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THE LATE PHASE OF CONGESTIVE SPLENOMEGALY (BANTI'S SYNDROME) WITH HEMATEMESIS BUT WITHOUT CIRRHOSIS OF THE LIVER

Further Observations on the Etiology of Banti's Syndrome and the Effect on Prognosis of Certain Variations in the Portal Venous Pattern

Louis M. Rousselot, A.B., M.D., Med.Sc.D., New York, N. Y.

(From the Departments of Surgery, Presbyterian Hospital and Babies Hospital, and the Spleen Clinic of the Vanderbilt Clinic)

THE early descriptions of Banti[1, 2] and his concept of the etiology and clinical course of so-called Banti's disease are so entrenched in medical literature and text that it becomes difficult to alter that which so long has received the sanction of common usage. The following quotation from Cecil[3] epitomizes the generally accepted textbook version of the disease: "Splenic anemia (Banti's disease) is a chronic disease of unknown origin, probably toxic, and primary in the spleen, and is characterized by splenomegaly, anemia, and leukopenia, a tendency to gastric hemorrhage, increased formation and destruction of blood cells, and later by cirrhotic changes in the liver, with ascites and jaundice."

The clinical sequence as described by Banti of (1) primary splenomegaly, toxic in origin, (2) an intermediate stage, and (3) finally, the third stage with cirrhosis of the liver, of course, has often been disputed. The German concept of *Stauungsmilz*[4] was an early attempt to controvert Banti's theory. This in general precludes the possibility of a toxic substance producing primary splenic enlargement and predicates the existence of chronic portal stasis as a precursor of the splenomegaly. It is surprising to find how little convincing evidence has been presented to prove either of the two aforementioned contentions.

The Spleen Clinic of the Presbyterian Hospital has presented a series of papers[5-7] in recent years supporting the view that the picture presented by Banti is confusing and misleading in respect to etiology, clinical course, and treatment. Our group is firmly convinced that the etiology of Banti's syndrome may be entirely explained on a mechanical basis; i.e., portal bed obstruction with an associated "portal hypertension."[8] The site of the obstructive factor causing the portal hypertension and splenomegaly may be intra- or extrahepatic. The term "congestive splenomegaly" suggested by Larrabee,[9] we believe, is more descriptive than Banti's disease or Banti's syndrome.

The usual intrahepatic lesion producing congestive splenomegaly is, of course, cirrhosis of the liver. This paper, however, is solely concerned with a selected group of 15 patients, all presenting the typical symptoms, signs, and blood studies usually associated with this syndrome. Severe gastrointestinal bleeding occurred in each of these patients, yet in no case was there coexistent liver cirrhosis. We shall demonstrate that in most of these patients an extrahepatic obstructive factor was present in the portal venous bed. In every instance in which splenic vein pressures were taken at operation, a "portal hypertension" could be determined. The nonexistence of liver cirrhosis in all was established by most of the available methods at our command. These procedures include various liver function tests carried out before operation, gross examination of the liver at operation, and liver biopsy on one or more occasions in the same patient. Furthermore, evidence will be advanced to show that cirrhosis not only was absent at the time of operation, but did not develop as established by the subsequent clinical course and further laboratory studies for varying periods up to nineteen years after operation. In four instances this lack of liver disease has had additional confirmation by autopsy as well.

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Evidence Demonstrating the Absence of Liver Cirrhosis in This Group of Cases

Table I will clarify the statements as to the nonexistence of liver cirrhosis in all these cases. In the first 2 patients operated upon in 1920 and 1922, there is the least evidence. In each instance there is only the surgeon's operative description of a normal liver. However, in each of the succeeding 13 patients, almost all of whom were operated upon by Allen O. Whipple or myself, at least two forms of corroborative evidence are presented. Histologic proof of normal liver tissue was available in 10 of the 15 cases. Liver biopsy was done at operation in 7 instances and 1 of these also had subsequent autopsy confirmation. Autopsy was also obtained in 3 additional patients who had no liver biopsy at the time of operation.

It is our experience that the bromsulphthalein test is a reliable index of hepatic damage. We have felt particularly secure in our interpretation of these tests because in every case no retention or only a slight trace of the dye was present in the blood after thirty minutes and no equivocal or border line determinations occurred. Bauman and Orr's[10] recent report on the accuracy of the bromsulphthalein test is of interest In not one instance in our series was there any inconsistency between liver function test, liver biopsy, or necropsy findings.

Nature of the Extrahepatic Lesions

As designated in Table I an extrahepatic obstructive factor was demonstrable in the portal venous system of 8 of the 15 patients examined (Cases 4, 5, 6, 7, 9, 11, 12, and 14). This was recognized at the operating table in 4 instances and 4 times it was only demonstrable at autopsy. The failure to discover an obstructive factor in the other 7 cases is not

TABLE I

EVIDENCE OF NONEXISTENCE OF LIVER CIRRHOSIS

CASE NO.	LIVER FUNCTION STUDIES BEFORE OPERATION	OPERATIONS*	OPERATIVE GROSS APPEARANCE OF LIVER OBSTRUCTIVE FACTORS	FINDINGS			
				MICROSCOPIC DIAGNOSIS OF LIVER BIOPSY	VENOUS PRESSURE	LIVER FUNCTION STUDIES AFTER OPERATION	AUTOPSY FINDINGS LIVER OBSTRUCTIVE FACTOR
1	----------	S	Normal Undetermined	No biopsy	----------	----------	----------
2	----------	E	Normal Undetermined	No biopsy	----------	----------	----------
3	----------	S	Normal Undetermined	No biopsy	----------	13 yr. postoperatively bromsulphthalein test normal Ser. prot. 7.2% Ser. alb. 4.3 Ser. glob. 2.9	----------
4	----------	S	Normal Undetermined	No biopsy	----------	12 yr. postoperatively bromsulphthalein test normal Ser. prot. 6.4% Ser. alb. 4.0 Ser. glob. 2.4	Normal liver Stenosis of portal vein just below liver
5	----------	S	Normal Thrombosis of splenic vein	No biopsy	----------	----------	----------
6	----------	S	Normal Undetermined	No biopsy	----------	----------	Normal liver Cavernomatous transformation of portal vein
7	Bromsulphthalein test normal Ser. prot. 5.5% Ser. alb. 3.6 Ser. glob. 1.9	S	Normal Undetermined	Biopsy: normal liver	----------	----------	Normal liver Cavernomatous transformation of splenic and portal veins
8	Bromsulphthalein test normal Ser. prot. 5.9% Ser. alb. 4.2 Ser. glob. 1.7	S	Normal Undetermined	Biopsy: normal liver	Splenic V.—370 mm. Arm V.—50 mm.	----------	----------
9	----------	S	Normal Thrombosis of splenic vein	Biopsy: normal liver	----------	2 yr. postoperatively bromsulphthalein test normal Ser. prot. 8.3% Ser. alb. 4.9 Ser. glob. 3.4	----------
10	----------	S L	Normal Undetermined	Biopsy: normal liver	----------	3 yr. postoperatively bromsulphthalein test normal Ser. prot. 6.2% Ser. alb. 4.2 Ser. glob. 2.0	----------
11	Bromsulphthalein test normal Ser. prot. 7.1% Ser. alb. 4.2 Ser. glob. 2.9	S	Normal Thrombosis of junction of splenic and portal veins	Biopsy: normal liver	Splenic V.—400 mm. Arm V.—170 mm.	----------	----------
12	----------	S	Not noted Undetermined	No biopsy	----------	----------	1 right lobe, no cirrhosis, focal necrosis present; left lobe represented by small nubbin of tissue Stenosis of lower end of portal, above splenic vein
13	Bromsulphthalein test normal Ser. prot. 7.7% Ser. alb. 4.4 Ser. glob. 3.3	S	Normal Undetermined	Biopsy: normal liver	Splenic V. 330 mm. Arm V. 55 mm.	----------	----------
14	----------	L	Not noted Thrombosis of splenic vein	No biopsy	----------	6 yr. postoperatively galactose tolerance test normal Ser. prot. 8.03% Ser. alb. 5.09 Ser. glob. 2.94	----------
15	Ser. prot. 6.9% Ser. alb. 4.3 Ser. glob. 2.6	S	Normal Undetermined	Biopsy: normal liver	Splenic V. 465 mm. Arm V. 110 mm.	----------	----------

*S, Splenectomy; L, ligation of splenic vessels; E, exploratory celiotomy.

necessarily a weakness in our hypothesis but is due rather to the technical difficulties involved in an operative examination of the portal venous bed away from splenic hilum, particularly behind the head of the pancreas.

In this connection it is of interest to note that one patient (Case 4), although reported in two previous papers in 1936 and again in 1939 as having an "obstructive factor" undetermined, has recently died, and a post-mortem examination was made. This patient had had a splenectomy at 5 years of age and was subsequently followed for thirteen years. During this period he continued to have recurring hematemeses and melena and was admitted to the Presbyterian Hospital twenty-four times. Bleeding was always severe and occurred approximately twenty-one times via the esophageal route and ten times per rectum. Because of (1) the normal appearing liver at operation, (2) the normal blood count between bleeding episodes, (3) normal liver function tests at the ninth, eleventh, and twelfth years after splenectomy, we felt secure in our belief that cirrhosis never did exist and that it subsequently had not developed. This patient finally died only seven months ago. Before section was started, one of our staff, William P. Thompson, predicted the finding of a normal liver and also an obstructive lesion in the portal vein close to liver. Both predictions were confirmed.

The nature of the extrahepatic lesions responsible for obstruction to splenic blood in this series are listed as follows:

1. Thrombosis of splenic vein (traumatic)	Case 5
2. Thrombosis of splenic at junction with portal vein (traumatic)	Case 11
3. Thrombosis of splenic vein (nontraumatic)	Case 9
4. Thrombosis of splenic vein (nontraumatic)	Case 14
5. Stenosis of portal vein (upper end) just below liver	Case 4
6. Stenosis of portal vein (lower end) above entrance of splenic vein	Case 12
7. Cavernomatous transformation of portal vein	Case 6
8. Cavernomatous transformation of junction of portal vein and splenic veins	Case 7

The extrahepatic obstructive lesions just detailed occurred in the respective sites indicated on Fig. 1.

THE ANATOMICAL PORTAL VENOUS PATTERN AS A DETERMINANT OF SYMPTOMATOLOGY AND PROGNOSIS

In an analysis of the long-term results following splenectomy in this entire group, one rather interesting observation has become apparent. From an examination of Fig. 2 it will be seen that 9 of these patients continued to have intestinal bleeding following operation for periods varying from eight months to thirteen years; whereas, 6 were immediately relieved of this serious complication and have remained so for long periods of time, in Case 1 for as long as nineteen years. Why should such a marked variation in clinical behavior occur? This phase

of the problem has been given a good deal of thought and it is our belief that we may have the answer. It is our purpose to demonstrate that (1) the site of the obstruction and (2) variations in the anatomical distribution of the veins forming the portal system may be the determining factors governing symptomatology, course, and eventual prognosis in any individual case. Referring again to Fig. 1, it seems obvious that a lesion occluding the portal vein close to the liver will impede the flow of a much greater volume of blood than a similar stenotic process located in the splenic vein close to the hilum of the spleen.

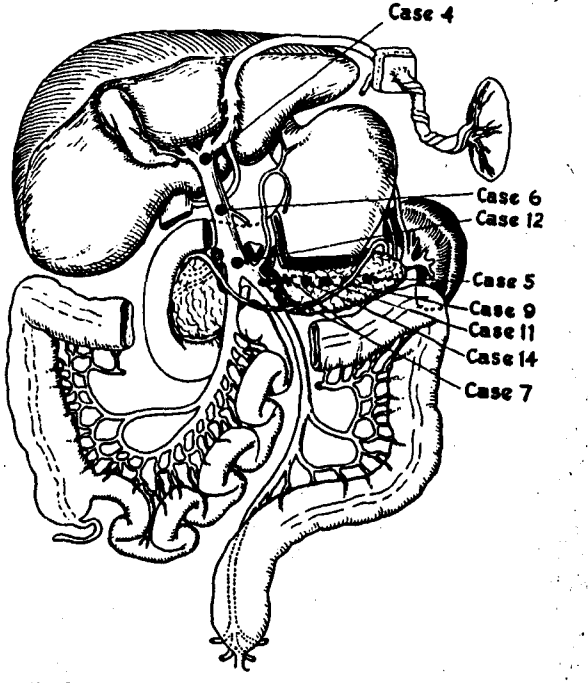

Fig. 1.—Schematic drawing (after F. Paitre, H. Lacaze, S. Dupret), indicating the sites of the obstructive factor in the cases designated.

The former involves the entire return flow of the portal system; whereas, the latter affects only the splenic fraction, or approximately 20 per cent. This question of the site of the obstruction is further qualified, we believe, by the second consideration just alluded to. This second consideration is the variations that occur in the anatomy of the veins forming the portal system.

Fig. 3 portrays six different anatomical patterns taken from six standard sources. Even cursory inspection of these figures will show that an obstructive lesion as indicated in *B* will produce much more profound alterations in intestinal physiology than the identical lesion

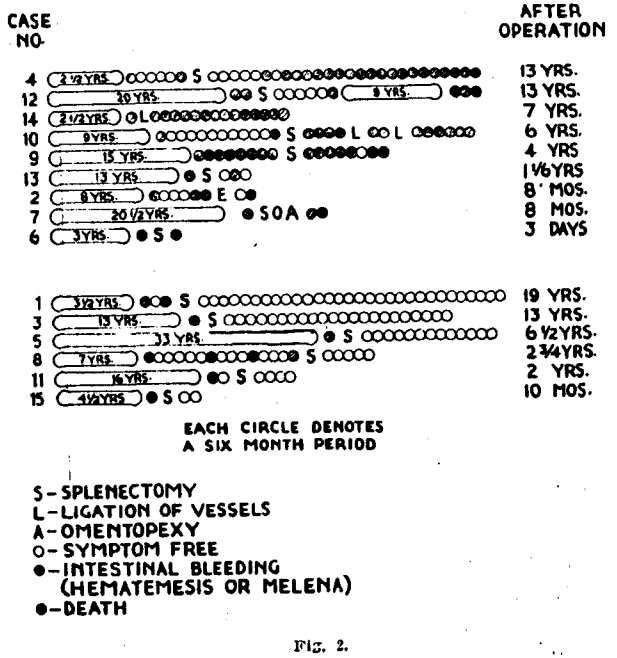

Fig. 2.

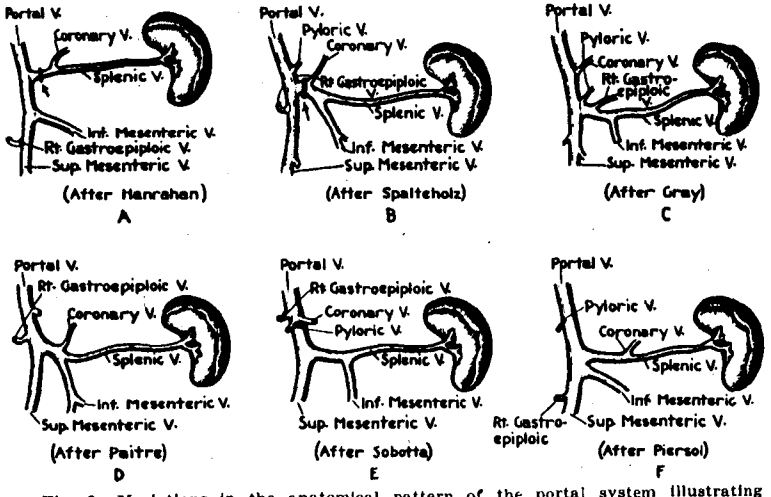

Fig. 3.—Variations in the anatomical pattern of the portal system illustrating how an obstructive factor of the same site in different individuals may result in marked differences in clinical behavior.

occurring in an individual possessed of a portal system as illustrated in *A*. Additional observations along these lines will explain many of the vagaries and alterations in clinical behavior in these patients. To quote specific examples from our series:

Case 4 showed a stenotic lesion of portal vein immediately subjacent to liver, and Case 12 lower down in the portal vein. Regardless of the venous pattern in such a situation the entire portal flow is impeded. Splenectomy, although returning the blood picture to normal, will only remove a small burden of the already overloaded collateral circuit. Intestinal bleeding in this type of case, of course, will continue and will not only evolve from esophageal varices but also from small and large bowel. The latter is due to blockage of the superior and inferior mesenteric venous outflow. These are the cases in which melena will always be a dominant feature.

In Cases 5 and 11, however, the obstructive factor was a thrombosis of the splenic vein. This involved only the splenic contribution to the portal flow. Establishment of a collateral circulation via the vasa brevia resulted in bleeding esophageal varices. Following splenectomy, this recurring thrust on the varices was removed. These patients had subsequently no further bleeding. Between these two extremes all gradations in clinical behavior are possible and are dependent, as mentioned before, on (1) the site of the obstruction and (2) the disposition of the accessory veins supplying the portal vein.

SUMMARY

A group of fifteen cases is presented, all with the characteristic features of Banti's syndrome and all with gastrointestinal bleeding as a dominant symptom. Although exhibiting the presumably late symptom of hematemesis, none of these cases had cirrhosis of the liver at operation or subsequently for long periods of time up to nineteen years after operation. The etiology of the splenomegaly can be entirely explained on a mechanical basis with a primary obstructive factor in the portal bed and an associated portal hypertension. The evidence presented therefore contradicts the sequence described by Banti of a primary splenomegaly and a subsequent cirrhosis due to a hypothetical toxic agent.

A variety of extrahepatic lesions have been listed, all capable of producing venous stasis. In the four instances in which splenic vein pressures were determined a definite venous hypertension was recorded. This confirms similar observations previously reported by this clinic in other forms of Banti's syndrome.

The frequency of anomalies in the portal system is stressed.

In this group of cases of congestive splenomegaly due to extrahepatic obstructive lesions, the prognosis and variations in clinical behavior

are dependent on two factors: (1) the site of the obstructive lesion and (2) variants in the anatomy of the venous pattern. To the best of our knowledge, this latter concept is herein presented for the first time.

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THE PROPHYLACTIC USE OF SULFANILAMIDE IN ABDOMINAL SURGERY

JOHN S. LOCKWOOD, M.D., AND I. S. RAVDIN, M.D.,
PHILADELPHIA, PA.

(From the Surgical Clinic of the Hospital of the University of Pennsylvania and the Laboratory of Surgical Bacteriology of the Harrison Department of Surgical Research, Schools of Medicine, University of Pennsylvania)

IN VIEW of the dramatically successful results of chemotherapy in such diverse infections as hemolytic streptococcic peritonitis, cellulitis, and septicemia, colon bacillus pyuria, gonococcic urethritis, and pneumococcic pneumonia, the question has logically arisen as to the possible effectiveness of sulfanilamide or one of its derivatives in peritonitis of intestinal origin. The literature on the surgery of the colon has been concerned to a large extent with measures designed to minimize the threat of peritonitis as a complication of such operations. That peritonitis continues to occupy the major position among the causes of death following surgical procedures on the large intestine is shown in Table I. The operative mortality of radical procedures averages about 18 per cent and about 40 per cent of the deaths are due to peritonitis. The data of Rankin[7] show that, while the mortality following simple colostomy may be very low, almost 60 per cent of the deaths that do occur are attributable to peritonitis. If sulfanilamide can be safely employed to reduce the incidence of postoperative peritonitis, a major advance will have been achieved.

In a previous publication,[9] dealing with the use of sulfanilamide in hemolytic streptococcic infections, expression was given to the opinion that the effectiveness of sulfanilamide in any lesion is conditioned more by the pathologic character of the infectious process than by the degree of susceptibility of the infecting organisms. As a result of a three-year experience in the use of sulfanilamide in the prevention and treatment of peritonitis on our surgical service at the Hospital of the University of Pennsylvania, we are inclined to reaffirm this opinion. In this paper we will undertake to supply a rational basis for sulfanilamide therapy in polymicrobic peritonitis and to present a summary of our experience to date.

BACTERIOLOGIC CONSIDERATIONS IN PERITONITIS OF INTESTINAL ORIGIN

Peritonitis of intestinal origin is usually a mixed bacterial infection in which the organisms most frequently found are the *Bacillus coli*, the green or nonhemolytic streptococci, and the Welch bacillus. The careful studies of Meleney, Harvey, and Zaytseff-Jern[10] on the bacterial flora of peritonitis, in which sound bacteriologic methods were used, showed

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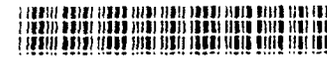

Call Number: 1964
Journal/Book Title: AM J MED

Volume: 51 Issue: ISSN/ISBN:
DATE: 1971
PAGES: 160-
Article Title: NONCIRRHOTIC PORTAL FIBROSIS

Author: SAMA, SK; BHARGAVA, S ET AL

Page Count 3
Reason Not Filled

Runner Note: CAON

124

Client Order Num.: 5393
Client Sequence 1: 5393
Client Sequence 2: BAN

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BFG36694

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