Chronic intestinal ischemia: Diagnosis and therapy

Paul E. Stanton, Jr., M.D., Paul A. Hollier, M.D., Terry W. Seidel, M.D., David Rosenthal, M.D., Michael Clark, M.D., and **Pano A. Lamis, M.D.,**
Johnson City, Tenn., and Atlanta, Ga.

Splanchnic arteriosclerosis is common among the elderly population, but intestinal angina is distinctly a rare entity. Extensive and efficient mesenteric collateral pathways make development of intestinal angina unlikely unless at least two major vessels exhibit hemodynamically important stenoses. Herein we describe the surgical management of 17 patients with chronic intestinal ischemia. The patients most commonly had postprandial pain and lost significant weight; angiography, including lateral aortography, confirmed the diagnosis. An average of 2.5 vessels in these 17 patients were arteriosclerotically involved. These 17 patients underwent 20 major splanchnic artery reconstructions altogether (average, 1.2 vessels per patient) for relief of symptomatic intestinal ischemia. Arterial reconstructions (16 bypass procedures and 4 endarterectomies) were undertaken with either autogenous saphenous vein (10 vessels) or Dacron prosthetics (6 vessels). Revascularizations involved the superior mesenteric artery (six patients), hepatic artery (three patients), splenic artery (seven patients), and inferior mesenteric artery (four patients). Five deaths occurred after operation, two early and three late, all from myocardial infarctions. All patients who survived have been relieved of their pain, and there has been no recurrence. The average length of follow-up has been 60.9 months and repeat angiography in six patients at intervals of up to 5 years has shown no evidence of revascularization occlusion. (J VASC SURG 1986; 4:338-44.)

When atherosclerotic constriction of the origins of the celiac and mesenteric arteries proceeds slowly, the eventual complete occlusion of these vessels does not always result in infarction of the small bowel. A profuse network of collateral channels may often maintain viability of the small bowel even with multilevel vessel occlusive disease.

Because almost all patients with chronic intestinal ischemia are admitted with abdominal pain and weight loss, the clinical significance of this prodrome lies in the fact that it may be a precursor of fatal mesenteric vascular occlusion.[1] Symptomatic intestinal ischemia as a result of splanchnic atherosclerotic occlusive disease is relatively uncommon, with the literature to date revealing a limited number of reports citing experience with 10 or more patients.[2-10]

The present report summarizes the clinical courses of 17 patients who underwent surgical intervention for chronic intestinal ischemia and outlines the presentation as well as diagnostic maneuvers leading to the prescribed surgical care (Tables I, II, and III). Careful follow-up to 9 years is reported.

PATIENTS AND METHODS

Seventeen patients, eight men and nine women, underwent evaluation and eventual operation for symptomatic chronic intestinal ischemia at either the Georgia Baptist Medical Center in Atlanta, Georgia or the Veterans Administration Medical Center (Mountain Home), Johnson City, Tennessee, between 1976 and 1985. The mean age of female patients was 65 years and the male patients was 64.5 years. Excluded from the study were nonatherosclerotic diseases of the splanchnic bed, including all forms of acute vascular insufficiency, fibrodysplastic diseases, entrapment syndromes, and inflammatory arteriopathies.

Clinical manifestations. The typical presentation of the 17 patients included abdominal discom-

From the Division of Vascular Surgery, Quillen-Dishner College of Medicine, East Tennessee State University, Johnson City, Tenn. (Drs. Stanton, Hollier, and Seidel) and the Division of Vascular Surgery, Georgia Baptist Medical Center, Atlanta, Ga. (Drs. Rosenthal, Clark, and Lamis).

Presented at the Tenth Annual Meeting of the Southern Association for Vascular Surgery, Cerromar Beach, Puerto Rico, Jan. 30-Feb. 1, 1986.

Reprint requests: Paul E. Stanton, Jr., M.D., Department of Surgery, Quillen-Dishner College of Medicine, P.O. Box 19, 750A, East Tennessee State University, Johnson City, Tennessee 37614.

Table I. Clinical presentation of patients with chronic visceral ischemia

Patient	*Age (yr)*	*Sex*	*Weight loss (lb)*	*Symptoms*	*Risk factors*			
					Cardiac disease	*COPD*	*Diabetes*	*Smoking*
A.M.	66	M	22/2 yr	Postprandial pain (2 yr); chronic abdominal pain (2 mo)	+	+	−	+
H.K.	76	M	60/1 yr	Postprandial pain (2 yr)	+	+	+	+
A.H.	62	F	35/2 yr	Chronic abdominal pain (6 mo)	+	+	−	+
V.M.	44	F	40/1 yr	Postprandial pain (2 yr)	−	−	−	+
A.B.	55	F	45/1 yr	Postprandial pain (12 mo); diarrhea (3 mo)	−	+	−	+
R.C.	44	M	30/6 mo	Postprandial pain (1 yr)/ chronic abdominal pain (4 mo)/diarrhea and melena (2 mo)	+	+	−	+
A.H.	81	M	40/9 mo	Postprandial pain (1 yr)	+	+	−	+
S.C.	78	F	30/1 yr	Postprandial pain (1 yr); chronic abdominal pain (2 mo)	+	+	+	+
I.K.	73	F	30/8 mo	Postprandial pain (8 mo)	−	−	−	+
D.H.	82	F	40/1 yr	Postprandial pain (1 yr)/ possible chronic abdominal pain (3 mo)	+	+	+	+
S.V.	71	F	20/6 mo	Postprandial pain (1 yr); nausea diarrhea (2 mo)	+	+	−	+
M.L.	56	F	12/6 mo	Postprandial pain (8 mo)/ chronic abdominal pain (3 mo)/diarrhea melana (1 mo)	−	−	−	+
O.N.	63	M	30/3 mo	Postprandial pain (3 mo); diarrhea (1 mo)	+	+	−	+
C.C.	57	M	20/1 yr	Postprandial pain (11 mo)/chronic abdominal pain (3 mo)/diarrhea (4 mo)	+	+	−	+
G.L.	63	F	30/6 mo	Postprandial pain (8 mo)	−	−	−	+
A.H.	65	M	25/4 mo	Postprandial pain (1 yr)	+	+	−	+
H.D.	66	M	32/6 mo	Postprandial pain (1 yr); chronic abdominal pain (3 mo)	+	+	−	+

COPD = chronic obstructive pulmonary disease.
NOTE: Plus sign indicates risk factor present; minus sign indicates absence.

fort, usually dull or aching in nature, appearing 20 to 30 minutes after a meal, and lasting from 1 to 3 hours. All 17 patients had markedly restricted their food intake because of the appreciated association of food ingestion with abdominal discomfort. The patients usually totally eliminated solid food ingestion in preference to liquids to obviate this problem. This change in dietary regimen led to an associated significant weight loss in all 17 patients. Eight patients eventually had chronic angina, in which the patient was never free of discomfort. These eight patients did have increased discomfort after any ingestion of solid food. The average weight loss in this study was 23.4 kg (range, 5.4 to 27 kg). Because of the significant weight loss, abdominal malignancy was often suspected. Other reported gastrointestinal complaints included nausea (one patient [6%]), diarrhea (six patients [35%]), and melena (two patients [12%]). Important physical findings were limited to the presence of an abdominal bruit in all 17 patients and signs of inanition in all patients.

Diagnosis. General roentgenographic and endoscopic procedures were performed in all patients but generally did not prove of value in the demonstration and recognition of intestinal ischemia (Table III). However, these procedures were beneficial in an exclusionary diagnostic fashion for other causes of abdominal pain and weight loss. Likewise, tests for intestinal absorption deficiencies, including provocative D-xylose studies, were of no diagnostic value in

Table II. Arteriographic results, treatment, and outcome for 17 patients with chronic visceral ischemia

	Arteriography of vessels involved					
Patient	*Celiac*	*SMA*	*IMA*	*Other*	*Operative management*	*Comments*
A.M.	Occ	Occ	Occ	90% left iliac stenosis + small AAA	AFB + endarterectomy with reimplantation + aortic aneurysmectomy	Good response at 2 mo had CVA
H.K.	Occ	Occ	Occ	+ small AAA	Iliac/SMA bypass (saph. vein) + aortic aneurysmectomy	Death at 1 mo of MI
A.H.	Occ	Occ	Occ	90% bilateral iliac stenosis, small AAA + Occ left renal artery	AFB + aortic aneurysmectomy + A/Hep (Dacron) + A/SMA	Death at 48 hr; MI
V.M.	Occ	50%	Occ	—	A/Sp (Dacron)	Good response to 9 yrs with 18 kg wt gain; repeat angio (normal) at 2 and 5 yrs
A.B.	75%	Occ	Occ	—	A/Sp (saph. vein)	Good response to 2 yr with 9 kg wt gain
R.C.	Occ	—	Occ	—	A/Sp (Dacron)	Good response to 4 yr; 11.25 kg gain, died of MI
A.H.	—	Occ	Occ	—	A/SMA (saph. vein)	Good response to 6 yr; 11.25 kg wt gain
S.C.	95%	75%	Occ	—	A/Sp (saph. vein)	Good response to 5 yr; 11.25 kg gain, died of MI
I.K.	50%	50%	Occ	6 cm AAA	Aortic aneurysmectomy and IMA endarterectomy with reimplantation	Good response to 5 yr; 13.5 kg wt gain
D.H.	—	90%	Occ	—	A/SMA (saph. vein)	Good response to 3 yr; 18 kg gain: anglo repeated and normal (1 yr); died of MI
S.V.	Occ	—	Occ	—	A/Sp (saph. vein)	Good response to 6 yr; 6.75 kg wt gain
M.L.	Occ	—	Occ	—	H/Hep (saph. vein)	Good response to 4 yr; 4.5 kg wt gain
O.N.	70%	—	Occ	Small AAA	Aortic aneurysmectomy and IMA endarterectomy with reimplantation	Good response to 5 yr; 9 kg wt gain
C.C.	Occ	—	Occ	—	A/Sp (Dacron)	Good response to 6 yr; wt stable repeat angio at 1 yr (normal)
G.L.	Occ	—	Occ	—	A/Sp (Dacron)	Good response to 5 yr; angio at 1 and 3 yr normal
A.H.	90%	Occ	Occ	—	A/SMA (saph. vein) A/Hep (saph. vein)	Good response to 7 yr; 9 kg wt gain
H.D.	90%	Occ	Occ	90% bilateral iliac stenosis	AFB + A/SMA (saph. vein) + IMA endarterectomy	Good response to 2 yr; 13.5 kg wt gain

SMA = superior mesenteric artery; IMA = inferior mesenteric artery; Occ = occluded; AAA = abdominal aortic aneurysm; AFB = aortofemoral bypass; CVA = cerebrovascular accident; A/Sp = aortosplenic bypass; MI = myocardial infarction; A/SMA = aorto-superior mesenteric artery bypass; A/Hep = aorto-hepatic artery bypass.

any of 10 patients in our series so tested. In all instances, the diagnosis depended on recognition of the distinct clinical syndrome superimposed on arteriographic documentation of advanced splanchnic arteriosclerotic occlusive disease. All patients underwent both anterior-posterior and lateral aortography. Lateral aortography was thought to be more useful in delineating proximal vessel occlusive problems than selective visceral arteriography. However, selective visceral arteriography was helpful in defining collateral conduits between splanchnic vessels and in assessing direction of flow along these conduits.

Pattern of disease. Extensive arteriosclerotic disease was observed in all patients. All patients had multivessel involvement (average, 2.5 vessels per patient) with disease evident in the celiac artery in 15

Table III. Special studies performed for chronic intestinal ischemia

Patient	*Barium study*	*Endoscopy*	*D-xylose absorption*
A.M.	–	–	–
H.K.	–	–	–
A.H.	+*	+†	Not done
V.M.	–	–	–
A.B.	–	–	–
R.C.	–	–	Not done
A.H.	–	–	Not done
S.C.	–	–	Not done
I.K.	–	–	Not done
D.H.	–	–	–
S.V.	–	–	Not done
M.L.	–	–	Not done
O.N.	–	–	–
C.C.	–	–	–
G.L.	–	–	–
A.H.	–	–	–
H.D.	+*	–	–

NOTE: Plus sign indicates positive results; minus sign indicates negative results.
*Left colonic "thumb printing" pattern of ischemic mucosal edema.
†Gray mucosa.

of 17 patients (9 of 17 total occlusions and 6 of 17 stenoses), in the superior mesenteric artery in 10 of 17 patients (6 of 17 total occlusions and 4 of 17 stenoses), and in the inferior mesenteric artery in 17 of 17 patients (17 total occlusions). All three splanchnic vessels were occluded in 3 of 17 cases.

Associated vascular disease was common. Included were 5 of 17 patients with associated abdominal aortic aneurysm, 3 of 17 patients with peripheral iliac-femoral arterial occlusive disease, 12 of 17 patients with known coronary artery disease, and 8 of 17 patients with cerebrovascular disease. Only 3 of 17 patients in this series were diabetic, but all were known long-term smokers. The average pack years for this group of smokers was 40.

Surgical management. Splanchnic arterial reconstructions were individualized depending on the anatomic pattern of occlusive disease, the character of existing collateral vessels, and the clinical status of the patient (Table IV). An average of 1.2 vessels per patient were revascularized so that 20 vessels altogether were reconstructed in all 17 patients. Bypass techniques were used in 14 of 17 patients with 16 total vessels grafted. These bypasses included six grafts to the superior mesenteric artery, three grafts to the hepatic artery, seven grafts to the splenic artery, and none to the inferior mesenteric artery. Multiple vessel revascularizations were done in two cases, both requiring bypass to both the hepatic artery and superior mesenteric artery. Endarterectomy as the sole procedure was used for the inferior mesenteric artery

Table IV. Operative management

	No. of patients(%)
Bypass technique (14/17 patients bypassed; 16 total vessels bypassed; 1.2 vessels/patient)	
Celiac artery bypass	
Hepatic artery	3 (19)
Splenic artery	7 (44)
Superior mesenteric artery bypass	6 (32)
Inferor mesenteric artery bypass	0 (0)
Endarterectomy (4/17 patients)	
Superior mesenteric artery	9 (0)
Celiac artery	0 (0)
Inferior mesenteric artery	4 (24)
Alone	3 (18)
With additional mesenteric bypass	1 (6)

Table V. Complications

	No. of patients	%
Death		
Early	2	12
Late	3	18
Morbidity		
Gastrointestinal	8	47
Renal	1	6
Cardiac	3	18
Pulmonary	5	29
Neurologic	1	6

in three cases and was performed at time of bypass to the superior mesenteric artery in an additional case. These endarterectomies all included direct vessel reimplantation into the aorta or an associated aortic graft. Neither the superior mesenteric artery nor the celiac trunk was endarterectomized in any of the reported 17 cases.

Bypass graft materials were either greater saphenous vein, used in 10 of 16 bypasses, or double-velour knitted Dacron, used in 6 of 16 vessel bypasses. Abdominal aortic aneurysmectomy was performed in 5 of 17 patients, with Y grafts placed for occlusive disease in 3 of 17 patients. Two deaths associated with the 17 operative procedures occurred in the early postoperative period (Table V).

RESULTS

The follow-up averaged 60.9 months and ranged from 2 to 108 months. All of the 15 patients who survived the initial postoperative period benefited from surgical intervention. Pain was ameliorated in all of these patients. Furthermore, all patients resumed normal dietary habits and 14 of 15 survivors gained weight in an expected fashion. Follow-up arteriographic studies were performed in 6 of the 15

patients who survived and confirmed graft or endarterectomy patency in all cases.

No patient died of mesenteric infarction during the postoperative period. The two early deaths occurred at 48 hours and at 30 days as a result of myocardial infarctions. In addition, three late deaths were all due to myocardial infarctions at 3, 4, and 5 years. One additional patient suffered a major cerebrovascular accident 2 months postoperatively.

DISCUSSION

Chronic visceral ischemia has been recognized relatively infrequently despite the prevalence of atherosclerosis in our aging population. Unlike acute mesenteric ischemia in which urgent revascularization is indicated to prevent or minimize visceral infarction, revascularization in chronic visceral ischemia is indicated to alleviate abdominal pain and to prevent the progression of chronic inanition. Such surgical intervention also serves to prevent the progression of chronic visceral ischemia to frank visceral infarction.

Intestinal angina is distinctly uncommon, yet mesenteric arteriosclerotic disease is an almost universal finding in older patients. The widespread distribution of splanchnic arteriosclerosis has been documented in autopsy studies of unselected populations and ranges from 35% to 70% of cases.[11-13] Reiner, Jimenez, and Rodriguez,[13] with the use of postmortem barium injections, showed extensive mesenteric arteriosclerosis in 69 of 88 sequential autopsies. Ostial stenosis of the celiac artery was noted in 86%, of the superior mesenteric artery in 55%, and of the inferior mesenteric artery in 70% of the autopsies.[13]

Chronic intestinal arterial occlusive disease has been recognized for more than a century. Chienne[14] described the anatomic pathologic features in a patient who died of a stroke in 1868. In 1901 Schnitzler was the first to associate the arterial disease with abdominal pain.[1] Connor[15] in 1933 compared the resultant syndrome with the angina of coronary artery disease. In 1936 Dunphy[16] reviewed autopsy cases of intestinal infarction and found that 7 of the 12 reported cases had prodromal symptoms of abdominal pain. He offered the admonition that ". . . the clinical significance of vascular pain in the abdomen lies in the fact that it may be the precursor of fatal mesenteric infarction." Mikkelson[17] in 1958 suggested a surgical approach to obstructive lesions of the superior mesenteric artery. Mikkelson described in detail the clinical manifestations of intestinal ischemia and the postprandial abdominal pain that is characteristic of intestinal angina. Since that time, postprandial abdominal pain characteristic of intestinal angina has been reported in more than 97% of all reported large series. Intestinal ischemia associated with a postprandial anginal pattern was a universal finding among our reported patients.

Weight loss, as was seen in all of our patients, has been frequently cited as an important component of the ischemic syndrome, noted in as many as 86% of previously described large series.*

Marston[19] in 1971 estimated that 1800 deaths each year were due to intestinal ischemia of all types. Most of these deaths are due to mesenteric embolization or low flow nonocclusive infarction. Acute mesenteric thrombosis superimposed on chronic occlusive disease accounted for only a small number of these deaths.

Although prior reports revealed a predilection for women in ratios of 2:1 to 4:1,[3,5,7,8] our present study did not confirm this and showed a female-to-male ratio of 1.1:1 (only 2 of 17 patients were from the Veterans Administration facility).

It is possible that proximal splanchnic arterial stenosis and occlusions develop as a secondary form of arteriosclerosis. Endothelial cushion defects or intimal dysplastic lesions may be the primary lesion, with arteriosclerosis occurring later.[7] Our present series, as well as many previous series, indicates an extraordinarily high incidence of associated cigarette smoking, even exceeding that usually seen in patients with more common arteriosclerotic disorders. The importance of smoking in the pathogenesis of splanchnic arteriosclerotic disease remains unknown.

The diagnosis of intestinal angina remains a clinical challenge, and it must be remembered that the diagnosis of intestinal angina does not exclude or preclude the existence of more common, nonvascular causes of abdominal pain. Conversely, if a diagnosis of mesenteric ischemia seems likely, despite the finding of a more common cause of gastrointestinal dysfunction, it should be pursued.

In the present series the average interval from onset of symptoms to diagnosis was approximately 1 year. Most of these patients underwent extensive gastroenterologic evaluation, including upper and lower barium contrast studies, endoscopy, computed tomographic scanning, and absorption studies.

There appears to be no test or study sufficiently sensitive to screen patients with suspected visceral ischemic pain. Furthermore, no pathognomonic laboratory test is available to confirm such a diagnosis, and although malabsoprtion studies have been re-

*References 1, 2, 4, 5, 7-10, 18.

ported helpful in isolated instances, this type of test does not appear to be of significant benefit. Weight loss in patients with severe arteriosclerotic occlusive disease of the splanchnic circulation is probably more a reflection of decreased food intake than of defect in absorption. At present, recognition of this disease remains dependent on identifying persons having classic symptoms of intestinal angina combined with weight loss. Despite the association of weight loss and postprandial abdominal pain in all 17 of our reported cases, only a few patients had intestinal ischemia as part of their initial differential diagnosis. Thus, the physician treating the elderly atherosclerotic population must maintain a high index of suspicion regarding intestinal ischemic patterns, and a patient so included in the differential diagnosis should undergo angiography including lateral aortography to document the presence of advanced splanchnic arteriosclerosis. Intravenous digital subtraction angiography may be of some use in screening patients with intestinal ischemia and has been reported beneficial by Baur et al.[10]

Treatment of patients with intestinal angina has been so uncommon, as well as so varied, as to preclude a rigid advocacy of any particular method of surgical intervention. Operation for mesenteric vascular disease in asymptomatic patients is probably not indicated because of the morbidity and mortality risks of surgery in the elderly atherosclerotic population.[10] In addition, the observation that isolated splanchnic arterial occlusions may be well tolerated for years has been reported.[10] An exception to this policy is made for the patient seen with asymptomatic visceral artery stenosis or occlusion who requires aortic surgery for an aneurysm or occlusive disease.[20] As pointed out by Baur et al.,[10] correction of splanchnic occlusive problems avoids the hazards of reoperation and the possibility of postoperative gut infarction.

Shaw and Maynard[21] reported the first superior mesenteric thromboendarterectomy for arteriosclerotic occlusive disease in the proximal mesenteric artery in 1958. Derrick and Logan,[22] in the same year, undertook the first mesenteric bypass with the use of an autologous iliac artery from the thoracic aorta to the celiac and superior mesenteric arteries. Stoney, Ehrenfeld, and Wiley[23] in 1977 reported early success with transthoracic endarterectomy of the visceral orifice and continue to recommend this method. Most other groups at the present time prefer the use of bypass grafting techniques. Some recommend that the graft originate from the supraceliac aorta to allow antegrade blood flow,[24,25] whereas others prefer the infrarenal aorta as an origin to allow retrograde flow.[2] We have preferred the bypass technique to endarterectomy but have used endarterectomy successfully on four occasions. We also prefer to originate the bypass from the infrarenal aorta, which avoids the need for suprarenal aortic clamping and dissection. We have used the common iliac artery on one occasion as the origin for grafting in a patient with a heavily calcified abdominal aorta.

The relative merits of autologous vein vs. prosthetic graft material are subject to controversy.[8,26,27] It appears that the relatively short, rigid prosthetic graft, exposed to high blood flow rates, have excellent patency rates in this area. Similarly, such grafts may be less likely to be deformed by shifts in the intestinal position with postural changes. Some authors recommend retroperitoneal positioning of the grafts to allow for more stability,[8] but they have not found this to be necessary for long-term patency in "free-floating" intraperitoneal grafts.

Single-vessel revascularizations are usually sufficient, as seen in the present series, to reverse intestinal ischemic symptoms, even when all three splanchnic vessels are diseased.[27-33] However, most contemporary authors recommend multiple-vessel revascularization as the optimal therapy when it is technically feasible and cite associated recurrent abdominal complaints with frequent revascularization failures as their reasoning.[3,8-10,23,25] Long-term benefits of these more extensive multiple reconstructive procedures may justify their use but this justification has not been found in the present study.

REFERENCES

1. Bergan JJ, Yao JST. Chronic intestinal ischemia. In: Rutherford RB, ed. Vascular surgery, ed 2. Philadelphia: WB Saunders Co, 1984:964-72.
2. Bergan JJ, Dry L, Conn J, Tripple O. Intestinal ischemic syndromes. Ann Surg 1969; 169:120-6.
3. Crawford ES, Maris JC, Myhre HO. Celiac axis, superior mesenteric artery, and inferior mesenteric artery occlusion. Surgical considerations. Surgery 1977; 82:856-66.
4. McCollum CH, Graham JM, DeBakey ME. Chronic mesenteric insufficiency: Results of revascularization in 33 cases. South Med J 1976; 69:1266-73.
5. Reul GJ, Wukasch DS, Sandiford FM, Chiarillo L, Hallman GL, Cooley DA. Surgical treatment of abdominal angina: A review of 25 patients. Surgery 1974; 75:682-9.
6. Rob C. Surgical diseases of the celiac and mesentery arteries. Arch Surg 1966; 93:21-32.
7. Stoney RJ, Wiley EJ. Recognition and surgical management of visceral ischemic syndromes. Ann Surg 1966; 164: 714-22.
8. Zelenock GB, Graham LM, Whitehouse WM, Erlandson EE, Kraft RO, Lindenauer SM, Stanley JC. Splanchnic arteriosclerotic disease and intestinal angina. Arch Surg 1980; 115:497-501.
9. Hollier LH, Bernatz PE, Pairolero PC, Payne WS, Osmond-

son PJ. Surgical management of chronic intestinal ischemia; a reappraisal. Surgery 1981; 90:940-6.
10. Baur JM, Malley DJ, Taylor LM, Porter JM. Treatment of chronic visceral ischemia. Am J Surg 1984; 148:138-44.
11. Derrick JR, Pollard JC, Moore RM. The pattern of arteriosclerotic narrowing of the celiac and mesenteric arteries. Ann Surg 1959; 149:684-9.
12. Demos NJ, Bahtuh JJ, Urnes PD. Comparative study of arteriosclerosis in the inferior and superior mesenteric arteries. Ann Surg 1962; 155:599-605.
13. Reiner L, Jimenez FA, Rodriguez FL. Atherosclerosis in the mesenteric circulation: Observations and correlations with aortic and coronary atherosclerosis. Am Heart J 1963; 66:200-9.
14. Chienne J. Complete obliteration of celiac and mesenteric arteries. J Anat Physiol 1869; 3:363-72.
15. Connor LA. Discussion of role of arterial thrombosis in visceral diseases based upon analogies drawn from coronary thrombosis. Am J Med Sci 1933; 185:13-8.
16. Dunphy JE. Abdominal pain of vascular origin. Am J Med Sci 1936; 192:109-13.
17. Mikkelson WP. Intestinal angina; its surgical significance. Am J Surg 1957; 94:262-9.
18. Eidemiller LR, Nelson JC, Porter JM. Surgical treatment of chronic visceral ischemia. Am J Surg 1979; 138:264-8.
19. Marston A. Acute mesenteric vascular occlusions. In: Boley SJ, Shwartz SS, Williams LF. Vascular diseases of the intestine. New York: Appleton-Century-Crofts, 1971:509.
20. Connolly JE, Kwaan JHM. Prophylactic revascularization of the gut. Ann Surg 1979; 190:514-22.
21. Shaw RS, Maynard DP. Acute and chronic thrombosis of the mesenteric arteries associated with malabsorption. N Engl J Med 1958; 258:874-8.
22. Derrick JR, Logan WD. Mesenteric arterial insufficiency. Surgery 1958; 44:823-9.
23. Stoney DJ, Ehrenfeld WK, Wiley EJ. Revascularization methods and chronic visceral ischemia caused by atherosclerosis. Ann Surg 1977; 186:468-76.
24. Fry WJ, Kraft RO. Visceral angina. Surg Gynecol Obstet 1976; 117:417-24.
25. Daily PG, Fogarty TJ. Simplified revascularization of the celiac arteries associated with malabsorption. N Engl J Med 1958; 258:874-8.
26. Rene GJ, Wukasch DS, Sandiford FM. Surgical treatment of abdominal angina: A review of 25 patients. Surgery 1974; 75:682-9.
27. Hertzer NR, Beven EG, Humphries AW. Chronic intestinal ischemia. Surg Gynecol Obstet 1977; 145:322-8.
28. Eklof B, Hoevlas J, Ihse I. The surgical treatment of chronic intestinal ischemia. Ann Surg 1978; 187:318-24.
29. Hansen JHB. Abdominal angina. Acta Chir Scand 1976; 142:319-25.
30. Morris GC, DeBakey ME. Abdominal angina. Diagnosis and surgical treatment. JAMA 1961; 176:89-92.
31. Connolly JE, Abrams HL, Kieraldo JH. Observations on the diagnosis and treatment of obliterative disease of the abdominal aorta. Arch Surg 1965; 90:596-606.
32. Jaffe MS. Status of abdominal visceral circulation via superior mesenteric prosthesis. Am J Surg 1971; 121:736-8.
33. Nunn DB. Chronic intestinal angina. A report of two patients treated successfully by operation. Ann Surg 1972; 175: 523-7.