GASTRO-ESOPHAGEAL RESECTION AND TOTAL GASTRECTOMY IN THE TREATMENT OF BLEEDING VARICOSE VEINS IN BANTI'S SYNDROME*

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OF THE VARIOUS OPERATIVE procedures that have been used for esophagogastric hemorrhage in Banti's syndrome, splenectomy, porta-caval shunt and injection of the esophageal varices with sclerosing solutions are the only ones of much value.

Splenectomy^{1, 2} has given its best results in the uncommon cases with blockage of the splenic vein. When extrahepatic obstruction of the portal vein is present, splenectomy, by removal of a large area of the portal bed, may ameliorate the hemorrhage for a variable length of time, but it usually recurs as the hypertension is built up again. Porto-caval shunt^{3, 4, 5} introduced by Whipple and co-workers is the ideal treatment, since in addition to reducing the elevated pressure and tendency to hemorrhage of the gastro-esophageal varices, it relieves the passive congestion of all structures drained by the portal system. But in some cases of fibrous or cavernomatous transformation of the portal vein, it has been impossible to anastomose either the main trunk or one of its large tributaries with the inferior vena cava even when a vein graft was utilized. Also when anastomosis was possible, the communication has sometimes narrowed or closed. Previous splenectomy precludes splenorenal anastomosis. After successful anastomosis, there is still the possibility of hemorrhage from the already established gastro-esophageal varices.

In certain hands^{6, 7} the injection of esophageal varices with a sclerosing agent has frequently controlled esophageal hemorrhage for worth-while periods of time. Moersch⁸ of the Mayo Clinic has recently made a follow-up study of 22 cases for periods of three or more years after injection. Twelve have had no more bleeding, four are living with continued hemorrhages, three are dead of hemorrhages, and four are dead of unknown or unrelated causes.

An additional measure to be considered when these techniques either have failed or for emergent or other reasons cannot be applied, is resection of the bleeding segment. This is especially true since the recent mortality for both transthoracic esophago-gastric resection⁹ and total gastrectomy^{10, 11} for carcinoma in experienced hands is under 15 per cent and after either operation at least a fair state of health may be maintained for an indefinite period. The first reported case of one-stage, transthoracic esophago-gastric resection, performed by Adams and Phemister,¹² is alive and well nine years after operation and patients have lived for years after total gastrectomy with only mild anemia and slight impairment of nutrition.

Two patients with Banti's syndrome have been treated, one by total gastrectomy and one by transthoracic esophago-gastric resection. In each case

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the hemorrhages began at an early age and persisted after splenectomy, the liver was normal and the general health was little impaired except by the bouts of bleeding.

CASE REPORTS

Case 1.—C. C., male, age 18 years, was admitted to the hospital 7-7-44 with the chief complaint of hemorrhages from the gastro-intestinal tract beginning at the age of six. Since the age of four his general health had been below par and he had had periodic attacks of malaise, weakness and fever. At the age of five an enlarged spleen was noted and since the age of six he had had repeated bouts of hematemesis and melena which were often associated with febrile attacks. At the age of ten he entered the Children's Memorial Hospital, Chicago, where he was found to have a markedly enlarged spleen and blood examination revealed R.B.C. 395,000, Hb 70 per cent, platelets 6800, W.B.C. 3000, Diff. Polym. 63, L. 32, M. 4, E. I, Ret. 3 per cent, coag. t. 3 min., Bl.t. 4 min.

Splenectomy was performed. The spleen weighed 325 Gm. and microscopically showed the picture characteristic of Banti's disease. Following the splenectomy he continued to have bouts of hematemesis and melena and at the age of 16 they increased in severity. Eighteen months before admission, an esophagoscopic examination elsewhere revealed extensive varices of the lower and middle portions of the esophagus. Injections of the varices with a sclerosing solution were made on eleven occasions at that time, on nine occasions five months later, and on five occasions seven months after that. But two to four months after each set of injections, very severe bouts of bleeding occurred calling for numerous transfusions. The only change was that after starting the injections, the blood all passed by rectum, whereas before it has passed both by mouth and by rectum.

On admission, two months after the last hemorrhage, he felt well except for moderate weakness. Physical examination revealed essentially normal findings aside from an old left upper paramedian laparotomy scar. There were no paraumbilical varices or hemorrhoids. The liver was not palpable and the cephalin flocculation, colloidal gold and bromsulphalein liver function tests were normal. Blood examination showed R.B.C. 5,100,000, W.B.C. 8,900 and Hb 14 Gm. per cent. On esophagoscopic examination the lining of the middle and lower esophagus was thickened from the previous injections and few varices were seen. Roentgenologically, when the Valsalva test was applied, the esophagus showed some irregularity of the barium shadow and the proximal stomach showed filling defects which were interpreted as due to varices. The stomach was manipulated under the fluoroscope and the patient soon presented symptoms of hemorrhage which became severe. Bleeding continued irregularly for 20 days, and necessitated 24 blood transfusions.

The diagnosis was made of bleeding gastric varices and a total gastrectomy decided upon. Severe bleeding started again on the early morning of the day for which the operation was scheduled. Rather than risk the chances of another severe bout of bleeding, an emergency laparotomy was performed. The stomach contained a large amount of blood and the coronary vein and branches to the lesser curvature were enlarged. There was blood in both small and large intestines and no sign of enlargements of the branches of the superior mesenteric vein. The liver was of normal appearance. Total gastrectomy, esophago-jejunostomy and jejuno-jejunostomy were performed. Bleeding continued during the operation and 3500 cc. of partly clotted blood was found in the excised stomach. Thirty-one hundred cc. of blood was transfused during and shortly after operation. The **postoperative** course was relatively uneventful.

Pathology.—Externally the stomach looked normal aside from the moderately dilated veins on the lesser curvature. The gastric mucosa had normal folds and was of normal color except for a few petechiae. No ulcer or definite point of bleeding was found.

Microscopically, the outstanding feature in sections from all parts of the stomach

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was the large size of submucosal veins and mucosal venules. Especially large, empty, submucosal veins were found near the lesser curvature, close to the cardia (Fig. 1). Some of them had thin walls, with plaques which appeared to have been formed from organized mural thrombi. No fresh thrombi or open vessels were observed, here or

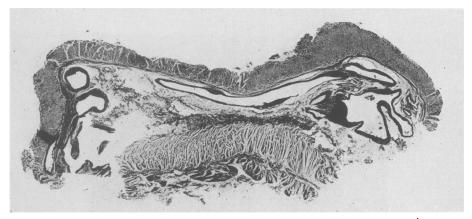


FIG. I.—Case I: Collapsed submucous gastric varices near the cardia. $(\times 9)$

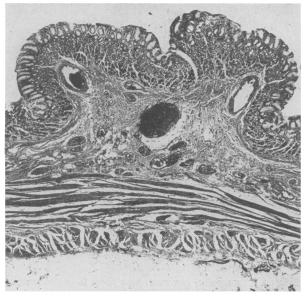


FIG. 2.—Case I: Dilated veins in the wall of the fundus. $(\times 27)$

elsewhere. The loose structure of the connective tissue adjoining the veins was probably an artifact. In the distended state the flat collapsed vein of Figure 1 must have caused the mucosa to bulge, and may have impinged on the dense fibrous tissue close to the muscular coats.

Many of the rugae contained numerous submucosal veins, sometimes as in the fundic

fold illustrated in Figure 2, stimulating an angioma. The two large veins in this fold protruded through the muscularis mucosae. Often connections could be traced between mucosal and submucosal veins. Such a channel in the mid-gastric region (Fig. 3) drained the basal venous plexus of the mucous membrane, and the numerous venules and capillaries between the gastric glands. It was sometimes possible to trace serially one of the large straight venules from a channel communicating with a large submucosal vein almost

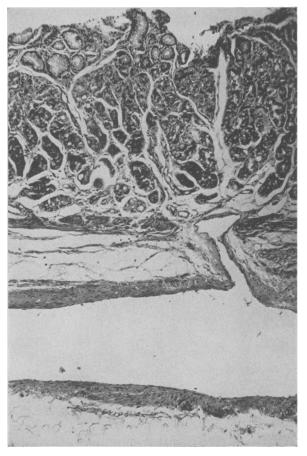


FIG. 3.—Case I: Mucosal venocapillary network communicating with large submucosal gastric vein. Mid-gastric region. $(\times 125)$

to the mucosal surface. At no point, however, was there a continuous demonstrable passage leading to the lumen. However, it is easily understood that bleeding might occur from minute surface erosions opening the tips of such vessels which would escape detection.

Even in the antrum and close to the pylorus venous distention and mucosal changes were evident, though nowhere so marked as near the cardia. Hemorrhage about a large pyloric vein as it passed through the muscle coats was probably the result of operative trauma. The largest external veins were close to the cardia. These coronary veins were not thrombosed, and were remarkable only because they were sclerotic, with walls thicker than those of the arteries they accompanied.

During the two and two-thirds years since operation, the patient has been in fairly good health aside from two bleeding spells, one moderate two years ago, and the other slight eight months ago. He has received injections of liver extract once a week and on 2-5-47, a blood examination revealed R.B.C. 4,320,000, W.B.C. 6.900 and Hb 13.6 Gm. per cent. Two years after operation, esophagoscopy revealed sclerosed bluish ridges in the lower esophagus but no definite varices were identified by needling and aspiration. This finding spoke against the reestablishment of important venous connections across the line of esophago-jejunal anastomosis. However, there was hematemesis at the time of the last bleeding, indicating that the blood came from esophageal varices.

Case 2.—E. K., a female age 21 years, was last admitted three and two-thirds months ago because of melena and other symptoms of hemorrhage of five hours' duration. She first entered the hospital at the age of nine years with a history of occasional bouts of hematemesis and melena since the age of four, some of which had been severe and associated with febrile reactions. Two days previously she became ill with a fever and on admission the temperature was 39°C. The only other positive finding on physical examination was a moderately enlarged palpable spleen. The initial blood examination revealed R.B.C. 4,960,000, Hb 96 per cent, W.B.C. 12,050. Ten hours after admission bleeding started with hematemesis, and marked signs of blood loss and melena followed. Six days later she was afebrile, the bleeding had stopped and the blood examination revealed R.B.C. 2,720,000, Hb 6.5 Gm. per cent, W.B.C. 4,700, coagulation time three minutes, bleeding time five minutes, platelets 260,000. Esophagoscopy revealed varices in the lower esophagus. A diagnosis was made of Banti's disease with bleeding esophageal varices.

Splenectomy was performed three weeks after admission when the patient had sufficiently recovered. The liver appeared to be essentially normal. The spleen weighed 167 Gm. and was normal in contour. Microscopically, there were the changes of congestive splenomegaly with early stages of Banti's fibroadenie. A hyaline thrombus was found in a small branch of the splenic vein near the hilus.

The postoperative course was uneventful. Blood examination six weeks after operation revealed R.B.C. 4,370,000, Hb 74 per cent, W.B.C. 9,150, platelets 828,000. Six months later the platelets were 215,000. At widely varying intervals, since the splenectomy the patient has had bleeding spells often associated with fever because of which she was repeatedly admitted for treatment. Otherwise she has been in good general health.

Five hours before the last admission, she developed weakness and faintness and soon passed a large amount of blood by rectum. Her condition grew worse and on admission she was in marked shock from hemorrhage. Blood examination revealed R.B.C. 2,110,000, Hb 9 Gm. and W.B.C. 15,600, Prothrombin 95 per cent. Unconsciousness set in shortly before transfusion was started but she responded well to the continuous administration of 500 cc. of plasma and 1500 cc. of blood, followed by 2500 cc. of blood in the course of the succeeding five days. The hemorrhage stopped and by the 8th day she was in good general condition. Roentgenography revealed varices in the lower esophagus (Fig. 4) which were verified by esophagogoscopy.

In view of the nearly fatal outcome, the patient readily consented to operation. By a left thoracic approach through the bed of the resected 8th rib, the lower esophagus was exposed. No enlargement was noted in the veins about it. The diaphragm was opened and the liver found to be normal in appearance. The veins of the stomach were not remarkable although it was not easy to judge those of the lesser curvature. Resection of the lower three and one-half inches of the esophagus and upper two inches of the stomach followed by esophago-gastrostomy was performed. The postoperative course was uneventful, there has since been no return of bleeding and she is now in good general health.

Pathology.—The mucosal surfaces of the resected segment are demonstrated in Figure 5. A few stringy brown clots adhered to the gastric mucosa which was grossly intact. Despite the previous oozing of much blood from the cut margins, tortuous submucosal

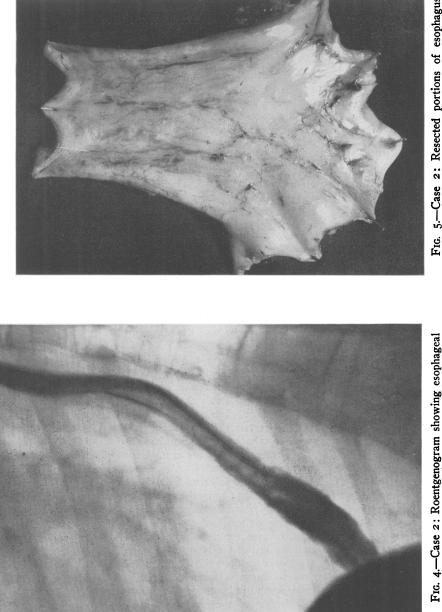


FIG. 5.—Case 2: Resected portions of esophagus and stomach; varices more extensive in esophagus than in stomach.



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veins were still conspicuous in both esophagus and stomach, but especially in the longitudinal folds of the esophagus. No gross defect in the esophageal epithelium could be demonstrated.

Microscopically, the largest esophageal veins were found to run in the submucosal tunic (Fig. 6). They impinged on and deformed the muscularis mucosae, and occasionally broke its continuity. Numerous collapsed smaller veins ran in the thickened mucosa, where occasionally one reached the proportions of the submucosal veins. As in the large



FIG. 6.—Case 2: Esophageal varices. $(\times 32)$

veins of Figure 6, the walls of the venous channels were of variable thickness. The fibrous tissue of the inner tunics was condensed, and was particularly compact close to the epithelium covering the longitudinal folds. The epithelium was thick, and on the tops of projecting folds hyperkeratotic. On the tops of projecting folds no epithelial defects were found.

Microscopically, the changes in the gastric wall were in general similar to but much less pronounced than those in Case I. The veins in the submucosa were considerably smaller than those in the esophagus but they were much more numerous than was apparent on gross inspection. The cross-section of a ruga close to the cardia, illustrated in Figure 7, demonstrates numerous wide veins. As in Figure 2 of Case I, large veins impinged on the mucous membrane, and lay within or above the muscularis mucosae. Figure 8 illustrates in detail one of these veins lying in the basal portion of the mucosal venous plexus. The thickest part of its wall represents an organized mural thrombus. Its large branch in the mucosa above is surrounded by dense fibrous tissue, representing an organized thrombotic vein. The artificially loose structure and distortion of the mucosa illustrated here was repeated in other sections which included large veins and wide venules. In some rugae the glandular pattern was even more distorted, resembling the changes illustrated in Figure 3 of Case I. No erosions nor points of recent bleeding were identified.

No grossly visible defects to account for the recent hemorrhages were found in either resected specimen. The conclusion based on clinical facts that the stomach was the important source of bleeding prior to the gastrectomy in the first case was supported by

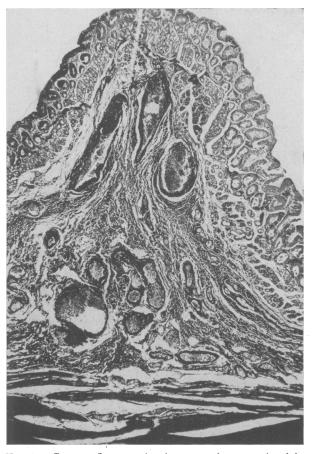


FIG. 7.—Case 2: Large veins in a gastric ruga. $(\times 60)$

histologic studies. Bleeding from the stomach as well as from the esophagus probably had occurred in the second case, judging from the presence of thrombosis in branches of the mucosa. Under some conditions, the gastric component of gastroesophageal varices may provide a greater danger of hemorrhage than the esophageal component.

DISCUSSION

The term "gastro-esophageal" fittingly characterizes the varices associated with chronic hypertension in the portal, gastric and splenic veins. Dilated gastric veins were demonstrated in both cases here reported. Especially in

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Case 1, where sclerosing injections had obliterated many of the esophageal varices, gastric veins were everywhere enlarged, particularly so near the cardia. In Case 2, with patent esophageal varices, venous dilatation was similar but less marked. Clinical and anatomical evidence of recent severe gastric hemorrhage was present in Case 1, without grossly visible defects in

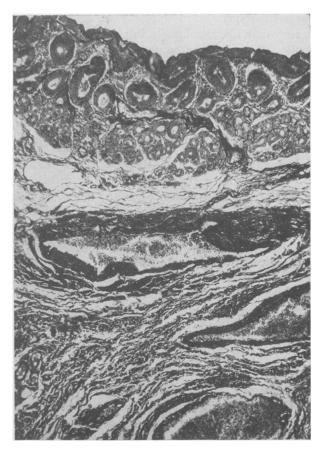


FIG. 8.—Case 2: Large vein lying internal to the gastric muscularis mucosae.

the gastric mucous membranes. The fact that bleeding may occur without gross erosions explains the frequent lack of emphasis on gastric hemorrhage associated with gastroesophageal varices.^{13, 14} The probable sources of the blood lost from the stomach without demonstrable lesion are numerous tiny ruptures through veno-capillary stomata, with the loss of too few epithelial cells to be appreciated grossly. In the average case of cirrhosis, or of Banti's syndrome, the esophageal varices are probably the source of most of the severe and sudden hemorrhages. However, these cases demonstrate how gastric bleeding may be added to esophageal bleeding, or as in Case I, may replace it in importance.

The time has not arrived for a final evaluation of all of the various operative procedures for the control of hemorrhage in Banti's syndrome due to extrahepatic portal block. Resection of the bleeding segment like obliteration of the esophageal varices by injection, throws more load on the remaining routes of collateral circulation; but this may be justifiable because they either do not bleed at all or bleed infrequently; and if patients with extrahepatic portal block and portal hypertension especially the splenectomized ones, do not bleed to death, they may live indefinitely in good health except at the time of the bleeding bouts. Transthoracic esophagogastric resection gets rid of that portion of the varicose segment which is the usual seat of major hemorrhage, divides the vessels of the lesser curvature and of a variable part of the greater curvature, thereby reducing gastric blood supply and establishes more or less of a permanent barrier in the esophagogastric venous collaterals at the line of anastomosis. Total gastrectomy gets rid of the bleeding points when the hemorrhage is predominantly from the stomach and establishes a connection between the esophagus and jejunum which is free of varices.

Theoretically and from the meager information furnished by these two cases, resection would appear to be worthy of a trial when control of hemorrhage is impossible by means of splenectomy, porta-caval shunt and injections. Whether in threatening and emergent cases such as these two, the percentage of failures with porta-caval shunt and injections may be great enough to warrant the use of resection in advance of one or both of those procedures, remains to be determined. Splenectomy might be combined either with transthoracic esophagogastric resection, or in case of gastric bleeding, with total gastrectomy, if on exploration it is found impossible to perform a porta-caval shunt. Since wider resections than were practiced here are possible, they might be called for in selected cases.

SUMMARY

A patient with Banti's syndrome and a normal liver had frequent severe hemorrhages from gastric varices after splenectomy and extensive obliteration of esophageal varices by injections had been carried out. Total gastrectomy was performed two years and eight months ago, since which time there have been only two bleeding spells from remnants of the esophageal varices, one moderate and the other slight.

A second patient with Banti's syndrome and a normal liver was subject to severe hemorrhages which continued after splenectomy. Transthoracic esophagogastric resection was performed and varices were found in the resected segment, greater in the esophagus than in the stomach. There was no further bleeding three and one-half months after operation.

The importance of gastric varices as a source of hemorrhage is discussed.

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DISCUSSION.—DR. ALFRED BLALOCK, Baltimore, Md.: Approximately two years ago I operated upon a 39-year-old woman who had bleeding esophageal varices with the intention of carrying out a procedure such as Doctor Phemister and Doctor Humphreys have described. On opening the left lower pleural cavity many varices could be seen on the outside of the esophagus. Two large plexuses of such vessels were removed (lantern slide showing dilated veins). We performed this operation rather than that of removal of the lower part of the esophagus because it appeared to be a less dangerous one. However, the bleeding continued and I wished subsequently that I had carried out the procedure described by Doctor Phemister.

Fortunately this patient had not had her spleen removed and in that respect she presented a problem somewhat different from that of the patients described by Doctor Phemister. Since the bleeding continued, we decided to perform a renal splenic vein anastomosis as developed and popularized by Dr. Arthur Blakemore and Dr. Allen Whipple.

(Motion picture showing renal splenic anastomosis.) The incision was performed through a left transverse abdominal incision. Other methods of approach have been recommended. For example, Doctor Linton of Boston uses a left transpleural approach. The spleen was used, exercising great care not to injure the splenic vein. The kidney was delivered into the incision. The renal artery was occluded temporarily. The renal vein was occluded proximally and distally with rubber shod clamps and a transverse opening was made into the vein between the points of occlusion. An end-to-side anastomosis was then performed between the proximal end of the splenic vein and the side of the left renal vein. The intima was approximated and everted by the use of ooooo silk on an atraumatic needle. By the use of the end-to-side anastomosis, it is not necessary to sacrifice the kidney. Furthermore, the experimental observations of Dr. T. N. P. Johns