Recognition and Surgical Management of Visceral Ischemic Syndromes

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PATHOLOGIC obstruction of the mesenteric arteries was first described a century ago by Chiene.[4] The term *abdominal angina* has since been applied to the symptom complex associated with chronic intestinal ischemia.[2] This syndrome was first successfully treated in 1958 by Shaw and Maynard,[14] who reported the removal of an atherosclerotic obstruction in the superior mesenteric artery. Fry and Kraft [6] and Morris and his associates,[12] in later reports of surgically treated patients, emphasized the importance of visceral ischemia as a cause of abdominal pain and the value of aortography in establishing a definitive diagnosis. Arteriosclerosis is probably the most common cause of symptomatic visceral artery obstruction.[9] Fibromuscular hyperplasia of the celiac axis has occasionally been reported as a cause of abdominal angina.[13] Dunbar and his co-workers [5] recently described several cases of visceral ischemia resulting from external compression of the celiac axis by the arcuate ligament of the diaphragm. In the present report observations on 14 patients operated upon for visceral ischemia at the University of California Medical Center, San Francisco, during the past three and a half years will be presented.

Clinical Material

In six patients arteriosclerotic obstruction or stenosis of one or more of the primary visceral branches of the abdominal aorta was diagnosed by aortography and confirmed at operation. Preoperative symptoms in five patients were similar to those discussed by Mikkelsen [11] in his description of abdominal angina. The presenting complaint in these 5 patients was epigastric cramping, aching pain which occurred within 30 minutes after eating and lasted for one to 3 hours. One patient had an unusual variation of the classical pain pattern. This 55-year-old woman, who had stenosis of the celiac axis and superior mesenteric arteries, had a 3-month history of severe recurrent attacks of pain in the right lower quadrant of the abdomen. The pain appeared suddenly, was unrelated to eating, and increased in intensity until relieved by morphine. During 3 hospital admissions for this acute pain, physical examination showed a soft, nontender abdomen and normal peristaltic sounds.

During aortographic examination in these patients, specific attention was directed toward defining the extent of occlusive disease in the 3 major vessels contributing to visceral arterial supply, that is, the celiac axis and the superior and inferior mesenteric arteries. Five patients had multiple lesions; in these patients, both the celiac axis and the superior mesenteric arteries were occluded or stenotic. Pressure gradients were abnormal in both vessels in 4 of the 5 patients. In the fifth, a gradient was present only in the superior mesenteric artery. Three of the 5 patients also had inferior mesenteric lesions.

Only one patient had occlusion of a single vessel. Aortography showed that the celiac axis was narrowed at its origin by approximately 60%. No pressure gradient across the palpable lesion could be demonstrated at operation. Because of this and the advanced arteriosclerotic involvement of the aorta, reconstruction was considered inadvisable. Although symptoms of recurrent epigastric pain continued, it is problematic whether the celiac lesion was a contributing factor.

Revascularization was performed in the 5 patients with multiple vessel involvement. Opera-

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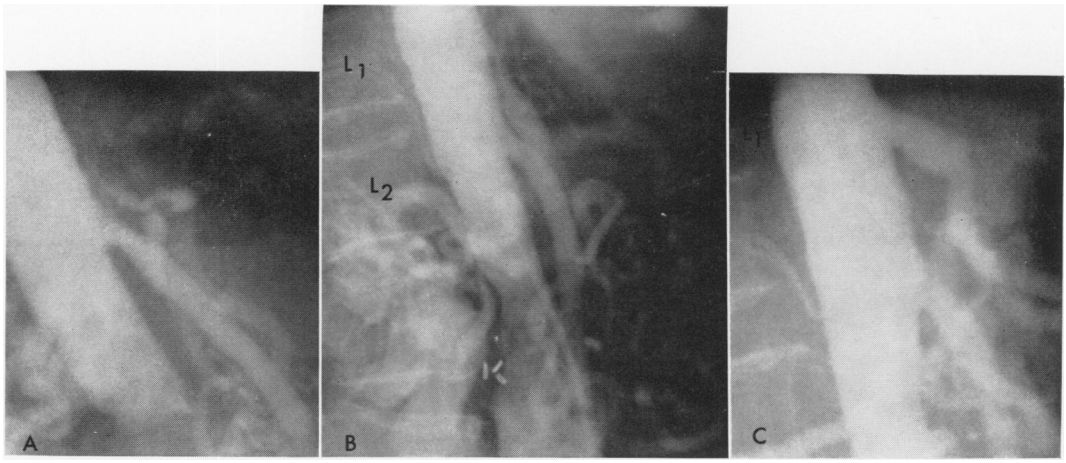

FIG. 1. Aortograms of a patient with arteriosclerotic stenosis of the celiac axis and superior mesenteric arteries and an aneurysm of the infra-renal aorta (A). Superior mesenteric endarterectomy at the time of aneurysmectomy restored a normal lumen (B). Later repair of the celiac lesion by autograft and replacement (C) was required to relieve recurrent symptoms of abdominal angina.

tions were limited to the celiac and superior mesenteric artery on the assumption that most patients can tolerate occlusion of the inferior mesenteric artery. Endarterectomy was the reconstructive technic used in three patients. The technic of endarterectomy varied, depending upon the problems of exposure and the degree of arteriosclerotic involvement. In one patient in whom the orifices of the celiac axis, superior mesenteric artery and both renal arteries were narrowed, all 4 vessels

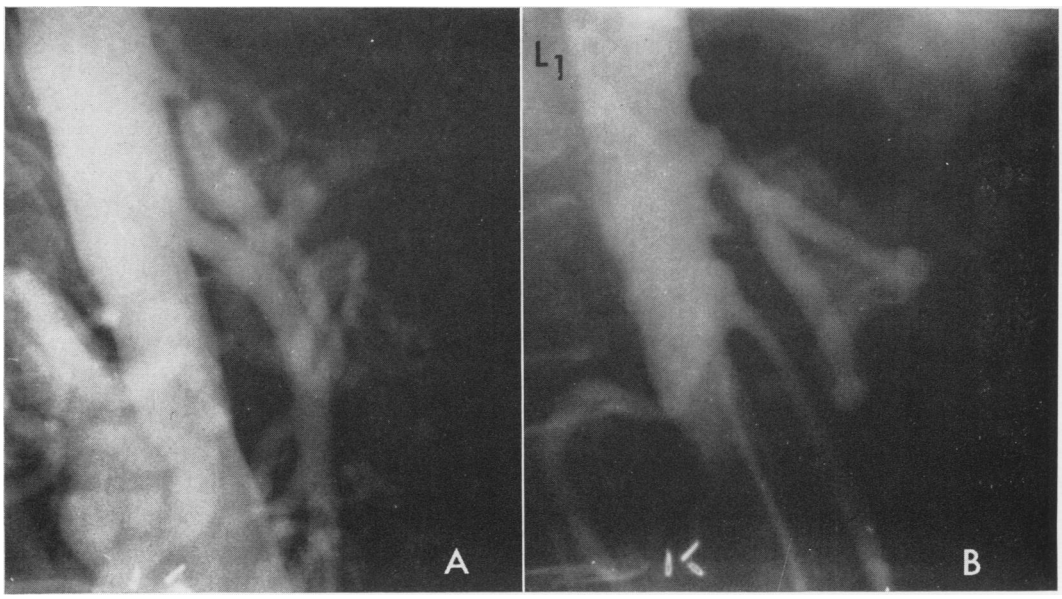

FIG. 2. Pre- and postoperative arteriogram of a patient with arteriosclerotic stenosis of the celiac axis and superior mesenteric arteries in whom the two arteries were reimplanted into the aorta at a favorable distal level. The superior mesenteric stenosis. although not demonstrated on the film was accompanied by a thrill and a pressure gradient. Note the collateral flow from the superior mesenteric artery to the celiac branches (A), and the reduction in size of the superior mesenteric artery postoperatively (B) once its requirement for collateral flow had been removed.

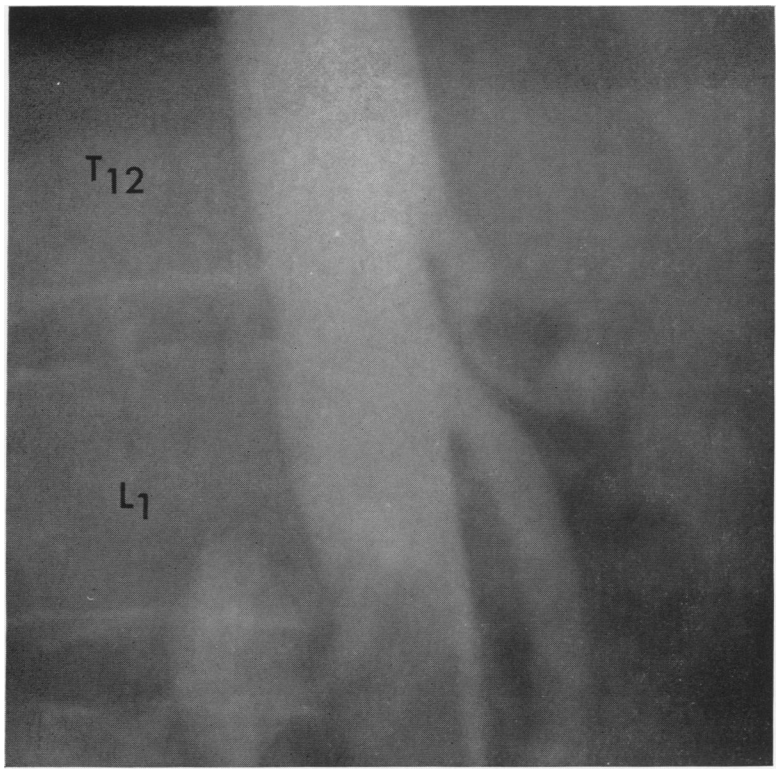

FIG. 3. Arteriogram in a 35-year-old woman with epigastric pain and an abdominal bruit showing the characteristic anterior compression defect in the celiac axis produced by the arcuate ligament of the diaphragm.

were opened by endarterectomy through two longitudinal aortotomies. In one patient who had an extensive lesion of the superior mesenteric artery and in whom a gradient was present only in this artery, endarterectomy was performed through a longitudinal superior mesenteric arteriotomy. Closure was supplemented with a vein patch. A third patient had stenosis of the superior mesenteric and celiac arteries and an infrarenal aortic aneurysm (Fig. 1). Aneurysmectomy and superior mesenteric endarterectomy gave symptomatic relief for 2 years. Abdominal angina then returned, and the proximal celiac artery was resected and replaced by an arterial autograft from the hypogastric artery. A 75-mm. pressure gradient disappeared after the grafting operation.

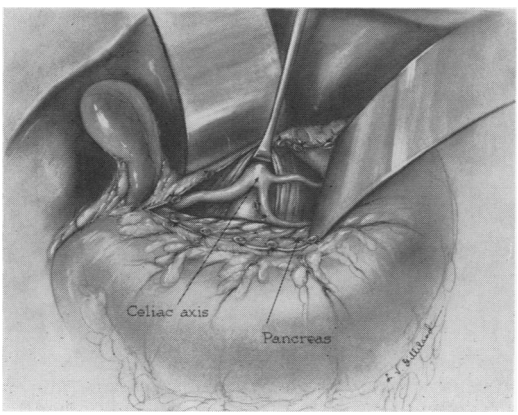

FIG. 4. Artist's drawing of the anterior approach to the celiac axis in a patient with diaphragmatic constriction of the celiac axis.

Arterial resection and reimplantation of the distal segment into the aorta was performed in 2 patients. In one of the patients both the celiac axis and the superior mesenteric arteries were anastomosed to uninvolved distal segments of the aorta (Fig. 2) Gradients of 75 mm. Hg. and 40 mm. Hg. were relieved. In the other patient, who had a long occluded segment of the superior mesenteric artery, only the celiac axis was reimplanted.

One death occurred in the 5 patients with arteriosclerosis who had undergone reconstructive operations. In the patient who died, gangrene of the mid-gut occurred after postoperative occlusion of the pancreaticoduodenal artery, which removed the collateral supply to the unrepaired, occluded superior mesenteric artery. Diffuse arteriosclerotic involvement of the celiac branches

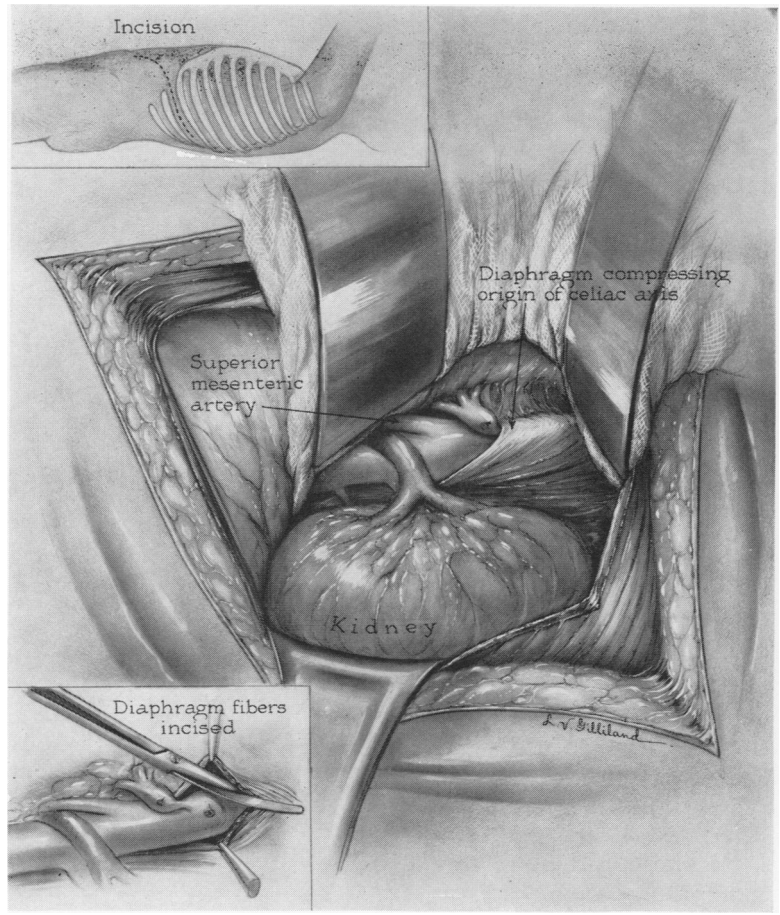

FIG. 5. Artist's drawing of the thoracoabdominal approach to the proximal abdominal aorta and incision of the arcuate fibers of the diaphragm. Note in the insert the appearance of the residual stenosis in the celiac axis.

had been recognized at operation. The 4 survivors have been relieved of pain and have regained their normal weight. Preoperative diarrhea in two of the patients has since ceased.

Continuing arterial patency of the operative segments has been confirmed by aortography in all patients except in the one who had a 4-vessel endarterectomy. In this patient, aortography 3 years after operation demonstrated recurrent asymptomatic occlusion of the celiac axis, and collateral flow to the distal celiac branches by way of branches of the superior mesenteric artery.

In six female and two male patients, visceral ischemic symptoms were considered to be due to constriction of the origin of the celiac artery by the diaphragm. Although the primary complaint in each case was epigastric pain, no common pattern of occurrence, duration or severity could be established. Distress varied from intermittent uncomfortable epigastric distention (two patients), to agonizing aching pain and cramping which required opiates for relief (4 patients). Attacks of pain occurred as often as 5 times a day in some patients, and only once a week in others. The pain lasted for one to 3 hours. Pain was never precipitated or aggravated by ingestion of food. Nausea, vomiting and diarrhea were rare. Studies of the kidney, gallbladder and gastrointestinal tract were always negative. A diagnosis of functional psychologic disturbance had been made previously in four of the patients. The only physical finding common to all patients was an epigastric bruit.

In all 8 patients, aortography showed stenosis of the proximal segment of the celiac axis by a defect on the anterior surface of the artery (Fig. 3). An abnormally high origin of the celiac axis, either at or above the level of the lower border of the 12th thoracic vertebra, was uniformly present. At operation, the artery was found to arise above the diaphragm and to emerge into the abdomen by passing beneath the median arcuate ligament of the diaphragm.

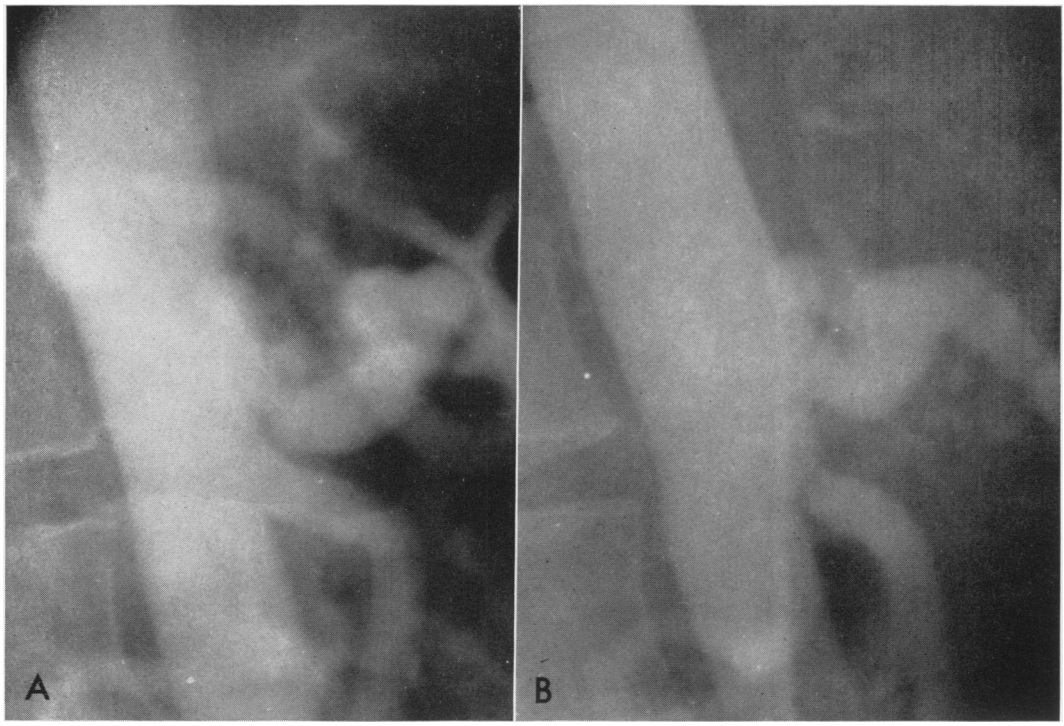

FIG. 6. Pre- and postoperative aortograms of a 50-year-old man with compression of the celiac axis (A). Relief of abdominal pain followed incision of the diaphragm, resection of the residual stenotic segment, and repair by simple end-to-end anastomosis (B).

This ligament was often no more than a collection of fibrous strands, forming an unyielding band which encircled the anterior aorta. Abnormal arterial pressure gradients varying from 20 to 60 mm. Hg were found in each patient at operation.

The primary purpose of operation in 6 patients was the relief of pain. In two patients for whom operation was planned for other reasons, preoperative aortography was performed to determine whether there was a relationship between existing symptoms of epigastric distress and an audible epigastric bruit. In these patients an incidental decompression of the celiac axis was performed to assess its value in relieving epigastric symptoms.

The celiac axis was approached either transabdominally or through a thoracoabdominal incision, depending upon the habitus of the patient (Fig. 4, 5). Operation in 4 patients consisted of simple division of the crural fibers encircling the aorta. Although the pressure gradients were relieved, mild hour-glass narrowing remained in the celiac arteries at the level of the former constriction.

Resection of the narrowed segment of the artery, in addition to incision of the diaphragm, was performed in the other 4 patients. The persistent, severe arterial narrowing was accompanied by a residual pressure gradient in two cases. Repair was accomplished by a simple anastomosis in three patients and autograft replacement in the fourth (the hypogastric artery was used as the donor vessel) (Fig. 6). Pressure gradients were absent at the conclusion of operation in these patients. Pathologic examination of the resected specimens showed fibrosis and intimal hyperplasia at the former site of constriction.

Two of the 4 patients with simple celiac decompression were relieved of symptoms; one is improved and one is unchanged. Postoperative arteriograms in the 4 patients showed persistence of the residual defect in 3 cases (Fig. 7). Symptomatic relief has been experienced by all 4 of the patients who had arterial resection.

Discussion

Mikkelsen,[11] in his discussion of the characteristics of abdominal angina, de-

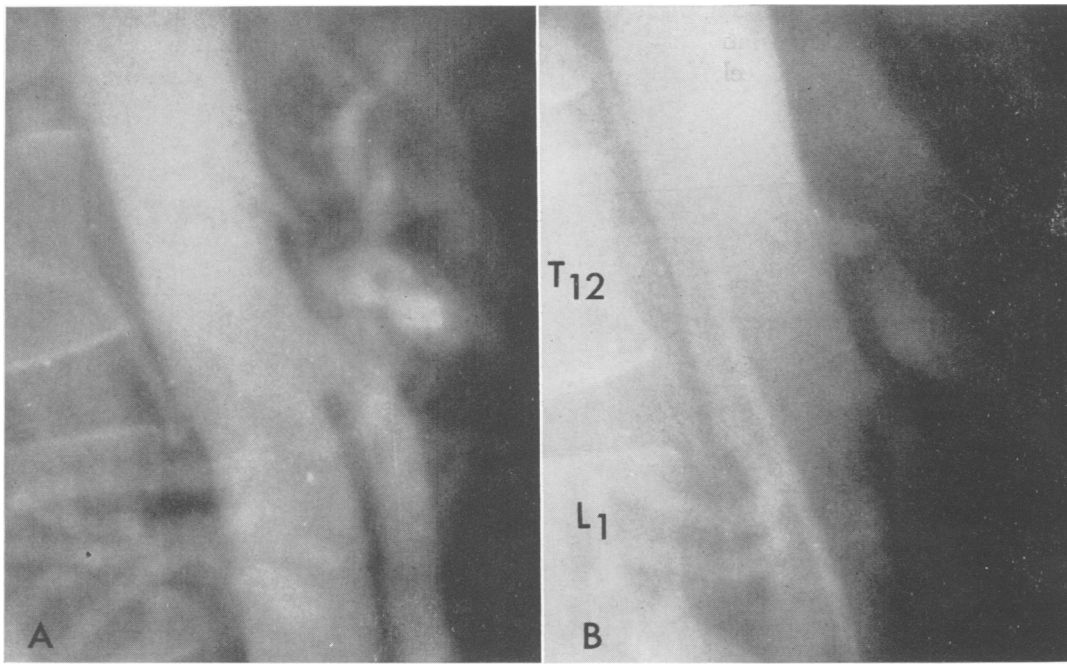

FIG. 7. Pre- and postoperative aortograms showing marked constriction of the celiac axis (A) only partially relieved by incision of the diaphragm (B). The preoperative pressure gradient, however, was relieved and the patient remains free of her former symptoms.

scribed post-prandial cramping abdominal pain, occasionally associated with a sense of distention. Mikkelsen commented that, "Distress usually develops 15 to 30 minutes after eating and persists from 1 to 3 hours. Its severity and duration depend upon the amount of food ingested. Nausea, vomiting, or diarrhea may occur. Reluctance to eat, along with malabsorption, may produce weight loss."

These symptoms were described by five of our six patients with visceral artery occlusive disease caused by arteriosclerosis. An estimation of the extent of visceral artery occlusive disease necessary to produce the classic pattern of pain of abdominal angina is difficult to make in the limited number of patients studied. Fry and Kraft [6] and Morris *et al.*[12] have suggested that more than one artery is usually involved. Four of our six patients who had symptoms considered typical of abdominal angina had lesions involving both the celiac axis and the superior mesenteric arteries. One patient who had postoperative celiac occlusion but whose superior mesenteric artery remained patent is free of symptoms. Another patient with involvement of both arteries failed to obtain permanent relief, however, until both vessels were reopened. Freedom from pain in patients with occlusion limited to the celiac axis probably depends upon the availability of collateral blood flow from the superior mesenteric artery via the pancreaticoduodenal collaterals.

Compression of the celiac axis by the diaphragm was reported as early as 1917 by Lipschutz,[9] who stated in part: ". . . the celiac axis is not infrequently covered at its origin by the diaphragm." Michels [10] in 1955 confirmed this observation and suggested that the constrictive action of the crura of the diaphragm may be responsible for the occasional diminished caliber of this vessel at its origin. The diaphragm

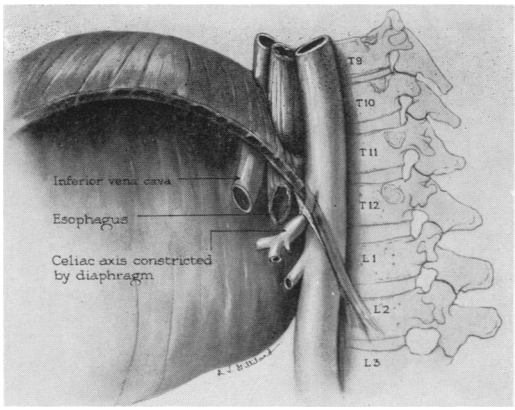

FIG. 8. Artist's drawing illustrating the constricting effect of the diaphragm on a high origin celiac axis.

usually encircles the aorta at the level of the T_{12}–L_1 interspace. Although the most common level of origin of the celiac axis is opposite the upper third of the first lumbar vertebra, anatomists have demonstrated that it has the widest variation in level of origin of all unpaired visceral branches of the abdominal aorta. In approximately 20 per cent of the cadaver dissections that have been reported, the celiac axis originated at or above the level of the twelfth thoracic vertebra.[3] George,[7] in reporting specifically on the relations of the celiac axis to the median arcuate ligament of the diaphragm, described a supradiaphragmatic level of origin of the celiac axis in eight of 38 patients. The high level of origin of the celiac axis and the constrictive action of the diaphragm are shown in Figure 8.

Aortographic studies on patients under study as candidates for prospective kidney donors in our own institution have demonstrated in two individuals an abnormally high origin of the celiac axis and proximal arterial constriction. Although neither patient complained of abdominal symptoms the appearance of collateral vessels from the superior mesenteric artery supplying the terminal branches of the celiac axis indicated that the celiac constrictions were impairing blood flow.

Dunbar *et al.*,[5] who reported the first observation of abdominal pain resulting from celiac axis compression by the diaphragm, found this disorder more prevalent in females. A similar prevalence is apparent in our own series. Adachi,[1] in a series of anatomic dissections, also found that a high celiac origin was more frequent in females.

It seems probable that celiac stenosis is frequently present in the absence of symptoms. Moreover, symptoms, when they do occur, often lack the specific characteristics of abdominal angina. The post-prandial pain pattern and the frequent diarrhea reported by Dunbar and his associates[5] in patients with this disorder did not occur in our eight patients. Aortography in patients with obscure abdominal pain syndromes and further observations on patients with celiac occlusive lesions are required before a clear understanding of the clinical significance of this disorder may be gained.

Summary

Clinical, arteriographic and operative findings in 14 patients with abdominal symptoms that were believed to be due to chronic visceral ischemia are described. Revascularization operations were performed in 13 of the 14 patients. Six patients had arteriosclerotic occlusive lesions of the visceral arteries. Five of the 6 underwent reconstructive procedures because of typical symptoms of abdominal angina and one of the 5 died. The four surviving patients have been asymptomatic up to 3½ years postoperatively. Continuing patency has been confirmed by aortographic examination.

Eight of the 14 patients had compression of the celiac axis by the diaphragm and had atypical abdominal pain. Decompression was performed in 4 of the 8 patients by division of the diaphragmatic fibers: 2 patients were relieved of symptoms, one

had some improvement and the fourth remained unchanged. The persistence of residual defects after operation was confirmed by postoperative aortograms. In the other four patients, operation included, in addition to division of the diaphragm, a resection of the stenosed celiac segment and arterial repair. All four patients are well and arteriograms demonstrate a normal celiac lumen.

Addendum

Since the preparation of this manuscript we have operated upon 3 additional patients with atypical abdominal pain and celiac stenosis secondary to diaphragmatic compression. Arterial reconstruction with the hypogastric artery was utilized in 2 patients, while in a third, who had an unsuitable hypogastric artery, the saphenous vein was substituted. All 3 patients have been relieved of symptoms thus far.

References

1. Adachi, B.: Das Arteriensystem der Japaner. Kyoto, Japan, 1928, p. 18, Band II.
2. Baccelli, Cited by Goodman, E. H.: Angina Abdominis. Amer. J. Med. Sci., n.s. **155**:524, 1918.
3. Cauldwell, E. W. and Anson, B. J.: The Visceral Branches of the Abdominal Aorta: Topographical Relationships. Amer. J. Anat., 73:27, 1943.
4. Chiene, J.: Complete Obliteration of Celiac and Mesenteric Arteries: Viscera receiving their Blood Supply through Extraperitoneal System of Vessels. J. Anat. Physiol. (London), 3 (second series) 65–72, 1868–1869.
5. Dunbar, J. D., Molnar, W., Beman, F. F. and Marable, S. A.: Compression of the Celiac Trunk and Abdominal Aorta. Amer. J. Roentgenol., **95**:731, 1965.
6. Fry, W. J. and Kraft, R. O.: Visceral Angina. Surg. Gynec. & Obstet., **117**:417, 1963.
7. George, R.: Topography of the Unpaired Visceral Branches of the Abdominal Aorta. J. Anat., **69**:196, 1934.
8. Laufman, H., Nora, P. F. and Mittelpunkt, A. I.: Mesenteric Blood Vessels: Advances in Surgery and Physiology. Arch. Surg., **88**: 1021, 1964.
9. Lipschutz, B.: A Composite Study of the Coeliac Axis Artery. Ann. Surg., **65**:159, 1917.
10. Michels, N. A.: Blood Supply and Anatomy of the Upper Abdominal Organs. Philadelphia, J. B. Lippincott Co., 1955, p. 140.
11. Mikkelsen, W. P.: Intestinal Angina: Its Surgical Significance. Amer. J. Surg., **94**:262, 1957.
12. Morris, G. C., Crawford, E. S., Cooley, D. A. and DeBakey, M. E.: Revascularization of the Celiac and Mesenteric Arteries. Arch. Surg., **84**:113, 1962.
13. Palumbinskas, A. J. and Ripley, H. R.: Fibromuscular Hyperplasia in Extrarenal Arteries. Radiology, **82**:451, 1964.
14. Shaw, R. S. and Maynard, E. P., III.: Acute and Chronic Thrombosis of Mesenteric Arteries Associated with malabsorption: Report of Two Cases Successfully Treated by Thromboendarterectomy. New Engl. J. Med., **258**:874, 1958.

Discussion

Dr. John Earle Connolly (Los Angeles): There are many aspects that I would like to discuss but I think that the most important one is diagnosis, and I would limit my discussion to mesenteric artery blocks due to arteriosclerosis. Of course, it is easy, as the authors have stated, to make the diagnosis when you have the classical findings of pain after eating, weight loss, and diarrhea, and an epigastric murmur. However, marked narrowing of the celiac and superior mesenteric artery take-offs may be present when these symptoms and signs are not.

The size of the central anastomotic artery connecting the inferior mesenteric artery and the superior mesenteric artery on a lumbar aortogram can be very helpful, I think, in the diagnosis of intestinal ischemia in those cases without symptoms.

(Slide) This slide demonstrates the size of the central anastomotic artery on the aortogram of a normal patient. (Slide) in this slide you see an aortogram demonstrating an enlarged central anastomotic artery occurring in a patient with intestinal ischemia. The lateral aortogram in this patient shows marked narrowing of the origins of the celiac and superior mesenteric arteries. (Slide) Now, for comparison, here is a greatly enlarged central anastomotic artery that we have seen in about a dozen cases now in routine aortograms over the past 3 years. This type of patient invariably has marked narrowing of the superior mesenteric and celiac axis take-offs also, but in our experience does not have any symptoms of intestinal ischemia.

Actually, this patient with the greatly enlarged central anastomotic artery was an obese woman. Our belief is that this artery, the central anastomotic artery, is able to dilate and elongate enough in some instance to take over the blood supply of the two stenotic arteries. Such patients then have no symptoms of ischemia.