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Aorto-enteric Fistula Following Abdominal Aorta Surgery

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AORTO-ENTERIC FISTULA
FOLLOWING ABDOMINAL AORTA SURGERY

by

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I certify that I have read this thesis and that in my opinion it is fully adequate, in scope and quality, as a thesis for the degree of Master of Science.

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PREFACE

My interest in the cause and prevention of an aortoduodenal fistula became acute on the morning of October 12, 1957. I was observing the post-mortem findings of an eighty-year-old man who had literally bled to death in the space of seventy-five minutes while his wife and I looked on. Nine days before I thought we had rescued him from such a fate when an arteriosclerotic abdominal aortic aneurysm and an aortoduodenal fistula had been resected. An orlon prosthesis replaced the resected aorta. I hated to lose what I felt I had every right to keep and gain.

Acknowledgement must be made for a certain measure of patience and resignation on the part of my wife and children while husband and father was absent from the family circle while working with dogs and journals.

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INTRODUCTION

The formation of a fistula between the abdominal aorta and the gastrointestinal tract is not a common event. This is in contrast to the incidence of rupture of an abdominal aortic aneurysm. In 1939 Lipshutz[1] reported two cases to make a total of 429 reported cases of ruptured abdominal aneurysm. In the succeeding two decades many more ruptures have been reported.

In 1950 Bagnuolo[2] reviewed the literature since 1843. He found 111 reported cases of nontraumatic aortic perforation into the gastrointestinal tract and added a case of tuberculous perforation into lungs, aorta and esophagus. Forty-five of the perforations were due to rupture of an aortic abdominal aneurysm. Salman[3] reported the first case of this type of rupture although Chamel and Dalmas[4] made previous mention of the condition. Nine years later only twenty-two additional cases were reported.[5]

Males have this problem three times oftener than females. Perforation usually occurs at points of contiguity, whether these be due to pathological fixation or anatomical fixation. The most common site of rupture (81.3 per cent) is some portion of the duodenum with the third portion being involved in 68.8 per cent of the sixty-seven cases. The age incidence varies from twenty to eighty-one years.

Some of the other points of perforation should be mentioned. Disease of the esophagus is a common cause of aortic fistula. This may be due to erosion of a carcinoma[2] or of a foreign body.[6] A few cases of stomach fistulization have been reported.[7] Most were secondary to an

esophagogastric anastomosis placed in the vicinity of the aortic arch.[8] The jejunum is a rare site of fistulization.[9]

In order to relieve pain and prevent rupture and fistulization several methods of dealing with the aneurysm sac were developed. In one ingenious procedure several meters of special alloy wire was inserted within the aneurysm and coagulation achieved by an electric current.[10] Wrapping the aneurysm with Ivalon[11] and polythene[12] was suggested later. These wrappings were hard to apply and were also found to be of very doubtful value.

The rapid changes and wonderful advances in vascular surgery the past decade are best illustrated by two articles on aneurysm treatment by Blakemore. The first one[13] extols the virtue of banding the aorta proximally with polythene and rubber bands plus the electrothermic wiring of the aneurysm. The second one[14] written four years later announces the abandonment of the procedure!

The birth date of the modern method of treating abdominal aortic aneurysms could well be December 9, 1953, and the birthplace the Southern Surgical Association meeting in Hot Springs, Virginia. DeBakey and Cooley[15] presented fifty consecutive cases in which aortic resection and homograft replacement was done. Twenty-six of these were for abdominal aortic aneurysms. In the discussion the authors were congratulated for their monumental work and for initiating a new era in the treatment of aortic aneurysms. As is the usual case others had gone before and paved the way. Hopfur[16] in 1903 achieved the first successful transplant of an arterial homograft when he implanted a femoral artery into the carotid artery of another dog.

The contributions of Carrel[17, 18] must not be overlooked. He

repeatedly demonstrated the feasibility of arterial and venous homografts in the experimental animal. The blood vessel suture technic he developed was reliable. Further impetus to the clinical application of arterial homografts and arterial anastomosis was supplied by Gross[19] in his treatment of coarctation of the thoracic aorta. Finally a Frenchman must be given credit for being the first to resect an abdominal aortic aneurysm and inserting a homograft. Dubost[20] did his epic operation on a fifty-year-old white male March 29, 1951. A left thoracoabdominal incision was used. The source of the homograft was the thoracic aorta of a twenty-year-old female who had expired three weeks before. The right iliac suture line was end to end with the graft while on the left the iliac end was sutured to the side of the graft. An aortagram after two months revealed good function. Clinically the patient was well after five months.

The etiology of aneurysms of the abdominal aorta has changed in the last twenty-five years. Kampmeier[21] found syphilis to be the most frequent specific cause in seventy-three cases. A majority of his patients died within six months of onset of symptoms. The increasing incidence of arteriosclerotic abdominal aneurysm has been ascribed to a decrease in cardiovascular syphilis and an increase in the age of the population. Arteriosclerosis accounts for 96 per cent of the abdominal aneurysms.[22]

In view of the serious and at times fatal complications that can occur following the resection of aneurysms and the insertion of grafts, one might wonder what would happen if nothing were done. A brief survival study would be in order. A widely quoted report of 102 abdominal aortic aneurysms was first presented in 1950 by Estes.[23] Ninety-seven

of these were considered to be due to arteriosclerosis. Of these 49.2 per cent survived three years or more. Only 18.9 per cent survived five years or longer. This survival time was lower than that of a normal population sixty-five years of age. In another survey of ninety-six aneurysms of the aorta, iliac and other abdominal branches, 80 per cent of the patients died in less than a year. Within the first month after symptoms appeared 30 per cent had died. Vascular rupture was the cause of death of 49 per cent who had abdominal aneurysms.[24]

Wright[25] chided the surgeons for being so eager to resect any and all abdominal aneurysms. However, he concluded by suggesting that each case be analyzed in terms of its own suitability for resection surgery. He felt that the great majority can be operated upon successfully.

A report on 101 aortic aneurysms by Roberts[26] indicated that one-half of patients with aortic abdominal aneurysms died within a year of onset of symptoms. One-half of those dying were found to have sustained a rupture of their aneurysm.

INCIDENCE OF AORTO-ENTERIC FISTULAE FOLLOWING ABDOMINAL AORTA SURGERY

The first reported resection for aneurysm of the abdominal aorta with insertion of homograft was recorded in 1952.[20] Four years later there appeared in print a report of a disastrous complication of aortic resection surgery. This is aorto-enteric fistulization. Humphries[27] recorded three instances of aortoduodenal fistula. This tragedy occurred on the fifteenth day, sixteenth day, and fourth month post-operatively. Freeze-dried homografts had been used for replacement. Because of the disparity between the host lumen and the homograft lumen a V-shaped wedge of host aorta was removed anteriorly and the cut edges reapproximated. This portion of the suture line at post-mortem examination was thought to be the site of the fistula.

Sixteen months following insertion of a nylon aortic bifurcation graft a sixty-six-year-old male noted weakness and bloody stools. He bled for several days. One episode of hemorrhage required a three-liter transfusion of whole blood. Gastrointestinal films were negative. Autopsy examination revealed a fistula between the right iliac suture line and the terminal ileum.[28]

The Committee for the Study of Vascular Prosthesis of the Society for Vascular Surgery reported two cases of aorto-enteric fistula brought to their attention in answer to the questionnaire sent to each member of the Society.[29] The first patient died of bleeding into the ileum from the iliac suture line of a nylon fabric prosthesis. The other patient showed evidence of a fistula eight months following

surgery in which an orlon fabric prosthesis replaced the aorta. The site of the difficulty was the proximal end of the prosthesis and the third portion of the duodenum.

Two instances of fistulization of an Ivalon prosthesis have been observed. The first[30] occurred six months following aneurysm resection and was located between the anterior surface of the graft and the overlying duodenum. The second[31] occurred in the same location fourteen months following resection surgery.

Another type of prosthesis was incriminated in fistula between the ascending portion of the duodenum and the surface of an Amylon graft. This occurred twelve months following placement of the graft.[32] The proximal suture line of a braided orlon prosthesis was found to be connected to the third portion of the duodenum six months following placement of the graft. For a week the patient had severe upper gastrointestinal bleeding.[33]

Posterior surface rupture of a homograft has occurred with the formation of a retroperitoneal false aneurysm which in turn ruptured into the duodenum. The difficulty occurred sixteen months following insertion of a homograft.[34] Sharf and Acker[35] noted the late development of a fistula between a bypass homograft and the jejunum. In another instance a homograft was replaced by a nylon graft because of ischemia of an extremity. Three months later the proximal suture line bled into the duodenum.

Three and a quarter years following the placement of a homograft,[36] massive quantities of blood were vomited and also passed by rectum. At surgery the bleeding was found to be between the graft and the third portion of the duodenum. The homograft was resected and

replaced by a braided nylon graft. Three other cases of homograft perforation were reviewed by Lawton and associates.[37] The interval between homograft placement and fistulization was three years, thirty months, and eight months. Boyd and Pastel[38] recorded their experience with two cases of fistula between the proximal suture line of a homograft and the distal duodenum.

Two[39, 40] of the Case Records of the Massachusetts General Hospital were concerned with massive gastrointestinal hemorrhage associated with a fistula between a Teflon-Dacron prosthesis and the duodenum. Both of these had secondary repair. Successful resection of two ruptured abdominal aortic aneurysms with replacement by homografts was accomplished by Elliot and associates[41] only to have fatal duodeno-homograft rupture take place six months and thirty-three months later.

Three late deaths, two at 1 1/2 years and one at 2 1/2 years following insertion of aortic homografts, were attributed by Sheranian and associates[42] to the formation of a false aneurysm that subsequently ruptured into the retroperitoneal portion of the duodenum. In reviewing the fate of ninety homografts DeWeese and associates[43] found two patients who expired as a result of rupture of the anterior surface of the homograft into the jejunum. It was felt that the site of fistulization corresponded to the site of ligated arterial branches of the homograft. A sixty-two-year-old male noted copious rectal bleeding five years following resection of an aneurysm of the aorta which was replaced by a bifurcation homograft. It took two laparotomies to locate the site of bleeding. The bleeding point was between the mid ileum and the anterolateral surface of the graft.[44]

Without much comment Julian and associates[45] record three cases

of spontaneous rupture of an abdominal aortic aneurysm into the duodenum. The aneurysms were resected, the duodenum closed and arterial homografts placed. Infection of the graft occurred and rupture resulted into the graft or into its suture line. One of these patients lived four months before final rupture. Julian[46] reports two more cases of aorto-intestinal fistula as a result of a pulsating hematoma of the suture line. One had an arterial homograft and the other had a plastic prosthesis.

The Baylor University group have recorded their evaluation of late failures after reconstructive arterial operations.[47, 48] One of thirteen patients with orlon taffeta tube aortic prosthesis died six months after surgery from hemorrhage associated with erosion into the duodenum. Six other patients had aortoduodenal fistulae. One died of hemorrhage almost immediately. In three cases the fistula occurred twelve to twenty-six months after insertion of homograft. No infection was present so homograft replacement was made. In two cases bleeding was caused by infection which allowed erosion (of Dacron tube in one case and braided nylon tube in the other) into the duodenum. The fistulization occurred at the proximal suture line.

Cordell and associates[49] encountered a rupture of an aortic endarterectomy suture line into the third portion of the duodenum. Their second case died following several operations for control of a fistula between the proximal suture line and the duodenum. The Dacron bifurcation graft had been in place ten months. Twenty-two months after the replacement of an aortic aneurysm with a homograft a fifty-eight-year-old male began to vomit large quantities of blood. After several operations directed at the gastrointestinal tract the patient died and an

aortoduodenal fistula was found at the proximal suture line.[50]

Szilagyi[51] mentioned two instances of aortoduodenal fistulae following implantation of arterial homografts. He feels that this complication is the result of a failure of the aortic suture line to heal, resulting in a false aneurysm that breaks into the adjacent small bowel.

In Table I will be found a brief summary of the cases reported in the literature and those reported in personal communication plus the case to be reported now.

CASE REPORT

On October 2, 1957 (Unit No. 66190), F. G. R., an eighty-year-old white man was referred to the Loma Linda Sanitarium and Hospital because of a three-week history of lower abdominal pain. The onset of the pain was following some yard work that required a lot of lifting and pulling. The pain was not severe and was intermittently present. Family history was noncontributory. The patient noted cramps in the calves of his legs on walking too far, particularly uphill or upstairs. Physical examination revealed a man who appeared somewhat younger than his stated age. The lungs were hyper-resonant to percussion. Palpation of the abdomen revealed a pulsatile mass compatible with an aneurysm of the abdominal aorta above the level of the bifurcation. Lateral abdominal films demonstrated calcification in such a manner as to indicate aortic enlargement. A diagnosis of abdominal aortic aneurysm was made. It was planned to do barium studies of the colon and stomach to rule out any associated disease. The first night of admission the patient was given castor oil in preparation for colon studies. About 1:30 a.m. the man vomited the castor oil and some blood clots. Pallor and sweating were noted and the systolic blood pressure dropped to 80 mm. Hg. Severe right lower quadrant pain appeared and then 150 ml. of clotted blood appeared in the vomitus. The serious situation was explained to the patient and his family. The added risk of surgery with absent popliteal, dorsalis pedis and posterior tibial pulsations and the history of calf cramps was considered.

At 5:45 a.m. on October 3, 1957, surgery was performed through a left paramedian incision. The findings were those of an aneurysm of the aorta at the bifurcation with attachment of the third portion of the duodenum over the aneurysm. The proximal aorta was mobilized as were the external iliac arteries and non-crushing clamps applied. The duodenum could now be freed from the aneurysm and the aortoduodenal fistula demonstrated. The ampulla of Vater was approximately two and

one-half inches proximal to this opening.

The aneurysm was excised and a knitted orlon bifurcation prosthesis inserted between the proximal aorta and both external iliac arteries. The eroded opening of the duodenum was closed with a two-layer closure, the outer layer of interrupted silk. A balloon type of catheter was placed in the stomach as a gastrostomy tube for gastric suction to avoid the use of a levine tube. At the conclusion of surgery his blood pressure was 160/85 and the femoral arterial pulsations were still present.

The postoperative course was good. On the third day there was some fever and disorientation but this subsided within twenty-four hours. He took fluids and sat up on the edge of the bed the third postoperative day. The diet was rapidly increased and the patient ambulated progressively. Progress was such that a tentative time for discharge from the hospital was set. The first hint of tragedy occurred at 2:30 a.m. of his ninth postoperative day. A large tarry and bright red stool was passed. The pulse was elevated to 148 and the blood pressure dropped to shock levels. The patient appeared pale and restless and complained of abdominal pain. Three pints of blood were pumped in under pressure to no avail and in the presence of his wife one hour and fifteen minutes after the first evidence of hemorrhage, he expired. My comment on the chart: "The duodenal wound must have become attached to the suture line or the aorta and digested through. We shall see at examination."

Post-mortem examination revealed no significant hemorrhage in the retroperitoneal space. At a point 0.5 cm. proximal to the upper suture line was a 0.5 cm. aortic fistula connected to the duodenum through a 1.5 cm. erosion. The suture lines were intact.

TABLE I

REPORTED CASES OF AORTO-ENTERIC FISTULAE FOLLOWING ABDOMINAL AORTIC SURGERY

Author and Date Reported	Age and Sex	Months Between Graft and Diagnosis	Type of Graft	Location of Fistulization	Results
HUMPHRIES and ASSOC. 1956	-----	15 days	Homograft	Proximal aorta into duodenum	Died
1956	-----	16 days	Homograft	Proximal aorta into duodenum	Died
1956	-----	4	Homograft	Proximal aorta into duodenum	Died
CLAYTOR and ASSOC. 1956	66 WM	16	Nylon	Right iliac suture line into distal ileum	Died
CREECH and ASSOC. 1957	-----	8	Orlon Taffeta	Proximal suture line to duodenum	Died
1957	-----	"In late follow-up period"	Nylon Fabric	Iliac anastomosis and ileum	Died
FITCH and DENMAN 1957	-----	6	Ivalon	Anterior graft into duodenum	Died
O'HARA and NAKANA 1958	53 WM	12	Amylon	Anterior graft into duodenum	Died

TABLE I, Contd.

Author and Date Reported	Age and Sex	Months Between Graft and Diagnosis	Type of Graft	Location of Fistulization	Results
HARDIN and VALK 1958	40 WM	6	Braided orlon	Proximal suture line into duodenum 3rd portion	Died
SCHARF and ACKER 1958	70 WM	15	By-pass homograft	Aorta into jejunum	Died
1958	59 WM	1 1/2	Nylon	Proximal suture line into duodenum	Died
MACKENZIE and ASSOC. 1958	70 WM	48	Homograft	Graft into duodenum	Survived
BROWN and ESSIG 1959	74 WM	14	Ivalon	Anterior graft into duodenum	Died
BOYD and PASTEL 1959	-----	2 1/2	Homograft	Proximal suture line into distal portion duodenum	Died
1959	-----	--	Homograft	Aorta into duodenum	Survived 3 weeks

TABLE I, Contd.

Author and Date Reported	Age and Sex	Months Between Graft and Diagnosis	Type of Graft	Location of Fistulization	Results
ELLIOT and ASSOC.					
1959	71 WM	33	Homograft	Proximal suture line into duodenum. Marked arteriosclerotic degeneration	Died
1959	75 WM	6	Homograft	Proximal suture line into duodenum	Died
CASE RECORDS MASSACHUSETTS GEN. HOSP.					
1959 No. 45282	68 WM	3	Teflon-Dacron	Proximal suture line into duodenum	Died
1959 No. 45522	62 WM	6	Teflon-Dacron	Midgraft into duodenum 3rd and 4th portion	Survived
SHERANIAN and ASSOC.					
1959	55 WM	16	Homograft	Anterior graft into suture line of duodenum	Died
1959	-----	18	Homograft	Graft into duodenum	Died
1959	-----	30	Homograft	Proximal suture line to duodenum	Died
DEWEESE and ASSOC.					
1959	74 WM	11	Homograft	Graft into distal jejunum	Died

TABLE I, Contd.

Author and Date Reported	Age and Sex	Months Between Graft and Diagnosis	Type of Graft	Location of Fistulization	Results
1959	54 WM	26	Homograft	Graft into proximal jejunum	Died
HAGLAND 1959	62 WM	60	Homograft	Graft into mid ileum	Died
JULIAN and ASSOC. 1959	-----	--	Homograft	Graft into duodenum	Died
1959	-----	--	Homograft	Proximal suture line into duodenum	Died
1959	-----	--	Homograft	Proximal suture line into duodenum	Died
1961	2 cases	--	Homograft & Prosthesis	Aorto-intestinal fistula	Died
LAWTON and ASSOC. 1959	64 WM	37	Homograft	Proximal suture line into terminal duodenum	Died
1959	62 WM	20	Homograft	Anterior graft into distal duodenum	Died
1959	65 WM	7	Homograft	Posterior proximal suture line into duodenum	Survived

TABLE I, Contd.

Author and Date Reported	Age and Sex	Months Between Graft and Diagnosis	Type of Graft	Location of Fistulization	Results
CORDELL					
1960	50 WM	9 days	Endarterectomy of aorta	Aortic suture line into duodenum	Died--23 days after endarterectomy
1960	48 WM	10	Dacron	Proximal suture line into duodenum	Died
CRAWFORD and ASSOC.					
1958	-----	6	Orlon Taffeta	Suture line into duodenum	Died
1960	-----	--	Dacron as aorto-femoral bypass	Proximal suture line into duodenum	Survived
1960	-----	--	Nylon as aorto-femoral bypass	Proximal suture line into duodenum	Died
1960	4 cases	2-30	Homograft	Proximal graft into duodenum	3 survived, 1 died.
MICHALS					
1961	80 WM	9 days	Orlon	Proximal aorta into duodenum	Died
POLLOCK and ASSOC.					
1961	58 WM	20	Homograft	Proximal suture line into duodenum	Died

TABLE I, Contd.

Author and Date Reported	Age and Sex	Months Between Graft and Diagnosis	Type of Graft	Location of Fistulization	Results
SZILAGYI					
1960	-----	--	Homograft	Aorta into duodenum	----
1960	-----	--	Homograft	Aorta into duodenum	----

DIAGNOSIS OF AORTO-ENTERIC FISTULAE FOLLOWING ABDOMINAL AORTIC SURGERY

Fistulization between the aorta and the intestinal tract must be ruled out when gastrointestinal hemorrhage occurs in an individual who has previously undergone abdominal aortic surgery. This may be difficult at times to do without the help of laparotomy.

Lawton[37] feels that any patient with a past history of abdominal aorta surgery must be carefully studied to rule out early erosion of a false aneurysm into the duodenum whenever epigastric pain or ulcer symptoms appear. One of his patients had ulcer symptoms for thirteen months before rupture. Another had occasional epigastric pain for a year prior to rupture. This pain was relieved by sodium bicarbonate. The third case had epigastric pain for two months before bleeding occurred.

Barium studies often are not helpful.[49] The films of the Massachusetts General Hospital cases[39, 40] were somewhat suggestive of duodenal pathology in the third portion.

Laparotomy must be carefully and thoughtfully performed to confirm these suspicions. One must state very frankly that on many occasions the laparotomy findings were reported to be negative for aorto-enteric fistula. Hardin and Valk[33] did a total gastrectomy and Roux esophagojejunostomy only to lose the patient a week later from an overlooked aortoduodenal fistula. Boyd[38] had to perform two laparotomies before an aortoduodenal fistula was finally demonstrated. An

aortojejunal fistula was not noted at laparotomy by DeWeese.[43] A Billroth II type of gastric resection was completed for an overlooked aorto-ileal fistula.[44] A similar procedure was accomplished in the presence of an aortoduodenal fistula.[37]

In one case the patient underwent gastrotomy as the first procedure. Partial gastrectomy was carried out at the second laparotomy and only on the third exploration was definitive surgery done for the fistula.[49]

Pollach[50] performed a Billroth I partial gastric resection and when bleeding persisted a total gastrectomy and esophagojejunostomy was attempted for an aortoduodenal fistula. This patient bled intermittently for three months before death occurred as a result of the fistula.

Even at laparotomy there may be difficulty in differentiating between an aorto-enteric fistula and some obscure cause of massive gastrointestinal hemorrhage. Julian[46] described the cause of hemorrhage in one of his patients to be due to an eroded large artery in the bed of an ulcer within a duodenal diverticulum.

Table II gives a summary of these operative experiences.

TABLE II

SURGICALLY TREATED CASES OF AORTO-ENTERIC FISTULAE FOLLOWING ABDOMINAL AORTIC SURGERY

Author and Date Reported	Age and Sex	Interval Between Onset and Surgery	Location of Fistulization	Procedure	Results
HARDIN and VALK 1958	40 WM	Several days	Proximal suture line into 3rd portion of the duodenum	Total gastrectomy and Roux esophago-jejunostomy	Died one week later
SHARF and ACKER 1958	70 WM	One day	Aortojejunal fistula	Repair of bowel Repair of homograft	Died eight days later of hemorrhage
MACKENZIE 1958	70 WM	-----	Homograft into 3rd portion of the duodenum	Resection of graft and fistula; nylon graft with bypass to external iliacs	Survived
BOYD 1959	------	-----	Aortoduodenal fistula	Negative laparotomy. Re-explored and fistula closed. Re-explored and patch graft applied	Died in 3 weeks of myocardial infarct

TABLE II, Contd.

Author and Date Reported	Age and Sex	Interval Between Onset and Surgery	Location of Fistulization	Procedure	Results
CASE REPORTS OF THE MASSACHUSETTS GENERAL HOSPITAL 1959 No. 45282	68 WM	1 week	Proximal suture line into the 4th portion of duodenum	Resection of portion of host aorta and a portion of Teflon-Dacron graft re-anastomosis	Died of uremia
No. 45522	62 WM	3 days	Teflon-Dacron graft to duodenum	Resection of redundant Teflon-Dacron graft. Closed duodenal opening. Flap of omentum over prosthesis	Survived
DEWEESE and ASSOC. 1959	74 WM	1 day	Homograft to distal jejunum	Exploratory laparotomy only	Died 6 days after surgery
HAGLAND and ASSOC. 1959	62 WM	-----	Homograft into mid ileum	Bilroth II gastric resection. Laparotomy and fistula closed. Re-explored and new homograft inserted	Died

TABLE II, Contd.

Author and Date Reported	Age and Sex	Interval Between Onset and Surgery	Location of Fistulization	Procedure	Results
LAWTON and ASSOC.					
1959	64 WM	15 days	Proximal suture line into terminal duodenum	Partial gastrectomy	Died 10th postop. day
1959	65 WM	Few hours	Proximal posterior suture line into duodenum	Duodenum repaired. 3 inches of homograft resected and replaced with new homograft	Survived
CORDELL and ASSOC.					
1960	50 WM	2 days	Aortic suture line into 3rd portion of the duodenum	Repair of aortic suture line defect	Died 14 days after repair
1960	48 WM	3-4 weeks	Proximal suture line into duodenum. Dacron patch over proximal suture line	Gastrotomy; re-explored for gastric resection; excision of portion of duodenum and a duodenojejunostomy performed at 3rd operation	Died

TABLE II, Contd.

Author and Date Reported	Age and Sex	Interval Between Onset and Surgery	Location of Fistulization	Procedure	Results
CRAWFORD and ASSOC.					
1960	3 cases	1-4 days	Homograft to duodenum	Suture closure of duodenum. Insertion of new homograft	Survived
1960	-----	-----	Proximal suture line into duodenum. Dacron bypass graft	Aorta suture ligated above and below and graft removed	Survived; amputation of one leg
1960	-----	-----	Proximal suture line into duodenum. Nylon bypass graft	Aorta suture ligated above and below and graft removed	Survived
POLLOCK and ASSOC.					
1961	58 WM	6 days	Proximal suture line into duodenum. Bled intermittently for 3 months.	Billroth I gastrectomy; esophagojejunostomy	Died

ETIOLOGY OF AORTO-ENTERIC FISTULAE FOLLOWING ABDOMINAL AORTIC SURGERY

Animal Experiments

At the start of this study it was hoped that by slightly modifying abdominal aortic surgery technic one could be able to consistently produce aorto-enteric fistulae in dogs. The second phase of the experiments would then be to find out how to prevent this fistulization.

This premise proved to be wrong for out of twelve animals only one such fistula was produced. When a dog is up and about gravity removes the pressure of the bowel from off the aortic anastomosis. This fact helps defeat an attempt to reproduce the findings that occur in man as a result of duodenal or jejunal pressure against the graft or suture line.

In four dogs a portion of the abdominal aorta was bared and freed of surrounding tissue. An incision was made in an appropriate portion of the duodenum. The bowel edges were traumatized much as one would find in a spontaneous or secondary aorto-enteric fistula. The bowel was then repaired with only a one-layer catgut closure. This suture line was applied to the bare aorta and there held in place with sutures. No fistulae formed. In human experience a somewhat analogous situation has produced fatal results. Wheelock[52] in elaborating on two reported fatal mycotic aneurysms of the iliac arteries[53] felt that a colon anastomosis should be placed just above or just below but not right over an iliac artery that had been denuded by a node dissection

for carcinoma. The same mechanism would be adequate to explain three reported cases of aortogastric fistulae.[54] These occurred in individuals who had a resection of an esophageal carcinoma with intestinal continuity restored by esophagogastric anastomosis at the aortic arch level.

The next group of four experimental animals had the abdominal aorta divided and reanastomosed with a continuous layer of fine silk. Again the duodenum was opened, the edges traumatized by pinching with a heavy clamp and a one-layer catgut closure accomplished. The bowel and vessel suture lines were held in contact one with the other by sutures. No aortoduodenal fistulae appeared.

The final group of four dogs sustained a resection of a portion of the abdominal aorta with implantation of a Teflon graft to replace the missing segment of vessel. Over one of these suture lines a one-layer suture repair of traumatized duodenum was approximated and held with suture. In one instance the graft became thrombosed. This did not limit the activity of the dog in any way. One dog died six months following surgery from massive rectal bleeding. At post-mortem examination there were found many nodes in the retroperitoneal region. A few drops of pus were noted grossly at the suture line and one could easily demonstrate an aortoduodenal fistula at the proximal suture line of the graft (Fig. 1 and 2).

An attempt was made to reproduce as nearly as possible some of the conditions that are found at the conclusion of aortic surgery in clinical practice. Antibiotics in minimal doses were given the animals for only three days postoperatively. One could have introduced factors not ordinarily found in clinical practice such as abnormal pressure on

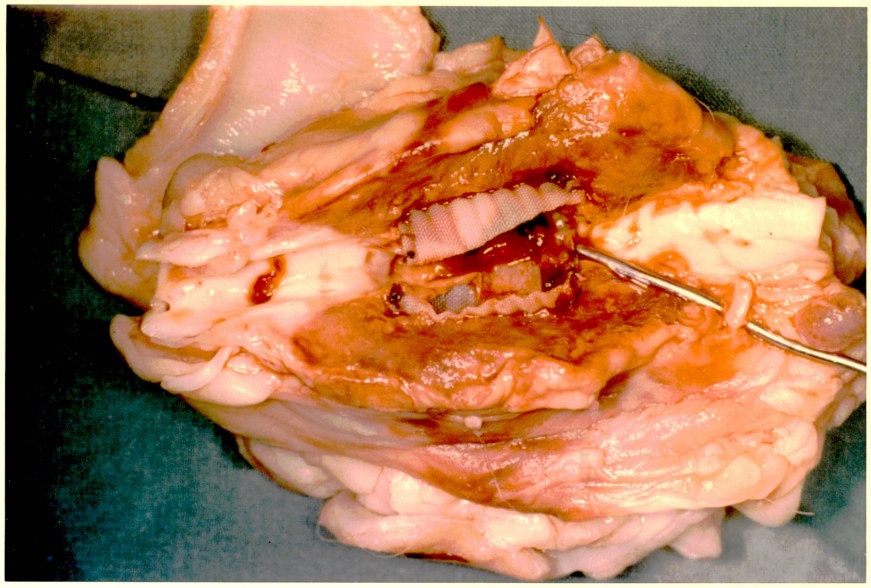

Fig. 1 Aortoduodenal fistula viewed from the aorta. This occurred at the proximal suture line of the aorta and Teflon graft.

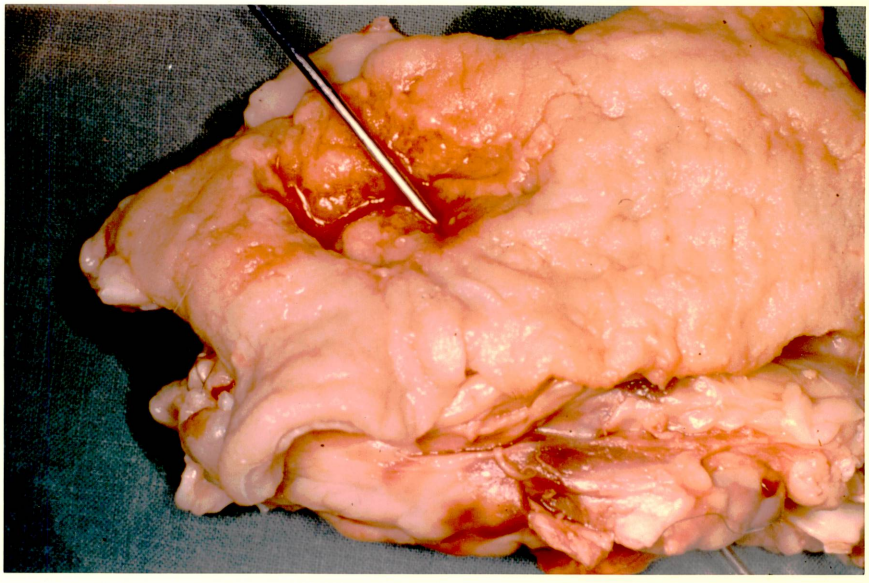

Fig. 2 Aortoduodenal fistula viewed from the duodenum. The probe is in the fistula tract.

the bowel suture line or the introduction of gross contamination and infection at the suture line.

Our attempt to consistently produce an aorto-enteric fistula was a failure. Therefore, further data as to cause and treatment of an aorto-enteric fistula must come from a detailed study of the reported human complications.

In Table III will be found a summary of the animal experimental findings.

Pulsatile Thrust of the Aorta

An experiment was reported by Griffen, Jr., and associates[55] to demonstrate the possible role of the pulsatile thrust of the aorta in the genesis of gastric ulcer. Retro-aortic placement of the fundus of the dog's stomach was followed frequently by the development of a gastric ulcer at the site of impingement of aortic thrust upon the gastric walls. Occasionally an aortogastric fistula formed. This mechanism might well operate in the aortogastric fistulae formed following esophageal resection.[54] An aortoduodenal fistula developed over a redundant and kinked Teflon-Dacron graft.[40] Such a redundancy would allow for more pulsatile thrust and thus bear out the experimental findings. Szilagyi[51] does not believe that the mere contact between bowel and blood vessel or vascular substitute has anything to do with these fistulae. He believes that inadvertent arterial wall injury may have taken place.

Infection

In my experimentally produced fistula there was gross evidence

TABLE III

SUMMARY OF ATTEMPTS TO PRODUCE AORTODUODENAL FISTULA IN DOGS

Dog No.	Date of Procedure	Procedure	Results
1	April 16, 1959	Duodenotomy with suture of wound to denuded aorta	Sacrificed Jan. 23, 1961 No fistula
2	May 29, 1959	"	Sacrificed Jan. 16, 1961 No fistula
3	June 5, 1959	"	Sacrificed Jan. 16, 1961 No fistula
4	February 24, 1960	"	Sacrificed Jan. 23, 1961 No fistula
5	March 2, 1960	Division and reanastomosis of abdominal aorta Duodenotomy sutured over the vessel wound	Sacrificed Jan. 23, 1961 No fistula
6	March 2, 1960	"	Sacrificed Jan. 3, 1961 No fistula
7	March 9, 1960	"	Sacrificed Jan. 9, 1961 No fistula
8	March 16, 1960	"	Sacrificed Jan. 9, 1961 No fistula
9	March 23, 1960	Aorta resected, Teflon graft inserted and duodenotomy sutured to proximal aortic anastomosis	Sacrificed Jan. 23, 1961 No fistula
10	March 23, 1960	"	Sacrificed Jan. 23, 1961 No fistula

TABLE III, Contd.

Dog No.	Date of Procedure	Procedure	Results
11	June 2, 1960	Aorta resected, Teflon graft inserted and duodenotomy sutured to proximal aortic anastomosis	Sacrificed Jan. 9, 1961 No fistula Graft thrombosed
12	June 9, 1960	"	Died of massive rectal hemorrhage Jan. 3, 1961 Aortoduodenal fistula

that infection was present and caused the fatal hemorrhage. Foster and associates[56] became interested in the problem of infection and grafting after three of their patients died of hemorrhage due to dissolution of the homograft in infected sites and contaminated wounds. In their experiments homografts and nylon grafts were implanted in the abdominal aorta. Infection was produced with canine feces suspension. The homografts broke down as did the host aorta at the suture lines of the nylon grafts. Antibiotic therapy proved helpful in preventing some of the infections.

A somewhat similar study was reported by Harrison[57] of forty-one dogs who sustained implantation of homografts and synthetic grafts in infected and contaminated areas. Fatal complications were higher in homograft implants because of rupture. This was felt to be due to the digestion of the homograft by proteolytic enzymes of the infection.

Good advice in regard to exposed extremity synthetic grafts is given by Creech in an editorial.[58] As long as three months have gone by without thrombosis or disruption occurring where local topical antibiotics such as neomycin or bacitracin are used daily in the infected area. Linton feels strongly that failure of graft function is due most frequently to infection with secondary hemorrhage.[59]

One would conclude that the avoidance of infection by careful technic and long-term antibiotic therapy would be in order. Ochsner[60] and associates figuratively raised eyebrows with a report of 931 patients operated upon for resection of aneurysm of the abdominal aorta. Six hundred forty of these (69 per cent) had associated abdominal and pelvic operations. These range between elective appendectomy, cholecystectomy, gastric resection, colon resection, to combined abdominal perineal

resection. No increase in mortality or morbidity was reported. Their advice was to tightly close off the retroperitoneal space as a means of preventing contamination of the graft that might occur as a result of the secondary procedure.

Rupture of the Host Aorta Proximal to the Suture Line

Proximal aortic rupture following resection of a coarctation of the thoracic aorta was reported by Sealy.[61] The twenty-two-year-old white male sustained an aortic perforation just proximal to the suture line on his seventeenth postoperative day. No cause was found for the necrosis of the aorta at this level.

DeBakey[62] described the death of a seventy-four-year-old white male on his thirteenth postoperative day due to proximal perforation of the aorta. This caused a fatal massive retroperitoneal hematoma. The proximal aortic clamp had been in place 105 minutes. The hemorrhage occurred from a small perforation of the aorta two centimeters proximal to the upper anastomosis. It was suggested that this was probably at the level of the occluding clamp which probably had fractured an atheromatous plaque.

Two cases of late rupture of the aorta proximal to the proximal suture line and precisely at the site at which the aorta had been cross-clamped led Schumacker[63] to design a new vessel clamp. He now uses a screw-down clamp which allows one to approximate the clamp blades just enough to shut off the flow of blood.

Further light on this problem comes from a recent anatomical demonstration of the vasa vasorum of the aorta. These vessels arise from the intercostals in the thorax and from the lumbar arteries in the

abdomen. They then partially circle the aorta and also travel in the long axis of the aorta. They do not cross over the posterior "mesentery" of the aorta but perforate the adventitia to enter the media. The absence of these vessels may well be the prime cause of homologous artery transplant failure.[64]

Injury to the wall of the aorta by cross-clamping is the most sensible explanation for the anterior aortic perforation proximal to the suture line that occurred in the case being reported in this paper.

Fate of Vessel Grafts

The natural fate of both homografts and synthetic grafts may help explain the formation of aorto-enteric fistulae. Butcher[65] modified an apparatus to measure quantitatively the response of segments of the human aorta to distention. The elastic properties of arteriosclerotic vessels are much the same as normal vessels unless the arteriosclerosis has progressed to advanced calcification. Implanted homografts had less distensibility. Loss of elasticity is greatest in grafts implanted the longest period of time.

In 1956 the Baylor group reported on the fate of aortic homografts fourteen, twenty-four, twenty-eight and thirty months after transplantation. Microscopic examination showed the homograft to retain much of its architectural pattern while it is being reinforced by fibrous connective tissue of the host. The conclusion was that while the ultimate fate of homografts is unknown, to date the aortic homografts can be utilized with confidence.[66] A more recent review by the same group[67] gave the gross and microscopic findings in homografts of human aortas in place twenty-four to eighty months and the changes in

synthetic grafts in place three to forty-three months. One patient developed an aneurysm of her homograft after thirteen months. This was resected. An aneurysm of the second homograft appeared fourteen months after surgery. This time most of the graft was excised and replaced with a Dacron prosthesis. Thirty-two months after the second homograft had been implanted, the remaining segment ruptured.

Of thirteen patients with homografts in place twenty-four to eighty months, three died as a result of failure of the graft. The common structural change is the gradual diminution of the elastic fibers with a progressive increase in the width of the hyalizing fibrous connective tissue encasing the graft. The longevity of the graft depends on its structural make-up at the time of transplantation and also on the condition of its new environment. No structural weaknesses were found in the Dacron prosthesis in place up to twenty-five months. One nylon prosthesis developed an aneurysm at the bifurcation after forty-three months.

Gross[68] reported on the fate of forty-two grafts of the thoracic aorta. Sixteen were in place three years. Nine of the grafts were functioning four years or more. One graft had been placed six years before. None of these displayed any aneurysms. Some faint calcification was noted in a portion of the grafts of three patients.

Szilagyi[69] reviewed his experience with homografts and found progressive loss of all tissue elements of the original transplant save for some elastic fibers. Section revealed a lack of true replacement of the graft by the host. Angiographic studies demonstrated changes in aorto-iliac homografts after two years. These degenerative changes increased in frequency and seriousness with the passage of time.

Further confirmation of this conclusion is made by Warren.[70] Four types of lesions were noted in his transplanted homografts--mural thrombosis, rapid degeneration with calcification, slowly progressive atheromatosis and aneurysm formation. All parts of the grafts did not undergo degenerative changes at the same rate.

Etiologic Factors Peculiar to Homografts

Disproportion in size between the homograft and the aorta has been mentioned as a cause of eventual fistulization.[34, 44] Synthetic grafts are available in a greater variety of sizes than can be found in the average homograft bank. The importance of this disproportion, which actually amounts to a physiologic stenosis, is nicely developed by Holman.[71] Experimental results and well-known clinical entities indicate that a dilatation tends to develop just beyond the stenosis. This is based on hydrodynamic principles and explains dilatation and even aneurysm formation in homografts beyond the proximal suture line.

Two authors have incriminated the vascular branches of the aorta as the eventual cause of the fistula. Hagland[44] believes aneurysm of the ligated aortic branches sometimes occurs. This erodes the bowel and then ruptures. DeWeese[43] noted the aorto-enteric fistulae occurred at areas compatible with the site of sutured arterial branches of the homograft.

Care must be taken during the preparation or reconstruction of homografts to prevent fracture of the media.[44] The reconstruction process of freeze-dried grafts takes a certain length of time and no short cut can be taken. Until the graft is fully reconstituted it is subject to injury if manipulated carelessly.

Gross[72] calls attention to the fact that homografts are apt to be a little softer and more pliable than the host vessel. Sutures therefore must not be tied or pulled too tightly for fear of cutting through the graft wall.

Suturing Technics

Poor tissue approximation is listed as one of the causes of aorto-enteric fistulae by Cordell.[49] Edwards and Lyons go into more detail on this point. There is a tendency to place sutures too superficially in a friable vessel. One must get a good substantial bite of adventitia even though the intima tends to protrude a bit. Careful deep sutures through all the vessel wall layers would prevent suture line leaks.[73] The posterior row of sutures on the host side of the anastomosis are the sutures that must be placed especially carefully.

Etiologic Factors Peculiar to Synthetic Grafts

In an attempt to prevent undue blood loss through the walls of the graft some surgeons coated the earlier synthetic grafts with plastic.[46, 74] This made the grafts rather stiff and more likely to produce erosion. Newer grafts of Dacron and Teflon are pliable and soft. They should not be a factor in erosion now.

Porosity of the synthetic grafts is important. The more porous the graft the more likely fibrous tissue will grow into the graft and help anchor the intima. Too much porosity leads to massive blood loss when the proximal clamp is released. The ideal porosity of a graft is just below the point of allowing excessive bleeding.[75] Knitted grafts allow for more immediate hemorrhage but also allow for better intimal healing.

Deterling,[74] Szilagyi,[51] and Kirklin[76] believe the use of a synthetic graft of Dacron or Teflon with careful placement of sutures will help prevent aorto-enteric fistula formation.

Results of Survey of Several Vascular Surgeons

Several prominent vascular surgeons were asked to outline their method of avoiding aortoduodenal fistula. Wheelock[52] takes great care to interpose some normal peritoneum, omentum or tissue of some kind between the cloth prosthesis and the duodenum. Creech[77] has not experienced this complication but carefully avoids it by moving the duodenum either up or down and to the right or left, as the case may be, in order to avoid the proximal anastomosis. DeBakey[78] suggests that a flap of omentum be interposed between the overlying duodenum and the aortic anastomosis. He believes it is important to properly place normal viable tissue around the anastomosis. Since using his own Dacron arterial substitute Szilagyi[51] has not observed any special precautions as to interposition of tissues. He stresses utmost care in placing sutures and in avoiding postoperative ooze. It is Deterling's[74] opinion that the danger of fistula formation following the use of soft commercially available grafts is very minimal. A peritoneal pedicle may be used for covering the upper anastomosis.

TREATMENT OF AORTO-ENTERIC FISTULAE FOLLOWING ABDOMINAL AORTA SURGERY

Control of Hemorrhage

The diagnosis or suspected diagnosis of an aorto-enteric fistula should soon be followed by surgical intervention. The first task is control of the bleeding point and/or compression of the proximal abdominal aorta. Crawford[48] temporarily controls circulation by placing a vascular clamp across the upper abdominal aorta at the hiatus of the diaphragm. After a segment of aorta between the renal arteries and the graft has been mobilized, a vascular clamp is applied at this level. Finger compression of the aorta against the vertebrae is a satisfactory but a tiring maneuver. Elliot[41] devised a wooden aortic compressor with sponge rubber pads. This allows an assistant to compress the aorta against the vertebral column. This method is less tiring and interferes less with the work of the surgeon. Thoracotomy for control of the aorta is unnecessary and undesirable in his opinion.

Another method of aortic control is the use of intra-aortic balloon catheter tamponade. Hughes[79] used a No. 10 French non-radiopaque Dotter Lukas balloon catheter 125 cm. long. This catheter was threaded into the aorta via a common femoral artery. After the balloon reached the ninth rib level in the midaxillary line it was inflated for a limited period of time. This stopped the flow of blood at the point of inflation. A simple method for stopping the blood loss was described by Lawton.[37] His patient vomited bright red blood and

went into shock while sitting in the hospital waiting room. In view of the patient's past operative history and his past complaints the diagnosis of aorto-enteric fistula was immediately made. While blood was rapidly infused the patient was carried to the operating room and under local anesthesia the abdomen was opened and the aorta compressed. Next the distal adherent duodenum was opened anteriorly and a finger placed in the fistula opening, thus controlling the bleeding. Appropriate clamps were then applied and the defective homograft excised.

Definitive Surgery

Whenever the fistulization involves a homograft or its suture line the homograft must be excised.[35] Gaspar[80] seriously advised the resection of the whole homograft when any part of the graft fails. This advice does not apply to Dacron or Teflon grafts. These can be partially excised and reanastomosed with good results.[39, 40] When a nylon graft fails it should be excised and replaced with Dacron or Teflon.[67] After placement of a new synthetic prosthesis the bowel opening is then resected and the bowel closed. The proximal suture line is covered with omentum or a peritoneal pedicle. The bowel anastomosis is kept away from the aortic suture line.

At times infection and abscess formation cause erosion of the vessel into the duodenum. When this occurs the grafts are resected but aortic continuity is not restored because of fear of recurrent infection. Instead the aorta is ligated above and the iliacs below the previous suture lines. This will lead to some distal ischemia. However, ischemia and a live patient are preferable to the almost certain fatal hemorrhage that will occur if a graft is left in place.[48]

SUMMARY AND CONCLUSIONS

Forty-six aorto-enteric fistulae following abdominal aortic surgery are recorded in this paper. One of these is a case report of an aortoduodenal fistula that was excised only to have the same condition recur with fatal results nine days later. Several other cases were gleaned from personal communications. These had not previously been recorded in the literature.

The most common site of fistulization of the bowel is the third and fourth portions of the duodenum. The jejunum and the ileum also have been involved. The anatomic fixation of the terminal portion of the duodenum must be considered as a preliminary factor. The most common site of fistulization of the vessel is the proximal suture line. The site of the vessel branches of the aortic homografts have occasionally been incriminated.

The most common cause of fistulization is the failure of the proximal vessel suture line. Proper placement of these sutures requires: proper tension to prevent leak, avoidance of too much tension that might cut through the host vessel or the homograft, placement of substantial bite sutures through all the layers of the host aorta to prevent later tearing out from the pulsatile thrust of the aorta.

Hematoma formation can be avoided by careful toilet of the retroperitoneal area before it is closed. Postoperative oozing should be avoided by preclotting the graft and by not using a too porous graft. Infection may be avoided by protecting the skin edges and not allowing the graft to touch the skin. Routine antibiotic therapy is in order.

Grafts should be inserted with some tension on the suture line so that redundancy will not occur when the graft is distended with blood.

Some of the etiologic factors hard to control are: injury to the host vessel by the occluding clamp and for advanced degeneration of the host vessel.

Diagnosis of aorto-enteric fistula requires a high index of suspicion of any upper abdominal digestive complaint in patients who have undergone abdominal aorta surgery. If gastrointestinal hemorrhage occurs in these patients an operative confirmation or denial of such a fistula must be done unless some definite source of bleeding can be demonstrated by X ray such as esophageal varices or a duodenal ulcer.

The treatment of these fistulae is operative and immediate. Control of the proximal aorta by clamping temporarily just below the hiatus or by manual compression of the aorta against the vertebrae at this level has been successful. Control of the aorta below the renals should be achieved as soon as possible.

Homografts and nylon grafts that have failed should be resected and Teflon or Dacron prosthesis implanted. Careful closure of the bowel opening with movement of the bowel away from the vessel is indicated. Omentum and peritoneum should be interposed between the proximal vessel anastomosis and the bowel.

Sixteen patients were subjected to surgery for control of bleeding resulting from aorto-enteric fistulization occurring after aortic surgery. Six of these had definitive surgery as their first surgical procedure. Three of the six survived the immediate operative period. Gastric resections, either partial or total, were done prior to recognition of the fistulization in four patients. The interval of

time from onset of gastrointestinal bleeding until time of surgery was reported to be from a few hours to as long as three weeks.

Prompt recognition and prompt attack of the fistulization will save lives that otherwise might be lost as a result of this serious complication. However, prevention is still better than treatment.

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COLLEGE OF MEDICAL EVANGELISTS

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AORTO-ENTERIC FISTULA FOLLOWING ABDOMINAL AORTA SURGERY

by

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ABSTRACT

Forty-six aorto-enteric fistulae following abdominal aorta surgery are here recorded. A number of these have never before been recorded in the literature. For the first time sixteen cases of these aorto-enteric fistulae that were subjected to surgical intervention have been tabulated. Twelve of these had definitive surgery either immediately or eventually. The definitive procedures are also tabulated.

An attempt was made to produce a fistula in dogs. Of twelve dogs, one died six months postoperatively of massive rectal hemorrhage due to an aortoduodenal fistula that followed the implantation of a Teflon vessel graft into the abdominal aorta. The cause of this fistula was infection and a hematoma.

The etiologic factors for the development of these fistulae are recorded from the literature and from personal communications. These factors are: use of homografts, use of nylon grafts, poor vessel suture technic, formation of a hematoma at the suture line, too great porosity of synthetic grafts, infection of the graft, injury of the host vessel, pulsatile thrust of the aorta, redundancy of the grafts, and direct contact of bowel with the vessel anastomosis.

Successful treatment of an aorto-enteric fistula following abdominal aortic surgery requires the following: prompt recognition of the presence of the fistula, control of hemorrhage, resection of the homograft or nylon graft, replacement of the graft with a Dacron or Teflon prosthesis, interposition of living tissue between the vessel

anastomosis and the bowel, and careful placement of sutures in the vessel anastomosis. If infection of the graft coexists the graft must be excised and the aorta closed by suture.