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NONTHROMBOTIC INTESTINAL INFARCTION IN HEART DISEASE

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Over a period of 3 years, a group of patients were observed at necropsy with widespread hemorrhagic infarction of small and large bowel, in which there was no evidence of organic vascular occlusive disease of the mesenteric vessels. The pattern of the areas of infarction did not correspond to the anatomical distribution of any of the large mesenteric vessels. The hemorrhagic necrosis was limited to the mucosa and did not involve the muscular or serosal layers. In no instance was there infarction of the mesentery of the involved portion of the intestines. In no case was there evidence of serous fluid in the peritoneal cavity as often occurs in organic vascular occlusion of a large mesenteric vessel associated with infarction of the bowel. Cardiac disease was a factor common to all these patients.

The association of nonthrombotic intestinal infarction with various types of heart disease (Berger and Byrne[2]) and hemorrhagic shock (Glotzer and Show[11], Haber, Brown and Schneider[12], Klemperer, Penner and Bernheim[17], Lillehei[19], Longerbeam *et al.*[21]) has been observed recently. Acute generalized and widespread necrosis of the gastrointestinal tract has been noted in acute myocardial infarction (Katz[16], Ming and Levitan[22]), aortic insufficiency (Hoffman, Zimmerman and Cardwell[14]), luetic aortitis (Hoffman, Zimmerman and Cardwell[14]), arrhythmias (Irving and Corday[15]), congestive heart failure (Corday *et al.*[3], Ende[8]), and digitalis therapy (Gazes *et al.*[10]).

This report relates our experience in 10 fatal cases seen in a 3-year period. The clinical manifestations and pathologic findings in these patients have been evaluated, and in this paper emphasis is placed on those features which could help identify this catastrophic event.

Case Reports. CASE 1. H.R. (D-57384). A 41-year-old white man was admitted to the hospital because of chest pain and increased dyspnea of several hours' duration. He had known rheumatic heart disease with mitral stenosis, questionable aortic insufficiency, pulmonary emphysema and atrial fibrillation.

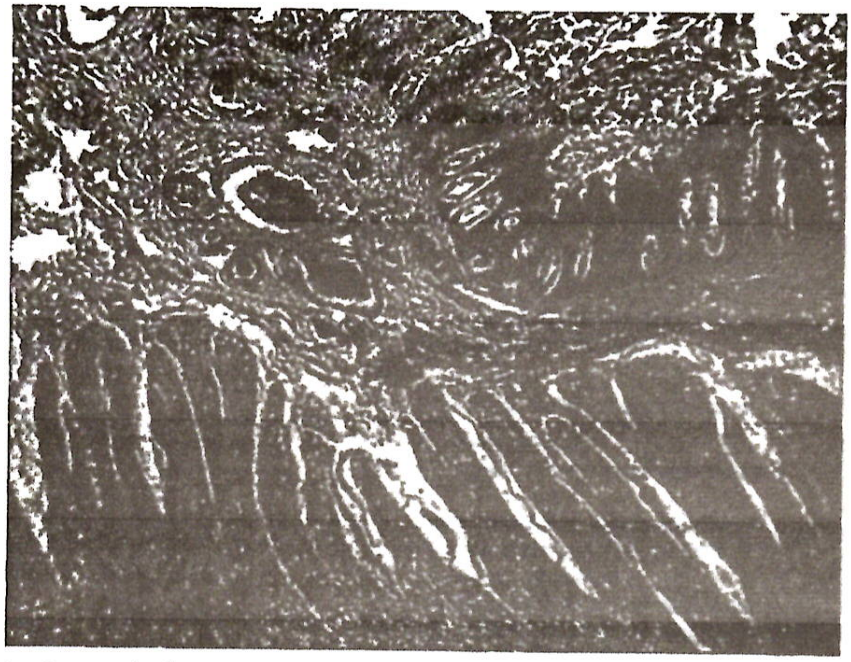

Fig. 1.—Section of colon (Case 1) showing necrosis and hemorrhagic suffusion into the mucosa. The submucosal blood vessels are dilated, but the muscular layer is unaltered. (× 35)

Examination on his final admission showed increased pulmonary congestion, hepatomegaly and peripheral edema. The leukocyte count was 19,220. Shortly after admission, he developed abdominal pain with a tender board-like abdomen, and his legs were noted to be cold and pulseless. Metaraminol was given because of a drop in blood pressure and a "shock-like" picture. The patient died 6 hours later.

Pathologic diagnoses included: old rheumatic heart disease (980 gm.) with mitral stenosis; partially obturating parietal thrombus on the atrial side of the mitral valve with subacute bacterial endocarditis; moderate edema of the lungs; bilateral hydrothorax; ischemic reddish-purple discoloration of the skin of the scrotum and both lower extremities; edema of the colon with a 15 cm. area of infarction in the sigmoid region; dilatation, edema, reddish-brown discoloration of the jejunum and ileum; blood-tinged fluid in the dilated intestines and stomach; segmental hemorrhagic necrosis of the colonic mucosa and advanced necrosis of the mucosa of the small bowel.

CASE 2. H.G. (D-11639) An 80-year-old white man was admitted to the hospital because of abdominal pain of one week's duration. He had a medical history of congestive heart failure and had been taking digitalis for 2 years. Shortly after admission the patient began to vomit. On examination, his abdomen was distended and peristalsis was diminished. There was tenderness in the right lower quadrant. A flat plate of the abdomen showed a dilated stomach and distention of the small and large bowel. The leukocyte count was 19,500. A Levine tube was inserted and the patient's abdomen became flat, but tenderness persisted in the right lower quadrant. The patient seemed to be improved, when suddenly he went into shock. A vasopressor drug was given, but the patient expired a few hours later.

Pathologic diagnoses included: mild bilateral hydrothorax; dilatation, edema and purplish-red discoloration of the colon and of the entire small intestine; ischemic necrosis with leukocytic infiltrations in the mucosa, submucosa, and inner muscular layer of the small bowel.

CASE 3. P.D. (D-7695) A 69-year-old white woman was admitted to the hospital because of abdominal pain of 3 days' duration, associated with vomiting and diarrhea. The pain was intermittent at first, but became constant and much more severe on the day of admission. There was a previous history of hypertensive heart disease with congestive heart failure. She had been taking digitalis for 2 years.

The morning after admission the patient abruptly went into shock and the abdomen became rigid and diffusely tender. The peripheral pulses were normal. A roentgenogram showed the small and large bowel to be distended. The leukocyte count was 28,750. Vasopressor drugs were given, but the patient remained hypotensive and expired 8 hours later.

Pathologic diagnoses included: moderate hypertrophy of the left ventricle (450 gm.), mild coronary atherosclerosis; dilatation, edema and purplish-red discoloration of the colon and small intestine; blood-tinged fluid in the bowel lumen; ischemic necrosis of the musosa with leukocytic infiltrations and colonies of bacteria in the small intestine and colon.

CASE 4. L.H. (D-43790) An 82-year-old white woman was admitted to the hospital because of upper abdominal pain, nausea and abdominal distention of several days' duration. She had a history of congestive heart failure and was well compensated on digitalis.

On examination, there were signs of congestive heart failure, atrial fibrillation and a diastolic murmur at the apex. The abdomen was markedly distended and there was the suggestion of a mass in the right upper quadrant. Peristalsis was diminished. An abdominal flat plate was interpreted as showing signs of diffuse ileus. The leukocyte count was 6,250.

Because of a drop in blood pressure, she was given metaraminol. On the third hospital day, she developed cyanosis of all four extremities. The cyanosis persisted and by the fifth day the right lower extremity and right hand became cold and pulseless. Prior to death, the lower right leg and right hand became gangrenous.

Pathologic diagnoses included: moderate hypertrophy of the left ventricle of the heart (520 gm.); moderate mitral stenosis; ascites; bilateral hydrothorax; early ischemic gangrene of right foot and right hand; moderate dilatation and purplish discoloration of the ileum; hemorrhagic necrosis of the mucosa of the ileum.

CASE 5. A.S. (D-51760). An 86-year-old white woman was admitted to the hospital because of abdominal pain, nausea and vomiting of 4 hours' duration. There was a medical history of hypertension and "poor circulation" in the lower extremities. There was no history of congestive heart failure or of digitalis therapy. On examination, the abdomen was distended and there was tenderness in both lower quadrants with decreased peristalsis. There was a grade III apical systolic murmur and rales at both bases. An electrocardiogram showed supraventricular tachycardia (140 per minute). A flat plate of the abdomen showed calcification of the aorta and distention of the stomach and small and large intestines. The leukocyte count was 23,150. Three days after admission, the patient became hypotensive and semiconscious. Metaraminol was given, but the patient expired shortly thereafter.

Pathologic diagnoses included: moderate concentric hypertrophy of the left ventricle of the heart (560 gm.); advanced coronary atherosclerosis; moderate dilatation and gangrenous necrosis of the mucosa of the small bowel.

CASE 6. C.K. (D-5632). A 63-year-old white woman was admitted to the hospital because of the sudden onset of severe pain in the right calf. There was a history of rheumatic heart disease with mitral stenosis, atrial fibrillation and congestive failure for which she required digitalis. The patient had been on long-term anticoagulant therapy.

On examination, there was a grade II diastolic murmur at the apex, and signs of congestive heart failure were present. The abdomen was soft and nontender. Femoral pulses were present bilaterally. The left leg was surgically absent at the midthigh, and the right leg was cold below the midcalf.

The anticoagulant therapy was continued. On the 14th hospital day, the patient developed sudden onset of crampy abdominal pain, became hypotensive, and developed cyanosis of the extremities. Metaraminol was given. The abdomen was distended, with diffuse tenderness and board-like rigidity. The leukocyte count which had been normal on admission rose to 26,750.

Anticoagulants were stopped, and an exploratory laparotomy was performed. The entire colon and approximately 80% of the small bowel were found to be gangrenous. The abdomen was closed and the patient expired on the following day.

Pathologic diagnoses included: rheumatic heart disease; mitral stenosis with a ball valve thrombus in the left atrium of the heart; dilatation of the small intestine and colon with edema and reddish-purple discoloration of the wall; hemorrhagic necrosis with extensive polymorphonuclear leukocytic infiltrations in the mucosa, submucosa and inner muscular layers of the large and small bowel.

CASE 7. B.F. (D-45746). A 67-year-old white woman was admitted to the hospital with a chief complaint of epigastric pain, nausea and vomiting of 48 hours' duration. The patient had a history of myocardial infarction 4 years previously and required digitalis for congestive heart failure. Anticoagulant therapy had been continued since the infarction.

On examination atrial fibrillation, with a slow ventricular rate, was present. The abdomen was moderately distended. There was

tenderness in the epigastrium, but peristalsis was normal. The right leg was surgically absent. Both femoral pulses were normal. The nail beds were cyanotic. An electrocardiogram was interpreted as showing evidence of subendocardial infarction. The leukocyte count was 32,400. Twenty-four hours after admission the patient's temperature rose to 103° F. and there was an associated shock-like picture. A Gram-negative septicemia was suspected. In spite of intensive therapy, including antibiotics and pressor amines, the patient expired on the following day.

Pathologic diagnoses included: mild hypertrophy of the heart (450 gm.); recent subendocardial infarction in the posterior wall of the left ventricle; moderate mitral stenosis; surgical absence of the right leg; dilatation, edema and reddish discoloration of the small bowel, descending and sigmoid portions of the colon; blood-tinged fluid in the intestinal lumen and stomach and hemorrhagic necrosis with extensive polymorphonuclear leukocytic exudation in the mucosa of the intestine.

CASE 8. S.G. (D-38153). An 81-year-old white woman was admitted to the hospital because of acute abdominal pain and a syncopal episode. She had known hypertension and coronary occlusive disease and had required digitalis for a period of 4 years.

On physical examination, there was venous distention of the neck veins, moist rales at both lung bases and a grade III systolic murmur over the entire precordium. The abdomen was distended and tender. There was no rigidity. The lower extremities were edematous, cold and cyanotic. The patient continued to do poorly, became hypotensive, and expired on the following day. This patient did not receive pressor amines.

Pathologic diagnoses included: moderate hypertrophy of the left ventricle of the heart (630 gm.); moderate coronary atherosclerosis; bilateral hydrothorax; dilatation of stomach, small bowel and colon with blood-tinged fluid; hemorrhagic necrosis and leukocytic infiltrations in the mucosa of the stomach and small intestine, and colonies of bacterial organisms in the mucosa of the small bowel.

CASE 9. J.L. (D-40985). A 74-year-old white man was admitted to the hospital because of an acute onset of pain and discoloration of the right foot. He had a myocardial infarction 2½ years before and required digitalis for congestive heart failure.

On admission, the blood pressure was 80/40. Marked signs of congestive heart failure were present. His abdomen was diffusely tender and rigid, but not distended. The peripheral pulses were present in the left leg, but absent below the femoral on the right. The right leg was mottled, cyanotic and cold. The leukocyte count was 18,500. The patient remained in shock despite pressor therapy and expired within a few hours.

Pathologic diagnoses included: marked hypertrophy of the left ventricle of the heart (700 gm.); advanced coronary atherosclerosis; an old healed infarct in the anteromedial wall of the left ventricle; moderately advanced calcific aortic stenosis; distention, purplish-red discoloration and blood-tinged fluid in both small and large bowel; hemorrhagic necrosis with heavy polymorphonuclear leukocytic infiltrations in the mucosa of large and small intestines.

CASE 10. A.C. (D-37328). An 85-year-old white woman was admitted because of cough and dyspnea of 3 to 4 weeks' duration. She was a known diabetic for many years. She had taken digitalis for congestive heart failure, but stopped the medication on her own volition.

Examination at the time of admission revealed obesity and signs of marked congestive failure. The leukocyte count was 9,850.

The patient improved on redigitalization. On the 8th hospital day, she developed acute abdominal pain with nausea and vomiting. The abdomen showed marked distention with generalized tenderness and rigidity. The lower extremities were cold and cyanotic. A flat plate of the abdomen showed dilatation of the stomach, small bowel and colon. The leukocyte count was 35,700. The patient became hypotensive. Metaraminol was given, but the patient expired 4 hours later.

Pathologic diagnoses included: moderate hypertrophy of the left ventricle of the heart (500 gm.); marked coronary atherosclerosis; marked distention of the intestines with blood-tinged fluid in the lumen; extensive hemorrhagic necrosis and heavy polymorphonuclear leukocytic infiltrations in the mucosa of the colon and small intestine.

PATHOLOGIC FINDINGS. All patients had in common, widespread hemorrhagic infarction of the small intestine, with dilatation, purplish-red discoloration and necrosis of the bowel mucosa. In 8 patients there was also involvement of the colon seen as segmental hemorrhagic infarction. The most prominent change in the microscopic study was the advanced hemorrhagic necrosis of the bowel musosa. Moderate polymorphonuclear leukocytic exudation was seen in the mucosa and submucosa of the involved bowel. There was marked dilatation of the submucosal and subserosal blood vessels. In 2 instances, colonies of bacteria were seen in the

ischemic mucosa. Essentially, the structural change was a moderately advanced hemorrhagic necrosis of the mucosa seen mainly in the small intestine and to a lesser degree in the colon.

Eight of the 10 patients in this series had moderate to marked hypertrophy of the heart. Five patients had old rheumatic heart disease. In 4 of these, mitral stenosis was demonstrated. One had an associated ball-valve thrombus; another had a partially obturating valvular thrombus which was complicated by subacute bacterial endocarditis. One individual showed calcific aortic stenosis. Advanced coronary atherosclerosis was present in 3 of the other 5 patients. In one of the last group there was a recent myocardial infarction.

There was no evidence of mesenteric vascular obturation due to thrombi or emboli in any of these patients. In each instance, the mesenteric arteries and veins were found to be patent after careful dissection. The mesentery was not involved in the process of infarction.

The abdominal aorta showed moderate to advanced sclerosis; lipid plaque formations and limesalt deposition in the vessel walls. Although the mesenteric vessels were moderately sclerotic, the morbid changes did not produce any significant reduction in the lumina. The areas of distribution of the hemorrhagic infarction did not follow the anatomical distribution of any mesenteric vessel or vessels.

The widespread involvement of both large and small intestines, as seen in the majority of our patients, could not have been the result of organic occlusion of any major vascular system.

PATHOGENESIS. Hemorrhagic necrosis of the intestinal mucosa is a retrogressive change related to reduced blood flow in the small and large bowel. Impaired cardiac function with reduced output, hypotension and shock can cause critical ischemia, in older individuals (Wilson and Qualheim[26]), who already may have only a marginally adequate intestinal blood supply. The resultant hypoxia may produce tissue death.

The question of whether mucosal necrosis is on the basis of mesenteric congestion due to increased portal venous pressure, or to mesenteric vascular angiospasm, has been resolved in a series of experiments by Corday and others[3,4,7]. These investigators measured the mesenteric blood flow and portal venous flow in hemorrhagic shock by means of a photoelectric dropmeter. Blood flow through the mesentery artery was reduced by vasospasm of the arterioles in the bowel wall. There was no evidence of splanchnic pooling or elevation of the portal venous pressure. The intestinal response to ischemia is an active spastic contraction which tends further to interfere with blood flow through the intestinal wall. Glotzer and Show[11] consider each organ to have its own specific vasomotor response. Constriction by the renal and mesenteric vessels is a compensatory mechanism in hypotensive shock, tending to prevent the reduction of blood flow to such vital organs as the brain and heart.

It would seem logical, in those patients who developed hypotensive shock as a complication of reduced cardiac output, that the mesenteric vascular insufficiency was largely the result of a compensatory spasm of the arterioles in the intestines. The prolonged intramural angiospasm led to ischemic necrosis of the mucosa.

A similar explanation would suffice for the peripheral vascular alterations seen in the extremities in 6 of our patients. The changes of coldness, cyanosis or actual gangrene reflect the reduced oxygenation of peripheral tissue as a result of reduced cardiac output, shock and vasospasm (Cotton and Bedford[5], Swan and Henderson[25]).

A role of "endotoxic shock" in helping to initiate the cycle of further hypotension has been suggested (Drucker *et al.*[6], Hall and Gold[13], Lillehei and MacLean[20]). Once the normal intes-

tinal barrier to the usual Gram-negative organisms is breached, absorption of endotoxins, or the possible development of a Gram-negative septicemia, may add to the shock state. The secondary role of such a process is apparent, since the vascular insufficiency must be of sufficient degree as to compromise the intestinal barrier to the Gram-negative organisms or their endotoxins.

COMMENT. The summary of the clinical data is contained in the accompanying chart. The age range of the 10 patients was from 41 to 86 years. If the youngest patient is omitted as an exception, the average age would be 76 years. Seven of the 10 patients were female.

CHART SUMMARIZING CLINICAL DATA IN 10 PATIENTS

TOTAL CASES	10
(AVERAGE AGE—72.8 YEARS)	
Congestive heart failure	9
Receiving digitalis	9
Drop in blood pressure—Shock (Below 90 mm. Hg-systolic)	10
Received pressor amines	9
Cardiac Arrhythmia*	6
Abdominal Signs and Symptoms:	
Severe pain	10
Tenderness—Rigidity	10
Nausea or vomiting	7
Distention	7
Decreased—Absent peristalsis	4
Roentgenographic evidence of reflex ileus†	4
Diarrhea	3
Melena	1
Leukocytosis	9
Cold cyanotic or gangrenous extremities	6
Dehydration	4
Elevated temperature	4
Known hypertension	4
On chlorothiazides	3
Received broad spectrum antibiotics	2
Taking Coumadin	2
Known diabetic	1

*4 had chronic atrial fibrillation
†Only 6 had abdominal flat plates taken

Prior to the terminal illnesses, all of the patients had evidence of cardiovascular disease. Only one patient did not have congestive heart failure requiring digitalis. This individual (Case 5) had hypertension and peripheral vascular disease. The high incidence (5 cases) of rheumatic heart disease in this elderly group of patients was surprising, particularly the frequency of "tight" mitral stenosis (4 patients).

All of the patients had abdominal pain, although the onset varied in relationship to the terminal episode. Six were admitted to the hospital specifically because of abdominal pain which had been present for 12 hours to 7 days. The other 4 patients were hospitalized because of cardiovascular disease and developed abdominal pain while in the hospital. None of the patients gave a prior history suggestive of "abdominal angina" (Sedlacek and Bean[23]).

The location and character of the abdominal pain, as described by the patients, were not uniform. In most, the pain was described as diffuse and severe. On examination, 5 patients had "board-like" rigidity of the abdomen and decreased peristalsis was present in 7 individuals. In 4 of the 6 patients on whom survey films of the adbomen were done, changes suggestive of reflex ileus were present. Nausea and vomiting were frequent (7 patients). The incidence of diarrhea (3 individuals) was not high and only one patient had melena. None had passage of red blood from the rectum. Despite the vomiting, clinical signs of dehydration were present in only 4 patients, preceding the terminal shock phase which occurred in all patients.

There was a high incidence of associated peripheral vascular changes. Six patients had changes in the legs described as coldness, cyanosis or actual gangrene. In 2 patients the changes were present on admission, and in the other 4 were noted as a near terminal event. One of the patients had gangrene of the right hand (Case 4). The association of cyanosis and gangrene of the lower extremities with myocardial infarction and congestive heart failure has been reported (Cotton and Bedford[5], Swan and Henderson[25]) however,

it has not been recorded as a common occurrence in hemorrhagic infarction of the bowel. Unfortunately for diagnostic clarity, hemorrhagic infarction of the bowel occurs frequently in clinical situations where embolization to mesenteric vessels cannot be excluded. The peripheral vascular changes can be misinterpreted easily as supportive evidence of an embolic etiology.

All 10 patients eventually developed a shock-like picture. Because of the hypotension, all but one patient received a pressor-amine, but only one received it for a prolonged period of time (3 days). During this period (Case 4) gangrenous changes appeared in the right hand and legs. The remaining 8 patients received pressor-amines for 2 to 12 hours prior to death. They all had abdominal pain and 5 had peripheral vascular changes before the administration of vasopressor drugs. It is unlikely that the pressor-amines caused either the hemorrhagic infarction of the bowel or the peripheral vascular changes. There is a strong possibility, however, that the administration of a vasopressor substance aggravated the peripheral and mesenteric vascular insufficiency both by the direct vasospastic action, and, with prolonged use, by decreasing cardiac output (Smulyan, Cuddy and Eich[24])

Only one patient had a normal white blood cell count. The other 9 individuals had a leukocytosis ranging from 18,000 to 35,700. Despite the elevated white blood cell count, the temperature curve was unpredictable. Half the patients had a subnormal temperature which rose to 102° to 103°F. terminally. The remaining half did not have a significant temperature elevation, but had an abrupt drop to subnormal levels just prior to death.

Only 2 of our patients had received broad spectrum antibiotics. Three individuals had been taking chlorothiazides, but none were of the enteric-coated variety with potassium, recently incriminated as causing ulceration of the small bowel (Baker, Schrader and Hitchcock[1]). Four patients had known hypertension. One patient was a diabetic. One patient had never taken digitalis, and there was no evidence of digitalis toxicity in those requiring the drug (Gazes *et al.*[10]). We do not think that the presence of hypertension, diabetes, diuretics, digitalis therapy or antibiotic therapy was of any significance in the etiology of hemorrhagic infarction of the bowel.

TREATMENT. The proper management of hemorrhagic infarction of the bowel is prevention. This is accomplished by the maintenance of an adequate circulating blood volume in the elderly cardiac patients. Congestive heart failure must be treated, arrhythmias terminated where possible, dehydration corrected and shock states avoided.

When the condition has occurred, attempts should be made to restore an effective circulating blood volume as rapidly as is consistent with the patient's basic cardiac disease. Intestinal intubation may prevent bowel distention and thus avoid further circulatory embarrassment (Corday *et al.*[3]). Appropriate antibiotic therapy helps prevent bacterial growth in the ischemic or necrotic bowel and may prevent secondary endotoxic shock. The intravenous administration of procaine, sympathetic blocking agents or other vasodilating drugs have been considered as a means of increasing circulation to the involved intestine (Drucker *et al.*[6]). However, there would be a theoretical objection to their use in that they might further lower systemic blood pressure. While the value of steroids has not been established, there is some rationale for their use because of the possible role of endotoxic shock.

Favorable results have been reported in correcting small bowel infarction with large doses of heparin followed by long-term Coumadin therapy (Frajndlich and Taylor[9], Laughman[18]). The occurrence of hemorrhagic infarction of the bowel in 2 patients (Cases 6 and 7), who were on anticoagulants, would raise strong doubt as to the effective-

ness of anticoagulant therapy in this condition.

Since metaraminol and similar vasopressor agents undoubtedly accentuate the mesenteric angiospasm, their use is probably not justified and should be avoided.

Ideally, surgery should be avoided in nonorganic infarction of the bowel. The severity of the cardiac disease and the profound shock will contraindicate surgical intervention in most patients. In the few individuals who survive the initial phase, exploratory laparotomy may have to be performed, because of the inability to exclude definitely embolic or thrombotic mesenteric vascular occlusive disease. A knowledge of this disorder will prevent a fruitless search for organic obturation, should exploration be done.

Summary. Ten cases of massive hemorrhagic infarction of the bowel associated with heart disease are presented. In none was there evidence of thrombotic or embolic mesenteric occlusive disease.

This condition occurs primarily in elderly cardiac patients and is a definite pathologic entity with a suggestive clinical picture. The elderly cardiac with a history of congestive failure who develops sudden abdominal pain, leukocytosis, hypotension, and clinical signs of diffuse bowel ischemia should be suspected of massive hemorrhagic infarction of the bowel. The frequent association of signs of peripheral vascular insufficiency can be misinterpreted as evidence of an embolic etiology of this intraabdominal catastrophy.

Treatment is primarily prevention, but therapeutic possibilities have been suggested.

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(Interlingua on page 628)