Splenectomy in Schistosomiasis[1]

Preliminary Report

By J. NOYA BENÍTEZ

From the University Hospital of the School of Tropical Medicine, San Juan, Puerto Rico

THE cases of schistosomiasis that develop splenomegaly, secondary anemia, leukopenia, thrombocytopenia, and cirrhosis of the liver, with a tendency to severe gastrointestinal hemorrhage, constitute typical cases of Banti's syndrome. They develop portal hypertension due to intrahepatic portal block, for the ova of the parasite pass from the portal tributaries to the liver, thus producing cirrhosis. Even if the acute schistosomal infection is adequately treated, most of these cases gradually go on to the later stages of Banti's syndrome, with ascites, atrophy of the liver, subicteric sclerae, hemorrhage from the mucous membranes, and death from hemorrhage or liver insufficiency. Few recover. The splenomegaly never disappears, neither do the hepatic functions return to normal. Some also die from severe hematemesis before reaching the final stage.

Splenectomy offers a probable solution to the above-named portal hypertension and the resultant tendency to bleeding from the esophageal varices. In these cases, the enlarged spleens may contribute even more than 40 percent of the blood to the portal stream so that their removal will proportionately alleviate portal hypertension (though perhaps only temporarily in cases of severe intrahepatic block). Splenectomy will also shut off many of the large collateral veins in the gastrolienal ligament that feed the varices around the diaphragm, the cardia, and esophagus. From a limited experience, it is the writer's impression that the removal of the spleen accomplishes something more than mere mechanical relief, more or less permanent, as improvement of the anemia, leukopenia, and thrombocytopenia shows. The cases studied bear out the foregoing statement well. It is difficult to say how this is accomplished, yet the hematopoietic properties of the reticulo-endothelial system return to normal, or nearly so, following the removal of its most prominent member, which obviously becomes hyperplastic, and probably diseased, in cases of Banti's syndrome.

Splenectomy is more radical and more efficient than omentopexy and, of course, more radical than the injection or coagulation method

1. Received for publication May 14, 1947.

of sclerosing esophageal varices which, even if successful, would apparently only serve to increase portal hypertension. It is not as radical, or mechanically as efficient, as spleno-renal or portacaval anastomosis (Eck's fistula), but a review of the findings in large venous anastomosis has made the writer hesitate to carry it out, in preference to splenectomy, on the type of patients now being studied.

Blalock[2] has reported that two out of four patients with spleno-renal anastomosis died since operation from recurrent bleeding of the esophageal varices. Blakemore[3] (working with Whipple and Humphries) reported 23 portacaval anastomosis for the relief of portal hypertension. In 13 cases, the site of the obstruction was extrahepatic; in 10, intrahepatic. Splenectomy and left nephrectomy, with anastomosis of the splenic vein to the stump of the renal vein, was performed fifteen times. In one case, already splenectomized, the splenic vein was anastomosed to the vena cava, end to side. In the remaining seven, the portal vein was also anastomosed to the vena cava, end to side (Eck's fistula). The four post-operative deaths occurred in cases of cirrhosis, i.e., intrahepatic obstruction. Of the 19 surviving patients, eight had one or more episodes of bleeding; two of these eight are now dead.

Blakemore adds that indications for portacaval shunt are clear-cut in cases of portal hypertension due to extrahepatic portal bedlock, with essentially normal livers. Of one thing he is certain—that unless the operation is carried out with infinite care as to detail—and these are many—the chances for the portacaval shunt remaining patent are practically nil.

REVIEW OF THE LITERATURE

In 1930, a large spleen weighing 1800 g. was removed from a patient with schistosomal Banti's syndrome.[4] The patient was followed up for eight years during which time he did exceedingly well (outside of developing filariasis, elephantiasis of one leg, and having an Auchincloss operation performed on him in 1931). However, he suffered no further hematemesis. By 1938, he had become a regular tramp, with no systematic way of life, yet showed no symptoms referable to the liver, portal circulation, or spleen.

2. A. Blalock, Personal communication. A. O. Whipple, The problem of portal hypertension in relation to the hepatosplenopathies. Ann.Surg., 122:449–475, 1945.

3. A. Blakemore, Indications for portacaval anastomosis. Analysis of cases. Surg., Gynec., & Obs., 84:645–653, 1947.

4 By Dr. Jorge del Toro, Attending Physician, University Hospital, San Juan, Puerto Rico.

In 1931, another successful splenectomy on a schistosomal case of Banti's syndrome was again performed, but there was no follow-up.

In 1934 and 1935, six splenectomies for schistosomal splenomegalies were attempted at the University Hospital and five at the Presbyterian Hospital.[5] Three cases died at the first institution and two, at the last named place (a mortality of 45 percent, approximately). Three other splenectomies—two for purpura and one for hemolytic jaundice—were also performed at the University Hospital without mortality.

During the next few years, the writer tried one such operation at the Presbyterian Hospital and one at the University Hospital. Both patients died. Similar pessimistic reports came from other surgeons on the Island, until there finally arose a strong feeling against splenectomies for schistosomal splenomegaly.

However, as the years went by, reports from the Spleen Clinic of the New York-Presbyterian Hospital confirmed the role of portal hypertension on the symptoms of portal cirrhosis with splenomegaly. In addition, treatment of cirrhosis of the liver with high protein, high carbohydrate, low fat, and a high vitamin diet progressed so much that a more helpful prognosis could be held for these patients. Meanwhile, the writer continued to see, time and again, patients suffering from Banti's syndrome with severe hematemesis from which most of them eventually died, arrive at the University Hospital. When by 1945, the Blood Bank was ready to supply ample amounts of blood, he decided to try once more the effect of splenectomy on a few selected cases. That year he performed a successful transthoracic resection of a case with Banti's syndrome at the Presbyterian Hospital, in San Juan. Late in 1945, he operated on another case at the University Hospital, this time through the abdominal route. The patients in question were better prepared with ample blood for transfusions, fibrin foam for local areas of bleeding, and later, oxycel gauze for the same purpose.

The writer afterwards communicated with Dr. A. O. Whipple, at the New York-Presbyterian Hospital, asking whether he should continue the series in view of the latter's work on spleno-renal and portacaval anastomosis. In a personal communication, the writer was encouraged to continue the series of splenectomies, as Dr. Whipple believed that the results of splenectomy in schistosomal splenomegalies might be better than in cases of portal hypertension due to other causes of intrahepatic shock.

5. By Dr. A. O. Whipple and Dr. L. M. Rousselot, of the College of Physicians and Surgeons, Columbia University, New York City

In 1946 Elliott,[6] also of the Spleen Clinic, reported the following results in a series that was divided as follows:

	Splenectomies	*Deaths*
A. Extrahepatic	18	10
B. Intrahepatic: 1. Cirrhosis	37	25
2. Cirrhosis (schistosomal)	14	2
C. Undetermined	27	6
	(3 from other causes)	

The results of splenectomy for extrahepatic block (various causes of portal vein obstruction) show about ten deaths in 18 cases, a prohibitive mortality. The cases of intrahepatic block from cirrhosis show 25 deaths out of 37 cases, an even higher mortality. In contrast, those cases of schistosomal cirrhosis show only two deaths out of 14 cases (which obviously do not include the Puerto Rican series). The deaths from undetermined causes show six (of which three are from other causes) out of 27—perhaps the best results obtained. In this last group, the liver was essentially normal, and it is to this fact that Elliott ascribes the reason for the satisfactory outcome.

Why operate on other cases if the results were so poor? White,[7] of the Spleen Clinic, found that the life span in operated cases was anywhere from three to six years after the development of cirrhosis was evident; in the unoperated cases, the length of life after such symptoms developed was only thirteen and a half months. In conclusion, Elliott emphasized the inadvisibility of delaying surgery in those groups where it was indicated, once a definite diagnosis had been reached. He believed that good results in the schistosomal series were due "to the fact that patients with this particular condition show no clinical or laboratory evidence of hepatic insufficiency." The writer's data, and those from numerous other cases of schistosomal cirrhosis studied at the School of Tropical Medicine and elsewhere, reveal, however, that these cases do show clinical and laboratory evidence of hepatic insufficiency and that many of them go on to die of liver failure. The author agrees with Elliott's unstated premise that the less the liver damage, the better the outcome and the better the prognosis.

Notwithstanding, there were other series from various clinics throughout the world of which a few will be mentioned here without discussing the findings or conclusions.

6. R. H. E. Elliott, Disorders of the spleen with special reference to those amenable to surgical therapy. Bull. N. Y. Acad. Med., 22:415–427, 1946.

7. M. White, cited by R. H. E. Elliott, *ibid.*

Pemberton,[8] from the Mayo Clinics, reported in 1931 a series of 167 cases with splenic anemia, or Banti's syndrome, for which splenectomy had been performed at that institution. His figures showed that 33 percent of fatalities following operation were due to hemorrhage and that 98 patients, who had complained of gastrointestinal hemorrhage before operation, suffered one to two post operations. Pemberton proposed the ligation of the coronary vein in order to reduce the enormous turgescence of the esophageal varices and so interrupt communication with the vena porta. He also performed an omentopexy for the purpose of creating an additional collateral circulation.

In Howell's[9] report of a series of 94 cases of splenic anemia and Banti's syndrome, 51 were treated by splenectomy and 43 medically. After following up the cases from one to 23 years, Howell's observations suggested that splenectomy does not arrest the progress of liver disease, nor does it abolish the risk of death from hematemesis whatever the stage of the illness when treatment is instituted. He summarized: "Our analysis of the results of treatment in 94 cases of splenic anemia, or Banti's syndrome, shows that splenectomy does not improve the expectation of life, nor prevent the progress of the cirrhosis of the liver or of the anemia or the occurrence of hematemesis and there is, therefore, no logical reason for returning to it as a routine."

Andrus and Holman,[10] in 1939, studied a series of 50 patients who had been subjected to splenectomy at the New York Hospital during the five years preceding their report. They showed eight cases of Banti's disease with no operative deaths. One patient died two years after operation of a progressive cirrhosis with ascites. In a follow-up of three and a half years (the case longest studied), two additional patients suffered hemorrhage recurrences.

In 1940 Barg and Dulin[11] reported a study, carried out from 1922 on, on 43 patients with Banti's syndrome. Twenty-two of them were splenectomized, and the remainder were treated conservatively. There were six operative deaths among the 22 operated cases; three of them occurred in patients over 60 years of age. In three of the

8. J. Pemberton, cited by A. Zeno, F. Ruiz, and H. Martínez, Fatal hematemesis occurring three years and eight months after a splenectomy performed in the treatment of Banti's disease. Bol. y Trab. de la Soc. de Cir., Buenos Aires, 17:333, 1933.

9. L. Howell, Treatment of splenic anemia and Banti's syndrome. Lancet, 1:1320–1324, 1938.

10. W. DeW. Andrus and C. W. Holman, Splenectomy in various blood disorders. Ann. Surg., 109:64–83, 1939.

11. E. H. Barg and J. W. Dulin, Splenectomy in the treatment of Banti's syndrome. Arch. Surg., 41:91–95, 1940.

six, the disease was in the non-ascitic stage. Three of the deaths were the result of hemorrhage from the operative site. One patient died of massive thrombosis of the superior and inferior mesenteric veins on the eighth post operative day; another died of multiple pulmonary infarcts on the tenth day after operation. The sixth died of cardiac failure; this patient was 66 years old. Two additional patients died in the hospital—one due to hemorrhage from the deep epigastric vessels after a paracentesis, and the other two months post operation of a massive hemorrhage from a ruptured esophageal vein. One living had an esophageal hemorrhage eight years after operation, yet he had never bled pre-operatively. Of the 21 patients followed up medically, ten died within one year after discharge from the hospital.

The above named authors concluded "that splenectomy is the treatment of choice and should be performed in the early stages. The procedure may be contraindicated for elderly patients because of the high operative mortality. However, three patients who were over 60 years of age have greatly improved after operation. Splenectomy has not assured relief of gastro-intestinal hemorrhages. In general, patients with rapidly developing symptoms before operation respond poorly to splenectomy."

Otuka[12] reported on 43 splenectomized patients suffering from Banti's syndrome. The mean surgical mortality went up to 20.9 percent. In the first stage the mortality rose to 50 percent; in the second stage it was 0 percent, but in the third stage, it shot up to 53.2 percent. These cases were followed up for thirteen years. The author emphasized the fact that, even in advanced cases, splenectomy may give good results, but he advised as early splenectomy as possible, as "the sole operative method of choice." Three cases of splenectomy in the third stage had their anemia and ascites regress after operation, but the patients remained well for a long time.

REPORT OF CASES

The writer has performed 20 splenectomies during the past year and a half: one in 1945, 14 in 1946, and five to date during the present year. The series was made up of 13 males and seven females, of which 13 were white and seven colored. The youngest patient was 15 years old, the eldest, 41; there were six between 16 and 20 years of age, 11 between 20 and 30, and three above 30. All came from established foci of schistosomal infestation in Puerto Rico.

12. T. Otuka, Resultate der Splenektomie bei der sogenannten Bantischen Krankheit. Zentralbl.f.Chir., 68:307–309, 1941.

The size of the spleen ranged from 500 g. (the smallest) to 2,220 g. (the largest). Ten gave no history of hematemesis, while the remaining ten had had one or several hematemesis; 15 had a history of melena. Six had a 2 plus Hanger-cholesterol-cephalin-flocculation test; four had a 3 plus test, while ten had 4 plus tests showing liver involvement. Eleven showed retention of bromsulphalein in the blood thirty minutes after the intravenous injection.

Protein studies were made on 18; 15 showed abnormalities in the albumin-globulin ratio, four showing reversal of said ratio. These last four gave the highest B.S.P. retention; a liver biopsy showed moderate to mild cirrhotic changes in them. The patients had been given antimony treatment for bilharziasis so that their feces were negative for ova of *S. mansoni* at the time of operation, even though they had been positive at one time or another before. One patient had also had a positive rectal biopsy. Four x-ray shadows were positive for esophageal varices; four were suggestive, two possible, while ten were negative.

All were hospitalized for rather long periods of time, ranging from 141 days for the first case in 1945 to 20 days for the thirteenth case. All patients received at least 500 cc. of blood during or after the operation; an occasional case received more if it was deemed necessary. Postoperatively, all patients had a rise in platelets, but none over 500,000 per cmm. In all, however, the red and white blood counts went up to nearly normal, or above. The longest follow-up period was one of twenty months and the shortest, four weeks. All except two patients gained weight ranging from four to 23 pounds, and none lost any.

The first case developed a bronchopneumonia and apparently a perinephritic abscess that absorbed without drainage, hence his prolonged hospitalization. Sixteen months later he developed a cervical adenitis; biopsy proved it to be tuberculous (April 8, 1947), so he was referred for x-ray therapy.

The fourth developed a respiratory paralysis during the anesthesia, followed by a neurogenic colon and bladder. This patient recovered from all the foregoing conditions, but it was necessary to perform a cystotomy for the neurogenic bladder. He had developed a rather severe cystitis (*B. proteus*), which finally responded to streptomycin therapy. He also developed a massive atelectasis of the left lung from which he recovered after bronchoscopic aspirations.

Case No. 6, on whom was attempted a manual emptying of the spleen after division and ligation of all vessels, except the large splenic vein, developed a cerebral malaria manifestation (*P. fal-*

ciparum) on the tenth day post operation. From then on, the practice of trying to autotransfuse the patient with his splenic blood (either by adrenalin or pituitrin injections, or manually) was discontinued, as malaria probably lurks in most of these enlarged spleens, and it was necessary to avoid complications as much as possible.

Cases Nos. 4, 8, 9, and 15 had moderate postoperative atelectasis, from which they recovered with conservative treatment.

Case No. 7 developed fever, abdominal pain, leucocytosis, and thrombocytosis fourteen days after operation. A diagnosis of thrombosis of the splenic vein was made and the patient placed on dicumerol therapy. He recovered.

Case No. 11 developed a generalized lymphadenopathy, apparently a serum reaction that subsided spontaneously.

Case No. 19 had a moderate hematemesis, with melena, during the second postoperative week when the red blood count went down to 2,000,000. The patient was transfused, given iron, and discharged on the sixteenth postoperative day with a red blood count of 3,400,000 and a hemoglobin of 72 percent.

Case No. 20 was operated on May 5, 1947.

The third case complained of a slight hematemesis during his visit to the follow-up clinic three months post operation, but has felt ill no more. Both his red and white blood counts and his hemoglobin have stayed up despite a hookworm infection for which he received treatment.

The seventh case had to be readmitted to hospital because of a moderate hematemesis five months after operation. After admission, in February, 1947, he had a dark stool but no further bleeding *per os*. RBC were 3,700,000; WBC, 3,400; hemoglobin was 85 percent. Under very conservative treatment, rest in bed, a bland diet, the patient did very well and is now up and about again. His esophageal varices were injected twice during February and March, 1947. He has not bled since and is in New York City at present enjoying good health.

Biopsies of the liver were performed on all cases except the first, second, and the fourth. All showed cirrhosis: three slight, one mild, six moderate, one advanced, and four pronounced. Two showed pseudotubercles in the specimen in addition to some cirrhosis.

SUMMARY AND CONCLUSIONS

Twenty splenectomies, performed on patients suffering from the pre-ascitic stage of Banti's syndrome due to schistosomiasis, have

Splenomegaly Due To Schistosoma Mansoni *Treated by Splenectomy*

	Operative Date[a]	*Pathology*	*Liver Biopsy*	*Complications*	*Hospital Days*	*Post-operative Platelets*	*WBC*	*Proteins*	*Weight Gain (lbs.)*	*Hematemesis*	*Melena*
1	9/12/45 S	Splenomegaly due to *S. mansoni*		Bronchopneumonia	141			5.26 3.4/1.8	12	0	0
2	3/29/46 S & A	Splenomegaly and splenic infarct		None	39		8,850	6.06 3.9/2.16	20	0	0
3	5/1/46 S & B	Splenomegaly and pulp fibrosis (schist.)	Advanced periportal fibrosis	None	33	175,000	9,200	6.80 4.2/2.6	10	+	+
4	5/15/46 S	Splenomegaly due to *S. mansoni*		Bladder paralysis Massive atelectasis	70	300,000	6,300		4	0	0
5	6/5/46 S & B	Splenomegaly	Slight fibrosis	Bronchopneumonia	29	360,000	9,920		10	0	0
6	6/21/46 S & B	Splenomegaly and pulp fibrosis (schist.)	Pseudotubercles	Malaria	27	367,000	7,840		22	0	0
7	7/12/46 S & B	Splenomegaly	Cirrhotic	Fever (7)	72	345,000	6,400	6.8 4.6/2.2	5	+	+
8	7/29/46 S & B	Splenomegaly due to *S. mansoni*	Marked cirrhosis	Atelectasis	33	385,000	8,280		14	0	0
9	8/30/46 S & B	Splenomegaly and pulp fibrosis (schist.)	Pseudotubercles	Atelectasis	35	302,000	10,400		4	0	0
10	10/14/46 S & B	Splenomegaly	Mild cirrhosis	None	28	190,000	4,760		16	0	0
11	10/21/46 S & B	Splenomegaly	Moderate cirrhosis	Adenopathy	25	178,000	10,400		15	0	0
12	11/15/46 S & B	Splenomegaly due to *S. mansoni*	Moderate cirrhosis	None	23	280,000	12,220		20	0	0
13	12/27/46 S & B	Splenomegaly	Pseudotubercles	None	20	250,000	9,700	5.74 3.75/2.19	4	0	0
14	11/21/46 S & B	Splenomegaly	Normal liver	None	34	315,000	6,700	3.84/2.72	0	0	0
15	3/15/47 S & B	Splenomegaly due to *S. mansoni*	Marked cirrhosis	Atelectasis	24	175,000	7,640		0	0	0
16	3/28/47 S & B	Splenomegaly and hematopoiesis	Slight cirrhosis	None	51	390,000	12,000		0	0	0
17	3/28/47 S & B	Splenomegaly due to *S. mansoni*	Moderate cirrhosis	None	31	435,000	14,000		0	0	0
18	4/9/47 S & B	Splenomegaly due to *S. mansoni*	Moderate cirrhosis	None	71	320,000	13,000		0	0	0
19	4/11/47 S & B	Splenomegaly due to *S. mansoni*	Moderate cirrhosis	Melena hematemesis	42	220,000	13,750		0	+	+
20	5/5/47 S & B	Splenomegaly due to *S. mansoni*	Moderate cirrhosis	None	41	230,000	20,300		4	0	0

[a]S = Splenomegaly
A = Appendectomy
B = Biopsy

The size of the spleen ranged from 500 g. (the smallest) to 2,220 g. (the largest). Ten gave no history of hematemesis, while the remaining ten had had one or several hematemesis; 15 had a history of melena. Six had a 2 plus Hanger-cholesterol-cephalin-flocculation test; four had a 3 plus test, while ten had 4 plus tests showing liver involvement. Eleven showed retention of bromsulphalein in the blood thirty minutes after the intravenous injection.

Protein studies were made on 18; 15 showed abnormalities in the albumin-globulin ratio, four showing reversal of said ratio. These last four gave the highest B.S.P. retention; a liver biopsy showed moderate to mild cirrhotic changes in them. The patients had been given antimony treatment for bilharziasis so that their feces were negative for ova of *S. mansoni* at the time of operation, even though they had been positive at one time or another before. One patient had also had a positive rectal biopsy. Four x-ray shadows were positive for esophageal varices; four were suggestive, two possible, while ten were negative.

All were hospitalized for rather long periods of time, ranging from 141 days for the first case in 1945 to 20 days for the thirteenth case. All patients received at least 500 cc. of blood during or after the operation; an occasional case received more if it was deemed necessary. Postoperatively, all patients had a rise in platelets, but none over 500,000 per cmm. In all, however, the red and white blood counts went up to nearly normal, or above. The longest follow-up period was one of twenty months and the shortest, four weeks. All except two patients gained weight ranging from four to 23 pounds, and none lost any.

The first case developed a bronchopneumonia and apparently a perinephritic abscess that absorbed without drainage, hence his prolonged hospitalization. Sixteen months later he developed a cervical adenitis; biopsy proved it to be tuberculous (April 8, 1947), so he was referred for x-ray therapy.

The fourth developed a respiratory paralysis during the anesthesia, followed by a neurogenic colon and bladder. This patient recovered from all the foregoing conditions, but it was necessary to perform a cystotomy for the neurogenic bladder. He had developed a rather severe cystitis (*B. proteus*), which finally responded to streptomycin therapy. He also developed a massive atelectasis of the left lung from which he recovered after bronchoscopic aspirations.

Case No. 6, on whom was attempted a manual emptying of the spleen after division and ligation of all vessels, except the large splenic vein, developed a cerebral malaria manifestation (*P. fal-*

ciparum) on the tenth day post operation. From then on, the practice of trying to autotransfuse the patient with his splenic blood (either by adrenalin or pituitrin injections, or manually) was discontinued, as malaria probably lurks in most of these enlarged spleens, and it was necessary to avoid complications as much as possible.

Cases Nos. 4, 8, 9, and 15 had moderate postoperative atelectasis, from which they recovered with conservative treatment.

Case No. 7 developed fever, abdominal pain, leucocytosis, and thrombocytosis fourteen days after operation. A diagnosis of thrombosis of the splenic vein was made and the patient placed on dicumerol therapy. He recovered.

Case No. 11 developed a generalized lymphadenopathy, apparently a serum reaction that subsided spontaneously.

Case No. 19 had a moderate hematemesis, with melena, during the second postoperative week when the red blood count went down to 2,000,000. The patient was transfused, given iron, and discharged on the sixteenth postoperative day with a red blood count of 3,400,000 and a hemoglobin of 72 percent.

Case No. 20 was operated on May 5, 1947.

The third case complained of a slight hematemesis during his visit to the follow-up clinic three months post operation, but has felt ill no more. Both his red and white blood counts and his hemoglobin have stayed up despite a hookworm infection for which he received treatment.

The seventh case had to be readmitted to hospital because of a moderate hematemesis five months after operation. After admission, in February, 1947, he had a dark stool but no further bleeding *per os*. RBC were 3,700,000; WBC, 3,400; hemoglobin was 85 percent. Under very conservative treatment, rest in bed, a bland diet, the patient did very well and is now up and about again. His esophageal varices were injected twice during February and March, 1947. He has not bled since and is in New York City at present enjoying good health.

Biopsies of the liver were performed on all cases except the first, second, and the fourth. All showed cirrhosis: three slight, one mild, six moderate, one advanced, and four pronounced. Two showed pseudotubercles in the specimen in addition to some cirrhosis.

SUMMARY AND CONCLUSIONS

Twenty splenectomies, performed on patients suffering from the pre-ascitic stage of Banti's syndrome due to schistosomiasis, have

Splenomegaly Due To Schistosoma Mansoni *Treated by Splenectomy*

	U.H. No.	*Age*	*Sex*	*Race*	*Hematemesis*	*Melena*	*Splenomegaly*	*RBC*	*WBC*	*Proteins*	*Platelets*	*Feces*	*Hanger*	*BSP*	*X-Ray*
1	A–5829	22	M	W	4+	4+	4+	2.98	1,500	10.40 4.37/6.03	162,000	0	4	8%	Suggestive
2	A–6681	20	F	W	Multiple	Multiple	1050	2.87	3,480	6.34 3.74/2.60		0	4	0	Negative
3	A–7983	23	M	C	2+	4+	4+	4.420	4,280		57,500	0	2	0	Negative
4	A–2047	26	M	W	0	Several	1450	4.210	4,800		217,500	0	4	0	Suggestive
5	A–3222	17	M	C	0	Several	4+	3.070	5,000	5.04 2.79/2.25	165,000	0	4	2%	Positive
6	A–7864	21	M	W	0	Positive	1100	3.740	2,640	6.29 4.18/2.11	95,000	0	2	0	Suggestive
7	A–7865	32	M	W	2+	Many	500	5.06	4,040	6.76 4.42/2.34	112,500	0	4	4%	Positive
8	A–8350	25	F	W	0	Many	980	3.660	5,760	6.00 3.86/3.14	250,000	0	2	12%	Positive
9	A–7935	17	F	C	+	Many	1270	4.34	7,360	4.27 2.76/1.51	100,000	0	4	4%	Suggestive
10	A–5713	16	M	W	3+	Many	2220	4.32	2,400	6.33 3.61/2.72	115,000	0	4	2%	Negative
11	A–9234	36	F	W	0	Occasional	700	3.87	6,840	7.48 3.18/4.30	80,000	+	4	2%	Negative
12	A–8816	21	M	W	0	0	570	4.060	3,760	5.79 3.61/2.18	167,500	0	4	2%	Negative
13	A–7703	16	F	W	0	0	1900	4.2	3,280	6.09 3.79/2.30	210,000	+	3+	0	Positive
14	A–9202	28	M	W	0	0	710	4.49	2,000	6.10 3.13/2.97	125,000	0	2	0	Negative
15	A–5230	16	M	C	0	0	1870	4.04	6,840	6.11 4.15/1.96	110,000	+	3	0	Negative
16	A–9901	26	F	C	+	+	1500	3.65	3,400	6.29 2.91/3.38	55,000	0	2–3	0	Negative
17	A–4056	21	F	W	3+	+	1740	3.87	3,350	6.15 3.48/2.67	165,000	0	4	16%	Positive
18	A–9703	15	M	W	0	0	1250	4.101 3.28	2,500	6.28 4.00/2.28	80,000	0	3+	0	Negative
19	A–10199	21	M	C	2+	Many	880	3.52	4,340	7.43 3.67/3.76	75,000	0	2+	16%	Negative
20	A–9726	41	M	C	4+	4+	1370	3.440	2,200	6.02 3.96/2.06	55,000	0	3+	16%	Positive

been presented. A short review of the literature with the findings of other clinics has also been given.

There has been no mortality in the series presented here.

Three cases (third, seventh, and nineteenth) had hematemesis once, three months, five months, and two weeks post operation, respectively. All of them recovered successfully under conservative treatment, except for the seventh case which was given two injections for esophageal varices. All cases showed improvement in general health and well-being, their blood counts, hemoglobin, and platelets having gone up to nearly normal limits and their liver function tests having shown considerable improvement.

The results so far show that splenectomy is strongly indicated and is the treatment of choice in cases of Banti's syndrome due to schistosomiasis. Notwithstanding, a more extended follow-up (for at least five years but preferably, ten, or as long as the cases can be followed up) and the study of a greater number of cases is necessary to definitely establish the value of splenectomy in cases of Banti's syndrome produced by schistosomiasis.