric ischemia was not recognized. The diagnosis of mesenteric vein thrombosis cannot be made at angiography which is often normal, and the condition is diagnosed at laparotomy. Thrombectomy is usually not possible, as the thrombi are wide spread and may involve smaller mesenterial veins, where it is not feasible to pass a Fogarty catheter. The ischemic lesion frequently involves the middle portion of the small intestine and colon involvement is rare. The recommended treatment is intestinal resection with a "second look" as subsequent infarction may occur. Anticoagulation should be initiated in the postoperative phase.

Finally, in a limited number of cases acute intestinal ischemia is caused by small vessel disease in a variety of collagen-vascular disorders. Isolated case reports have described intestinal infarction in patients with polyarteritis nodosa, SLE, rheumatoid arthritis and dermatomyositis. The incidence of intestinal involvement in these disorders is unknown. In one series of 342 patients with various forms of vasculitis, only 5 patients suffered intestinal infarction. Due to the rich collateral circulation in the mesentery, intestinal vasculitis will more often produce focal segmental ischemia, rather than total intestinal infarction.

The clinical presentation of patients with short segment ischemia varies, and three distinct patterns have been recognized. One group of patients may present with an acute abdomen simulating acute appendicitis and on physical examination an inflammatory mass may be palpated. KUB will often be normal at the early stage but later show a "sentinel loop." If a small bowel series is performed an isolated loop of small intestine may demonstrate the special findings of acute ischemia: thumbprinting, spasm or ulceration. In the case of benign physical findings, the underlying disease is treated with a high dose of corticosteroids. However, if peritoneal signs are present, then laparotomy is indicated with segmental resection of the affected bowel loop.

Another group of patients will present with symptoms suggestive of chronic inflammatory bowel disease with abdominal pain, diarrhea, weight loss and fever. A small bowel series may be indistinguishable from Crohn's disease of the small intestine with a thickened bowel wall with mucosal nodularity and narrowing. At laparotomy the affected loop may also have the gross appearance of a Crohn lesion, and the true nature of the disease is first revealed at the histological examination (specific involvement of the arteries; absence of granuloma formation).

The third and most common group present with symptoms of chronic small bowel obstruction. Sometimes the patients have experienced a more acute abdominal episode several months prior to the development of obstructive symptoms. A small bowel series will reveal a smooth, tapered stricture with proximal dilatation and normal mucosa distally. Apart from the obstructive symptoms, the patients may also have features of a "blind loop" syndrome with bacterial overgrowth and malabsorption. The treatment is surgical with resection of the strictured loop which usually results in resolution of the symptoms.

## Chronic Intestinal Ischemia

The syndrome of chronic intestinal ischemia was first proposed as a disease entity by Schnitzler in 1901 but did not gain widespread recognition until the late 1950's, when corrective surgery was first employed. The syndrome has been described with many names, such as intestinal claudication, abdominal or intestinal angina to indicate that the main feature of the syndrome is meal-related abdominal pain and is presumed to be caused by inadequate splanchnic perfusion during intestinal exercise (motility and absorption), when increased perfusion is called upon by a meal analogous to exercise induced angina pectoris.

The inability to increase splanchnic blood flow is due to arteriosclerotic involvement of at least two of the main splanchnic arteries with usually complete occlusion of the celiac and SMA.

The incidence of splanchnic involvement in the arteriosclerotic process is high. In several autopsy series it was found that in persons over 45 years about 50% had some arteriosclerotic narrowing of the celiac and/or SMA. In persons with diffuse arteriosclerosis up to 75% will have atheromatous plaques at the origin of the splanchnic arteries and the celiac and SMA are involved in equal proportions. In other words, partial occlusion to complete obstruction of these arteries is a frequent finding in the elderly population.

So far the diagnosis of chronic intestinal ischemia has been one of exclusion and has usually been based on the constellation of meal-induced abdominal pain and angiographic demonstration of total occlusion of two of the main splanchnic arteries. It should be emphasized at the outset that chronic intestinal ischemia is a rare disease and the diagnosis is difficult since meal-related abdominal pain is unspecific and since positive findings on angiography are frequent. What is clearly needed is a functional test of splanchnic perfusion (exercise test?), or a direct measurement of splanchnic blood flow at rest and after meals.

The cardinal symptom in chronic intestinal ischemia is meal-related pain, which occurs from 20 to 60 minutes after meals, often mid-epigastric and of a cramping colicky nature. With time the pain may become so severe that the patient decreases food intake and loses weight. In a limited number of cases a malabsorption syndrome has been documented in association with chronic intestinal ischemia.

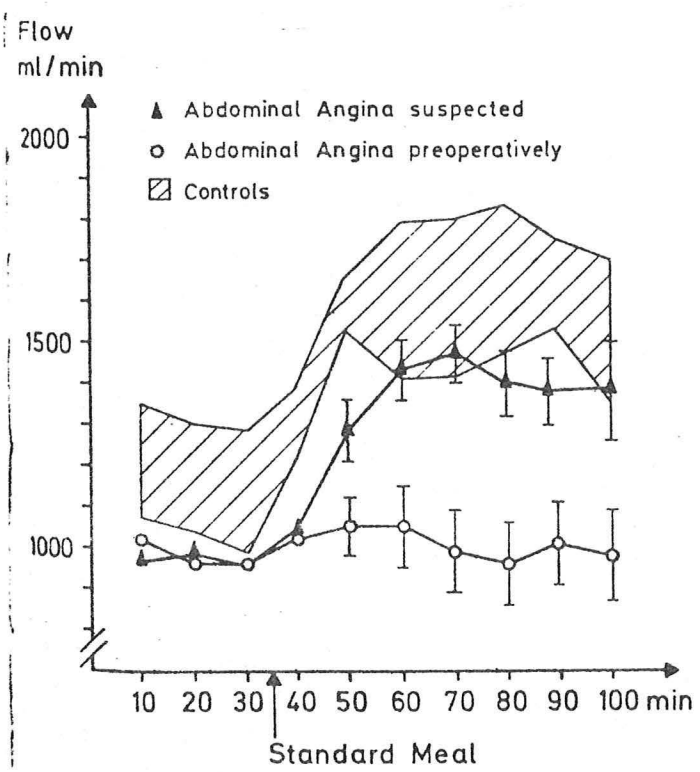
On physical examination the patient may have evidence of weight loss but abdominal findings are usually sparse. The presence of bruits over the abdominal vessels are unspecific.

Barium studies of the small and large intestine are usually normal which may prompt the clinician to perform angiography as a last resort in a patient with unexplained abdominal pain.

Angiography should be performed both with a flush aortogram with lateral and AP projections to demonstrate occlusions of the celiac artery, SMA or IMA and evaluate collateral circulation and, if possible, with selective injections in the individual arteries to evaluate the degree of stenosis. The presence of prominent collaterals suggests a chronic process. The presence of

total occlusion of all three arteries, however, does not confirm the diagnosis, as this finding may be seen in persons without gastrointestinal symptoms.

In an attempt to improve the specificity of the diagnostic workup, a group in Denmark measured total splanchnic blood flow in a small number of patients using the indocyanine-green method. The series included four controls (abdominal pain with normal angiogram), five with suspected abdominal angina (pain and moderate narrowing of one or two of the splanchnic arteries) and six with abdominal angina (total occlusion of at least two arteries). The splanchnic blood flow was studied at rest and after a standard meal of 1000 calories. As shown in Figure 22 (below), the mean blood flow in controls increased by 40% after the meal. The blood flow at rest in the patients with suspected or typical abdominal angina was about 16% lower than controls but more importantly after the meal blood flow did not increase in patients with typical abdominal angina, whereas it increased to control values in suspected abdominal angina. Moreover, the six patients with abdominal angina underwent arterial reconstruction and became asymptomatic. A repeat estimation of splanchnic blood flow after the operation revealed values within the normal range both at rest and after meals as seen in Figure 23.



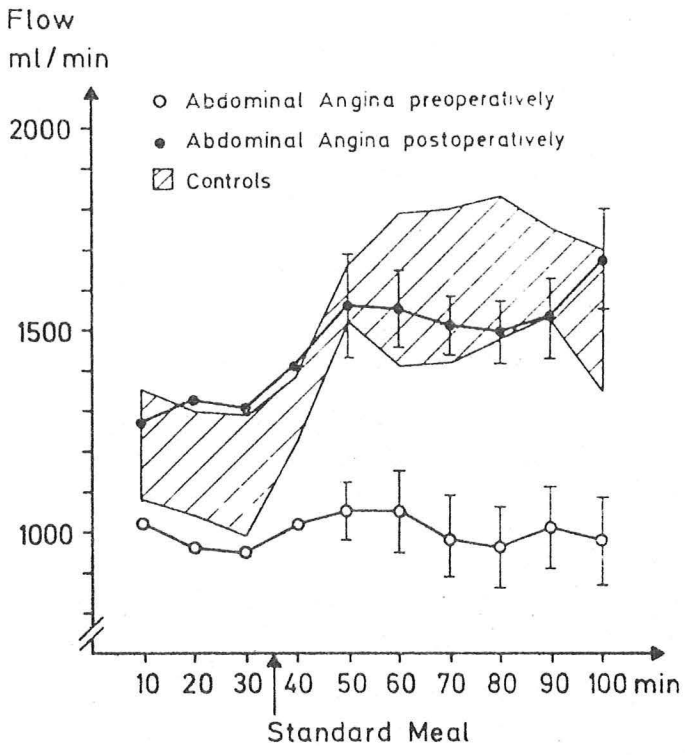




Fig. 22 & 23: Splanchnic blood flow at rest and after a standard meal in controls (n=4), suspected abdominal angina (n=5) and abdominal angina (n=6) (Buchardt Hansen, 1977)

The authors suggested that a positive test should demonstrate a low fasting splanchnic blood flow (< 1000 ml/min) with no increase after meals. The test is technically demanding and requires catherization of an artery and a hepatic vein, and so far nobody has attempted to reproduce the observations. However, it is the first report that has documented inadequate splanchnic blood flow during meals in patients with chronic intestinal ischemia.

The indication for surgical treatment of chronic intestinal ischemia is still difficult to determine and is mainly based on clinical judgment, i.e., the severity of symptoms and angiographic findings and should be weighed against added operative risks in patients with evidence of diffuse arteriosclerotic processes. If surgery has been decided upon the most common approach is to construct a bypass graft between the aorta and the SMA at a site distal to the occlusion. It may sometimes prove difficult to insert the graft into the abdominal aorta if it is involved by severe arteriosclerosis, but usually a site can be found around the origin of IMA.

The results of bypass operations for chronic intestinal ischemia have been assessed from six recent and reasonable large series comprising 188 patients (Reul; 1974; Hansen, 1976; McCollum, 1976; Hollier, 1981; Stoney, 1977; Zelenoch, 1980). The overall postoperative mortality was 7% but more than 90% of the survivors obtained symptomatic relief (from 90 to 96%). The average length of follow up was from 5 to 10 years in these series and late deaths averaged 21%, which is not surprising as these patients are elderly with systemic arteriosclerosis. Until recently, it was the customary surgical practice only to bypass a single vessel, usually the SMA, although most patients have multiple vessel involvement. In a retrospective analysis of 56 patients operated on at the Mayo Clinic, it was observed that the recurrence rate correlated with the number of arteries revascularized. The recurrence rate was 50% when only one artery was bypassed, but only 11% if all occluded vessels were revascularized. Most vascular surgeons now agree that all occluded vessels should be bypassed. Both the celiac artery and the SMA can be bypassed with a single bifurcated graft inserted into the abdominal aorta with one limb placed in the celiac and the other limb into the SMA.

Recently, transluminal angioplasty has been attempted to dilate stenosis of the celiac artery or the SMA and further experience may prove that this approach has a place in treatment of chronic intestinal ischemia.

## Ischemic Colitis

Ischemic colitis was not recognized as a disease entity until 1966 when Marston described a series of patients with vascular disorders and characteristic colon lesions suggestive of ischemia. Ischemic colitis has since been described by many groups and has been accepted as a separate entity and constitutes an important differential diagnosis in chronic inflammatory bowel disease (Crohn's disease and ulcerative colitis) especially in the older population.

The lesions observed in ischemic colitis is a result of local tissue hypoxia, which leads to destruction of the mucosal integrity with secondary infection when the denuded or ulcerated mucosa is exposed to the high concentration of bacteria present in colonic contents. Two areas of the colon is especially vulnerable to ischemic injury: the splenic flexure and the

sigmoid where the anastomotic supply by collaterals may be poorly developed. The location of ischemic colitis in 117 patients is illustrated in Fig. 24

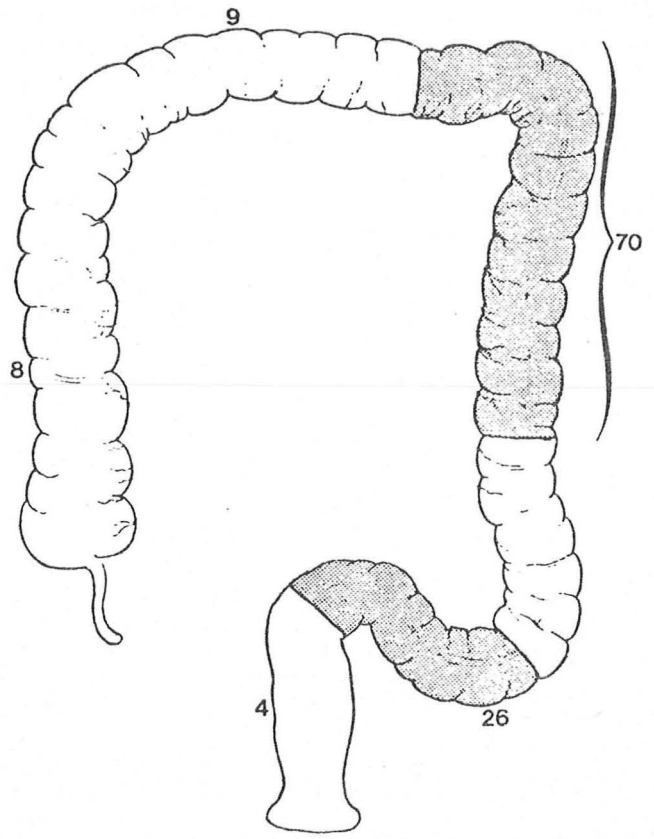



Fig. 24: Location of ischemic injury in 117 patients with ischemic colitis (Saegesser, 1981).

and demonstrates the predominance of left colonic involvement.

As in other intestinal ischemic injuries colonic ischemia is most often associated with occlusive diseases of the major splanchnic arteries and severe arteriosclerotic heart disease. In these patients the colonic perfusion may be just adequate when they are compensated. Any event which results in decompensation (congestive heart failure, myocardial infarction, digoxin toxicity, hypotension, etc.) may seriously affect splanchnic circulation and focal ischemia may develop. The reduced splanchnic perfusion and tissue ischemia may both result in vasoconstriction, which further aggravates the ischemia. Another factor which may contribute to local ischemia is colonic distension due to increased intraluminal pressure. Ischemic lesions have been observed in patients with neoplastic colon obstructions in an area proximal to the lesion.

Ischemic colitis may follow three different patterns according to severity and have been termed: 1) transitory, 2) stenotic, and 3) gangrenous ischemic colitis. It is the duration of the ischemic episode that will determine whether a given patient will develop transitory colitis or frank gangrene.

Transitory ischemic colitis is the mildest form of ischemic injury of the colon and is often a diagnosis of exclusion. Typically, the patient is in the sixth or seventh decade with known arteriosclerotic heart disease and recent decompensation who suddenly develops left lower quadrant pain and bloody diarrhea. Physical examination will show tenderness in the left lower quadrant and bloody stools on rectal examination. Laboratory studies are nonspecific with moderate leucocytosis as a general finding. The differential diagnosis at this point will include bacterial colitis, chronic inflammatory bowel disease, and diverticulitis. A KUB is usually normal but may occasionally show thumbprinting in one area of the colon. Barium studies have previously been a very useful diagnostic tool in the diagnosis of ischemic colitis and the typical apperance has been well described in many reports. The earliest radiologic sign of ischemia is thumbprinting which is a series of blunt projections into a narrowed loop of colon. The pathological basis for this characteristic sign is submucosal edema and hemorrhage. Thumbprinting may be present as early as three days after the onset of symptoms and may disappear within two days or persist up to several weeks depending on the severity of the ischemic injury. If the ischemic injury is severe mucosal irregularity and ulceration may be demonstrated on barium enema. The changes seen at this stage are very similar to those seen in ulcerative colitis and also Crohn's disease, and the radiological findings may not allow a distinction between these diseases.

Colonoscopy has not been advocated as a diagnostic tool in ischemic colitis for fear of perforation of the ischemic segment. Recently, several limited series have been published where colonoscopy was performed without incident and furthermore appeared superior to barium enema. In a series of 15 patients with suspected ischemic colitis nine had normal barium enemas whereas all 15 had distinctive findings on colonoscopy (Scowcroft, 1981). In the acute state (day 1-3) patchy, edematous and hyperemic mucosa alternate with more pallor areas. The hyperermic areas are friable. Over the next two days small (2-4) mm) superficial ulcerations develop, and the appearance is often indistinguishable from ucerative colitis. In the subacute stage (day 3-7), the ulcers enlarge in a longitudinal or serpiginous pattern (1-2 cm wide, 3-4 cm long) much like the ulcers seen in Crohn's colitis. Sometimes the ulcers are covered by an inflammatory exudate forming "pseudomembranes," and the condition may be confused with pseudomembranous colitis. From this point there is usually progressive healing within the course of a few weeks to several months and repeat colonoscopy will demonstrate normal mucosa or a fine granular pattern. The rapid resolution of the colonoscopic finding with supportive therapy alone will help to distinguish ischemic colitis from chronic inflammatory bowel disease, which require specific treatment. Unfortunately, there are no distinct histological features on biopsy specimens to confirm ischemic injury, and the diagnosis is still most often made retrospectively based on the transient nature of the disease. In the acute situation, however, it is often impossible to make a correct distinction between ischemic colitis and CIBD, and the diagnosis will be based on clinical judgment alone.

The incidence of chronic inflammatory bowel disease shows a bimodal distribution with a peak in the third and fourth decades and a second smaller peak in the sixth and seventh decade (Fig. 25).

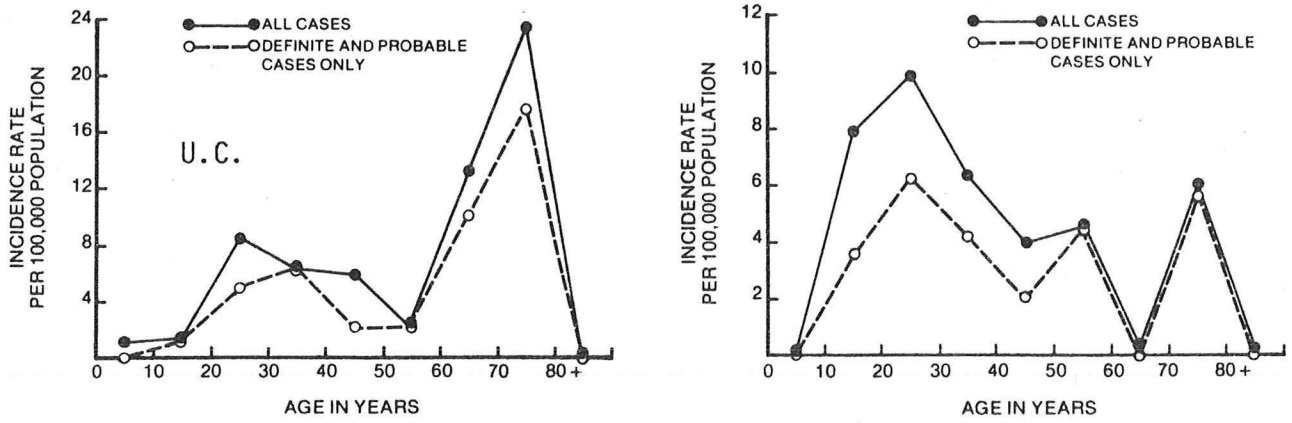



Fig. 25: Age-related incidence of ulcerative colitis and Crohn's disease in white males (Garland, 1981)

It has recently been questioned whether this second peak indeed represents a true increase in the incidence of CIBD in the older population and whether possibly ischemic colitis has been misdiagnosed as CIBD. In a retrospective analysis of 81 patients with colitis where the symptoms started after the age of 50, it was found that 60 patients (75%) had ischemic colitis (Brandt, 1981). In this analysis stringent clinical, radiological and pathological criteria were used to discriminate between ischemic colitis and other types of colitis. As shown in Table II,

Table II

| | Initial Diagnosis | Changed to Ischemic Colitis | Final Diagnosis |
|---|---|---|---|
| Ischemic Colitis | 31 | - | 60 |
| Ulcerative Colitis | 30 | 15 | 15 |
| Crohn's Colitis | 11 | 5 | 6 |
| Diverticulitis | 3 | 3 | 0 |
| Nonspecific Colitis | 6 | 6 | 0 |
| Total | 81 | 29 | 81 |

ischemic colitis was the initial diagnosis in 31 patients but the final diagnosis in 60. Ulcerative colitis and Crohn's disease were the initial diagnosis in 41 patients (50%) but the final diagnosis in only 21 (25%). The message from this retrospective study is that ischemic injury is the most frequent cause of colitis in the elderly and that ischemic colitis is often misdiagnosed as CIBD. These findings, however, need to be confirmed in a prospective study.

Angiography is rarely indicated in ischemic colitis since most cases are observed in nonocclusive disease.

If the ischemia has been more severe or prolonged, the necrotic process will involve the muscle layers, and the healing process may result in stricture formation. In two large series of ischemic colitis (Marston: 122 patients; Saegesser: 117 Patients) a fibrotic stricture developed in about 30% of the patients. The strictures may be short or involve a long segment of the colon. In both series the ischemic lesions occurred in the left colon in about 70% of cases. In many instances the development of ischemic strictures will not cause any symptoms and surgical intervention is only indicated when these strictures are associated with recurrent bleeding or obstructive symptoms.

The progression to a gangrenous lesion is rare in ischemic colitis. It occurred in only 2 out of 122 patients in Marston's series and in 21 of 117 (18%) in Saegesser's series. These patients will present with positive peritoneal signs and usually require immediate surgical resection of the gangrenous segment of colon with primary colostomy and later reestablishment of colonic continuity. In Saegessers series the mortality was 53% in the 21 patients operated for gangrenous colitis.

It is well recognized in the surgical literature that aneurysmectomy of the abdominal aorta may result in ischemic colitis with an incidence of 2-4%. With this operation the IMA is often ligated which is generally tolerated well. However, if the patient has significant occlusive disease of the SMA with development of collaterals through the arc of Riolan, the ligation of this artery may be crucial and result in colon ischemia. Thus, angiography is indicated prior to aneurysmectomy in order to define the splanchnic circulation. If SMA occlusion is demonstrated, these patients should have a bypass graft on the SMA, or reimplantation of the IMA into the aortic prosthesis in order to prevent development of ischemic colitis.

Celiac Axis Compression

Finally, in a discussion of ischemic syndromes of the intestinal tract, the celiac axis compression syndrome should be mentioned. The existence of this syndrome is still a controversial issue and has recently been critically attacked. It was originally described by Harjola in 1963 in a single patient with postprandrial pain and narrowing of the celiac axis on the angiogram which at operation was found to result from constriction by the celiac ganglion. The constriction was released, and the patient became pain-free. Since that time a multitude of case reports and small series have been published where the compression of the celiac axis was thought to result from either compression by the median arcuate ligament, or from constriction by the celiac ganglion. The median arcuate ligament of the diaphragm crosses the celiac axis at the level of the XII thoracic vertebra, and in most persons

some compression on the celiac axis can be seen on the angiogram during exspiration (Fig. 26).

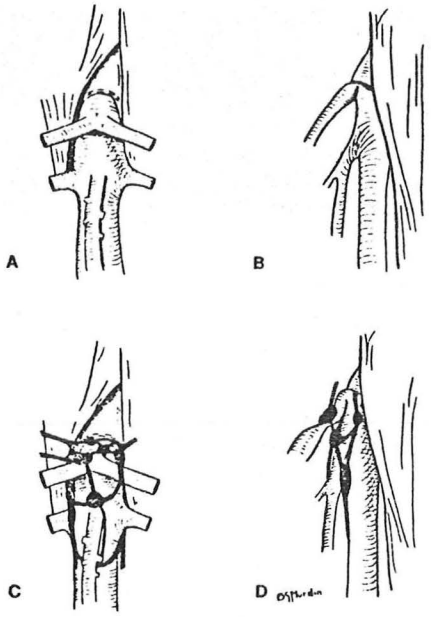



Fig. 26: Schematic illustration of the compression of the celiac artery by the median arcurate liagment and the celiac ganglion

The fibers from the celiac ganglion surrounds the celiac axis at its origin and may sometimes be joined by fibrous tissue to form a thick shield on the anterior aspects of the celiac axis. The patients are typically in a younger age group, often females, who complain of postpandrial pain for which no other cause can be found. Angiography is then performed as the ultimate test, and a narrowing of the celiac axis at its origin may be demonstrated. The critical issue is whether there is a causal relationship between the observed narrowing and the abdominal pain.

Szilagyi recently reviewed 157 angiograms from three groups of patients: One group with suspected intestinal angina, another group with various gastrointestinal diseases without pain, and a third group without gastrointestinal problems. The incidence of celiac axis narrowing was similar in all three groups (about 50%). Szilagyi does not accept the existence of the syndrome and suggests that the combination of celiac axis compression and postpandrial pain is casual rather than causal. Secondly, it is generally accepted that significant intestinal ischemia does not develop until at least two major vessels are occluded, and in this syndrome only one vessel is affected and only partly stenosed. Thirdly, in animal experiments a partial occlusion of the celiac axis will not result in ischemic injury when SMA and IMA are patent due to collateral circulation. The general consensus now is that the pain is not ischemic but may be neurogenic arising from the pulsatile pressure on the celiac ganglion.

The initial enthusiasm with which this syndrome was accepted has cooled considerably because long-term results have generally been poor. In a series

by Evans, which included 47 patients operated on for this syndrome, 83% were symptom free at six months postoperatively, but only 41% remained symptom free in a follow-up period from 3 to 11 years. Moreover, in another 12 patients who were not operated, 9 became pain free on symptomatic treatment. In several series it was observed that the postpandrial pain persists despite reestablishment of normal celiac blood flow, and, conversely, that patients improved dramatically despite persistence of celiac axis narrowing. The placebo effect of a laparotomy should not be underrated.

In a recent editorial in the Lancet on the celiac axis compression syndrome, it was concluded that the diagnosis should be made with great caution, and operation should only be undertaken after very careful thought.

## Summary

In summary, acute intestinal ischemia continues to carry a high mortality rate, but a more aggressive approach with early recognition and rapid therapeutic interventions has at least in some centers succeeded in reducing the mortality rate to about 50%. It is doubtful whether the mortality rate can be reduced even further as these patients often have severe cardiovascular disease.

It is evident, however, that a certain proportion of patients, who present with an acute ischemic catastrophe, have had symptoms of chronic intestinal ischemia for several months preceding the acute incident. The recognition and diagnosis of chronic intestinal ischemia remain difficult. The presence of meal-related abdominal pain and angiographically documented occlusion of two of the main splanchnic arteries is suggestive evidence but not necessarily diagnostic. A functional test of the capacity of the splanchnic circulation to increase intestinal perfusion during a meal may be helpful to define patients at risk. Although the disease is rare, it is important to make the diagnosis since reconstructive vascular surgery is of proven benefit, and surgery may also prevent development of acute ischemia at a later date.

Finally, ischemic colitis is the most frequent cause of colitis in the older population. The differential diagnosis against chronic inflammatory bowel disease remains difficult, since clinical and colonoscopic findings are indistinguishable, and the diagnosis is most often made retrospectively based on the transient nature of the ischemic injury.

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