The liver suffers most severely and the damage is present throughout life. With the early lesions of necrosis life is short, and in its acutest form the syndrome constitutes erythroblastosis foetalis. The subsequent development of fibrosis may terminate in a few cases in the clinical features of cirrhosis of the liver.

The spleen suffers less and changes are slower to develop. Milder cases live to middle or later decades and the spleen enlarges comparatively harmlessly.

The aetiological factor is rhesus incompatibility in the parents and the action of rhesus agglutinins on the foetus. The disease is the continuing stage of erythroblastosis foetalis in survivors.

The spleen has no influence on the incidence of haematemesis or on the condition of the liver and progress of the lesions, and only a slight secondary influence, if any, on the blood. There is no evidence that splenectomy is beneficial. The operative mortality is high owing to the extensive adhesions.

Summary

Banti's disease and splenic anaemia form the syndrome of erythroblastosis foetalis as it appears in survivors at various ages affected in different degrees of severity.

Splenectomy has no beneficial result.

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THE SURGICAL TREATMENT OF BANTI'S SYNDROME*

BY

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In 1894 Guido Banti, professor of pathological anatomy at Florence, described a disease in which splenomegaly is associated with cirrhosis of the liver and, to use his own words, "which progresses slowly, but it inexorably drags the patient to death." It was later shown that this was not a distinct disease, so the term "Banti's syndrome" was coined. It is loosely applied to patients who attend, in the first place, with large overactive spleens and gastro-oesophageal varicosities, from which there has usually been at least one haemorrhage. It is now known that this condition is associated with portal hypertension (Eppinger, 1920; Rousselot, 1940, 1949) due to interference with the free flow of portal blood, through the liver in cirrhosis or to the liver in extrahepatic obstruction. There may be portal-vein thrombosis as well as cirrhosis. The syndrome therefore develops erratically. It may occur in an otherwise comparatively healthy individual with a normal or only mildly cirrhotic liver, or it may be superimposed on a

case presenting essentially with liver disease. On the other hand, many cases of advanced cirrhosis do not have enlarged spleens or oesophageal varices. Thus the groups of cases overlap each other to a considerable extent. An investigation into treatment should therefore embrace the whole group.

The present report is based on a study of every patient with this syndrome or disease referred to me during the past five years, and whose investigation and initial treatment have been completed. It is in no way a selective group, and the majority with cirrhosis were referred to me on account of haemorrhage, or ascites after failure of medical treatment. They number 68. In 12 the portal obstruction was extrahepatic; in 11 there was mild cirrhosis; in 30 the cirrhosis was advanced; and 15 were in the terminal stages of chronic liver disease.

Group 1: Extrahepatic Portal Obstruction (Table I)

These 12 cases may be divided into two subgroups: (1) those in which portal vein obliteration is the primary condition (8 cases), and (2) those in which the obstruction is secondary or incidental to some other disease (4 cases). In the former the obstruction is probably an extension of the obliterative process affecting the umbilical vein and the ductus venosus at birth (Thompson, 1940). The normal portal vein often disappears and is replaced by a bunch of tortuous varicosities, the so-called cavernoma (Fig. 1). Occasionally the vein is present but constricted. The patients are children or young adults.

It is now generally agreed that the correct treatment is to remove the hyperactive spleen and to anastomose the splenic vein to the left renal vein — end-to-side if possible (Fig. 2), but end-to-end if the renal vessels are not conveniently arranged. There is usually no other serviceable portal venous channel, as shown in Fig. 1. A successful suture anastomosis is curative in that it enables the portal venous pressure to drop to a safe level (Fig. 3).

FIG. 1.—Venogram of portal cavernoma at operation. The portal vein is replaced by many tortuous channels. The dilated left gastric vein establishes connexion with the oesophageal varicosities (Case 49, Table I).

FIG. 2.—Diagram of end-to-side spleno-renal anastomosis, clamps in place.

*Report based on a Hunterian Lecture given at the Royal College of Surgeons on February 7, 1952.

TABLE I.—GROUP I—EXTRA-HEPATIC OBSTRUCTION

CASE NO.	AGE	SEX	CAUSE	BEFORE 1946	1946	1947	1948	1949	1950	1951	PRESENT CONDITION
1	17	F	=	H	-----	-----	(S-R)	HH	(T)	H-H	IMPROVED
21	29	M	P			H	-----	HAEMORRHAGE			
6	25	M	1935 (S)					H (P-C)			(H) WELL
12	5	M	IDIOPATHIC STEATORRHEA		H		A	-----	(S)	H	MESENTERIC THROMBOSIS
			= SPLENIC & PORTAL VEIN THROMBOSIS								
49	10	F	=				(S)	J	(P-C)	(B)	IMPROVED
56	16	F	=						H	(S-R)	VERY WELL
59	6	M	=					H	-----	(S-R)	VERY WELL
63	5	F	=						H	-----	(S-R) WELL
			= PORTAL VEIN CONSTRUCTION								
20	67	F	=		(S)	LYMPHOSARCOMA					
			= SPLENIC VEIN THROMBOSIS								
47	56	M		H	-----					(N) PG	VERY WELL
			= CIRROID ANEURYSM & HYPERNEPHROMA RIGHT KIDNEY, DUODENAL ULCER								
23	52	F	=					J A	(E)	CARCINOMA GALL BLADDER	
			= SPLENIC ARTERIO-VEIN ANEURYSM								
55	50	M						H	-----	HA HA	HAEMORRHAGE
			= MYELOID LEUKAEMIA								
			= PORTAL VEIN THROMBOSIS WITH CAVERNOMA								

KEY TO SYMPTOMS

A = ASCITES
H = HAEMORRHAGE
J = JAUNDICE

KEY TO OPERATIONS

B = BILIARY
E = EXPLORATORY LAPAROTOMY
N = NEPHRECTOMY
CE = CESOPHAGO-GASTRECTOMY
P-C = PORTA-CAVAL ANASTOMOSIS
PG = PARTIAL GASTRECTOMY
S = SPLENECTOMY
S-R = SPLENO-RENAL ANASTOMOSIS
T = TANNER'S GASTRIC TRANSECTION
H = HERNIOTOMY

Four of my eight patients (Table I) had spleno-renal shunts. In Case 1 a Blakemore-Lord tube was used and the anastomosis thrombosed. In Case 63 the splenic vein was such that I doubt the shunt will remain patent. The other two (Cases 56 and 59) are very satisfactory.

In Cases 6 and 49 the spleen had been removed previously. In both an anastomosis of sorts was made between a cavernomatous channel and the inferior vena cava. Both thrombosed. These tortuous varicose veins (Fig. 1) give a most unsatisfactory shunt, and it is usually futile to try to utilize them. In addition, the normal anatomy (Douglass *et al.*, 1950; Falconer and Griffiths, 1950) is so badly disturbed and the mass of brittle, hypertensive vascular tissue so difficult to handle that there is great risk of damage to the biliary passages. This happened in Case 49, in which the common hepatic duct has since been anastomosed to the jejunum.

In cases in which the spleen has been removed, or in which a spleno-renal shunt has thrombosed, severe haemorrhages continue to occur. These patients make up the majority of the group of so-called post-splenectomy bleeders, and they are very difficult to treat satisfactorily. The condition will become rarer when it is more widely recognized that every care should be taken to make a good spleno-renal shunt at the first operation. The anastomosis should be by intima-to-intima approximation by suture. Simple splenectomy should be done only when there is proof positive that splenic vein obliteration is the sole manifestation of portal disease. Venography (Learmonth, 1947) is essential to demonstrate this and the true state and actual position of the portal vein, or to show if a patent stump of splenic vein remains, or to test a previously constructed shunt (Fig. 1). Blake-

more and Fitzpatrick (1951) emphasize the difficulties of dealing with these cases and describe a method of anastomosing the stump of the splenic to the renal vein by way of a vein graft. This operation might have been possible in the child, Case 12 (Table I), who finally died of mesenteric thrombosis. Only at necropsy was it disclosed that the hepatic end of the splenic vein was patent.

It is tempting to use the superior mesenteric vein in the making of a shunt. However, its radicles are bunched up in such a way that it is difficult to obtain a sufficient length of vein by dissection.

Failing a shunt—and even Phemister and Humphreys (1947) agreed that a shunt is best—good results sometimes may be obtained by resecting the lower 4 in. (10 cm.) of the oesophagus and the proximal two-thirds of the stomach. Such an operation was done in Case 6 (Table I). It is interesting to note that it was 14 years between his splenectomy and the next haemorrhage, but this required altogether 24 pints (13.6 litres) of transfused blood. The result is satisfactory, and a venogram which was done at a subsequent herniorrhaphy shows that two and a half years after the resection there is no gross communication between portal and oesophageal veins at the anastomosis.

Oesophagoscopy confirmed the absence of oesophageal varices.

The portal pressures (Fig. 3) are of interest in that they are in no way related to the presence or absence of liver disease or to the degree of ascites.

Papers appear from time to time suggesting that the operative risks are too great to justify these large shunting operations. In my experience the risks of a properly conducted spleno-renal anastomosis in this and the second group are small, as Tables I and II show. Not to operate may lead to a tragedy, as with Case 21 (Table I), this patient dying

Fig. 3.—Portal (jejunal) venous pressures in 3.8% sodium citrate. (Only pressures considered accurate are included on this chart.) Note: (a) The low pressure immediately after a gastro-oesophageal haemorrhage (Case 6). (b) The greatest fall (470 mm.) in Case 56. (c) The difference between direct portal and jejunal pressures (Case 66). (d) The fall from 260 to 150 mm. pressure resulting from arterial ligation (Case 61). (e) The lack of correlation between the portal pressure reading and (i) the presence or absence of cirrhosis, (ii) the extent of liver disease, and (iii) the degree of ascites.

TABLE II.—GROUP 2—MILD CIRRHOSIS

CASE NO.	SEX	AGE	CAUSE	BEFORE 1946	1946	1947	1948	1949	1950	1951	PRESENT CONDITION
29	M	42	P				DUODENAL ULCER		(P-G)		WELL
10	M	29	1931 J INFECTIVE HEPATITIS			H		H	(S-R)		VERY WELL
26	F	49	CHRONIC ARSENIC POISONING					A	(A ²)		WELL
15	M	33	P				H	H	(S-R)	(A ²)	VERY WELL
30	M	22	P			H	(E)	H	H	(S-R)	VERY WELL
31	M	54	1944 J-H SYPHILITIC			AJ	(E)	A POTASSIUM IODIDE AND PENICILLIN		(A ²)	WELL
32	M	45	1937 H INFECTIVE HEPATITIS			H		S	(S)	PULMONARY EMBOLUS, 3 MONTHS	
54	F	40	BILIARY					(B)	J, GE	(A ²)	IMPROVED
44	F	38	1939 H					H		(S-R)	VERY WELL
66	M	26	P					(S)	PORTAL VEIN THROMBOSIS	H	MESENTERIC THROMBOSIS, IMPROVING
70	M	26	P						H	H	

KEY TO SYMPTOMS
A = ASCITES
H = HAEMORRHAGE
J = JAUNDICE

KEY TO OPERATIONS
A² = APPENDICECTOMY
B = BILIARY
E = EXPLORATORY LAPAROTOMY
GE = GASTRO-ENTEROSTOMY
P-C = PORTA-CAVAL ANASTOMOSIS
P-G = PARTIAL GASTRECTOMY
S = SPLENECTOMY
S-R = SPLENO-RENAL ANASTOMOSIS

of his second haemorrhage. In the very young (Cases 12, 59, and 63) operation should be deferred as long as possible, because a satisfactory shunt of good size is difficult to construct with their small vessels. However, an attempt must be made when repeated haemorrhages and persistent splenic anaemia endanger the child's life.

Sometimes one finds patients with enormous natural portal-to-systemic communications in safe regions of the body (McIndoe, 1928); but the existence of these channels is of no significance if the portal venous pressure is high and the patient bleeds (cf. Beswick and Butler, 1951). They are thin-walled and tortuous and are easily compressed. The flow through them is sluggish. It is possible to demonstrate a rapid fall in pressure between portal tributary and abdominal parietal communicating vein—for example, 570 to 260 mm. water in Case 36 (Table III).

An artificial shunt, as distinct from a natural collateral anastomosis, is a flood channel which drains off the high-pressure portal blood by a short, direct, and protected course.

Incidental Portal Obstruction.—There were four cases, of which two were seen only at necropsy and are included by permission of Mr. Rupert Corbett and Dr. Edward Cullinan (Table I). The prognosis for these patients depends on whether the primary disease can be successfully treated or not, and on whether the associated tendency to bleed and splenic anaemia can be relieved. Case 47 is fortunate so far in both respects. Case 20 was temporarily improved. Case 55 illustrates that a cavernoma can form in adult life. All four provided fascinating problems in diagnosis.

Group 2: Mild Cirrhosis (Table II)

Clinically these patients are difficult to distinguish from those in Group I. The liver-function tests are almost always normal; but on the whole the patients are older, there may be a history of some factor predisposing to cirrhosis, the liver may feel hard, and a liver biopsy will often be diagnostic (Terry, 1949). The distinction is important, because in cirrhosis it is likely that both the splenic and the portal vein will be available for a shunt.

In the present series, 7 of the 11 patients with mild cirrhosis required treatment for Banti's syndrome. Five of the seven had spleno-renal anastomoses (Cases 10, 15, 30, 44, and 70). The results are excellent. These patients have all been changed from apprehensive invalids, usually

chronically anaemic and tethered almost literally to a haematologist's apron strings, to normal young people, doing whatever they want to do, going where they want, and living lives which would have been considered impossible before the pioneer work of Whipple (1945), Blakemore and Lord (1945), Blakemore (1947, 1949a, 1949b, 1951), Rousselot (1940, 1949), Linton *et al.* (1948), Linton (1949), Welch (1947, 1950), Blalock (1948), and others. Blakemore, who has the greatest experience, in general favours the porta-caval anastomosis, but it is difficult to judge from his reports if he takes the same view in respect of the mild cirrhotic with marked hypersplenism. The patients are young adults with large vessels. In the circumstances it seems logical to treat the hypersplenism by splenectomy (Fig. 4) and use the splenic vein for a shunt there and then. Stomata of between 10 and 18 mm. diameter can be constructed.

Two of the seven patients had splenic and portal-vein thrombosis respectively, which implies that portal venography may be necessary in this group to disclose the true state of the portal venous tree. The patient (Case 32) whose platelets were very high after unavoidable splenectomy has since died of pulmonary embolism. Case 66, a post-splenectomy cirrhotic bleeder, had, perforce, a porta-caval anastomosis done in spite of partial portal-vein thrombosis. This had been followed by a carefully controlled course of heparin. A few months later he developed intestinal obstruction and coma from mesenteric thrombosis, which was successfully treated with heparin and "aureomycin." He is well again, but remains under close supervision.

Heparin is now not usually used as an adjunct to operation, owing to the danger of post-operative haemorrhages, retroperitoneal or otherwise. Regional heparinization (Blakemore and Fitzpatrick, 1951), however, may be valuable in certain circumstances.

Four patients under observation for cirrhosis (Table II) required treatment and operations for conditions other than bleeding varices or splenic anaemia. The results of medical treatment were very good when a specific cause for the cirrhosis could be stopped (Case 26) or treated (Case 31). Case 54 illustrates the tragic progress of biliary obstruction caused by trauma. Biliary cirrhosis must be treated in stages, and the first stage, undoubtedly, is to relieve the

Fig. 4.—Graph showing rapid increase in platelets and white blood cells on removal of an overactive enlarged spleen (Case 30, Table II)

biliary obstruction. Congenital atresia of the bile ducts is the most difficult of such conditions to attempt to alleviate, and in this series it has been impossible to benefit three such cases.

Group 3: Advanced Cirrhosis (Table III)

Advanced cirrhosis presents a far more difficult problem—the failing liver. So far we have been concerned with how best to stop haemorrhage, preferably by reducing portal pressure, and to correct hypersplenism—in effect, to relieve symptoms by mechanical means. When the liver is badly damaged, however, preservation of liver function is the first consideration. Haemorrhages, which in Groups 1 and 2 could be considered simply on their merits as bleeds, must also now be thought of as acute episodes precipitating further anoxic degeneration of a deteriorating liver. They must therefore be stopped. Yet the best way of stopping them, by diverting portal blood from the liver, may cut off so much that the liver fails completely.

Ascites, though not so dramatic, is a very grave matter. The days of an advanced ascitic are numbered, and such life as remains is little more than a misery. Occasionally medical treatment may be effective, particularly if there is a specific cause which can be remedied; but usually the condition is progressive, the functions of the heart and lungs are impaired, and the oedematous patient wastes away sooner rather than later.

In studying Table III we must recognize that the disease we are considering is malignant in all but microscopical pathology. Every one of these patients was referred to me on account of haemorrhage or because medical treatment had failed. It was inevitable that they would have died. The purpose of surgical treatment was to try to save some of them, however great the risk.

Portal-to-systemic anastomosis seemed the only effective method of treatment in the earlier years. Altogether 15 shunt operations were done in this group. Six of the first eight (Cases 2, 3, 8, 9, 11, and 13) resulted in death, and one patient (Case 14), whose shunt thrombosed, has since died following resection. However, only two of the last seven had a fatal outcome. One was an operation of exceptional difficulty, and the patient (Case 51) died of post-operative shock. The more often the patients have been operated on in the past, and the longer the symptoms of cirrhosis have been present, the more difficult the operation tends to become. In Cases 62 and 67 the tissues around the portal-vein remnants were impossible to handle, probably on account of repeated thrombosis and recanalization. One patient (Case 36) has since died of serum hepatitis.

Encouragement may be drawn from the survivors (Cases 7, 38, 50, 52, and 65). Their clinical condition has improved, there has been no further haemorrhage, jaundice has cleared, and ascites has disappeared. Three had had infective hepatitis, and in one the cause of the cirrhosis was obscure. In the patient (Case 7) who had advanced arsenical cirrhosis with gross ascites a porta-caval anastomosis in continuity was completely successful (Fig. 5). Whether an end-to-side anastomosis would have been equally effective is a moot point.

TABLE III.—GROUP 3—ADVANCED CIRRHOSIS

CASE NO.	SEX	AGE	CAUSE	BEFORE 1946	1946	1947	1948	1949	1950	1951	PRESENT CONDITION
2	F	30	?	1940 H	HJ	H	H	P-C HAEMORRHAGE (HEPARIN) 3 DAYS			
18	M	53	?	2 ALCOHOLIC			A	S LIVER FAILURE, 10 DAYS			
3	M	38	?	1938 H	H		H	P-C LIVER FAILURE, 6 DAYS			
7	M	51		CHRONIC ARSENIC POISONING			A	P-C			VERY WELL
24	F	30		TOXAEMIA OF PREGNANCY			A	TP A		A	IMPROVED
9	F	38		CARDIAC		A		P-C			
8	M	36	?				H	P-C LIVER FAILURE 4 DAYS			
11	M	53		1938 H	GE DUODENAL ULCER		A	A	S-R LIVER FAILURE, 15 DAYS		
				1945 A	STARVATION (P.O.W.)						
14	M	36		SYPHILITIC ?			H	S-R		H	LIVER FAILURE
13	M	45		1932 J			H	A H	P-S LIVER FAILURE, 6 DAYS		
27	M	45	?	1929 H	S	H (+S)			E		ACUTE NEPHRITIS
22	F	20		1945 J				HA		HJ	LIVER FAILURE
				INFECTIVE HEPATITIS							
41	F	20	?						A		LIVER FAILURE
36	F	48	?		RETINITIS PIGMENTOSA			H	S-R		SERUM HEPATITIS, 3 MONTHS
34	F	45		1937 B		J J		J A	J	E	WORSE
				INFECTIVE HEPATITIS							
38	M	28		1934 H	S	H		J	H	P-C	VERY WELL
				INFECTIVE HEPATITIS, RECURRENT							
35	F	67		BILIARY		ADVANCED PARKINSONISM		J	A	B	LIVER FAILURE

Thus experience has taught us that in this group the majority of patients will not tolerate a shunt, though clinical assessment and careful estimation of the liver function will probably enable one to select those that will. The tests, however, are tests of failure rather than of function. The discovery of true quantitative liver tests will be of the greatest value in this respect and in tracing the progress of the disease.

TABLE III (CONTD).—GROUP 3, CONTD.

CASE NO.	SEX	AGE	CAUSE	BEFORE 1946	1946	1947	1948	1949	1950	1951	PRESENT CONDITION
45	M	35		1942 J G (4)				H J A		CE LSA PNEUMONIA AND LIVER FAILURE, 7 WEEKS	
				INFECTIVE HEPATITIS, RECURRENT							
53	M	49		1932 B				B	H	J	MUCH IMPROVED
				BILIARY							
51	F	43	?				H	T A H J	P-C		SHOCK 7 HOURS
48	M	30		1944 J	J			A		CE A L	PNEUMONIA AND LIVER FAILURE, 4 TH DAY
				INFECTIVE HEPATITIS, RECURRENT							
50	F	14	?					H		S-R	WELL
52	M	11						J H	H	HA J S-R	WELL
				INFECTIVE HEPATITIS							
58	F	48	?					A		TM	IMPROVED
62	M	52	?	1919 H G (2)		H G (9)				E	I.S.Q.
				S							
65	F	13				J				H G (3)	WELL
				INFECTIVE HEPATITIS							
61	F	57	?							A E	WELL
69	F	15		1940				SPLenic ANAEMIA		S-R	LIVER FAILURE 13 TH DAY
				INFECTIVE HEPATITIS (?)							
60	M	55	?					J	E	HA	PNEUMONIA AND LIVER FAILURE, 12 TH DAY
67	F	54						J	E	HA	HEART FAILURE AFTER OPERATION
				CONGENITAL BILIARY ATRESIA							

KEY TO SYMPTOMS

A = ASCITES
H = HAEMORRHAGE
J = JAUNDICE

AL = ARTERIAL LIGATION
AN = ALLISON'S INTERRUPTION
B = BILIARY
E = EXPLORATORY LAPAROTOMY
GE = GASTRO-ENTEROSTOMY
IM-C = INFERIOR MESENTERIC-INFERIOR VENA CAVA ANASTOMOSIS
CE = OESOPHAGO-GASTRECTOMY

KEY TO OPERATIONS

P-C = PORTA-CAVAL ANASTOMOSIS
P-S = PERITONEUM-SAPHENOUS VEIN ANASTOMOSIS
P-S-E = PORTAL-SYSTEMIC EXCHANGE
S = SPLENECTOMY
S-R = SPLENO-RENAL ANASTOMOSIS
T = TANKER'S GASTRIC TRANSSECTION
TM = TALMA MORISON OMENTOPEXY
TP = TERMINATION OF PREGNANCY

Porta-caval shunt, end-to-side

Porta-caval shunt, in continuity

FIG. 5.—Diagrams of the two types of porta-caval anastomosis.

informed of it by Dr. William F. Rienhoff, jun., of Baltimore. His early results are good. As an operation it sounds very final, but it seems that an extended and well-controlled trial is justified. In the present series it was done on four patients with ascites (Cases 48, 58, 60, and 61), with or without haemorrhage, in whom the cirrhosis was too advanced for a shunt. The original operation was done in Cases 48 and 60, but both patients died of bronchopneumonia, the latter after recovering from hepatic coma. Case 61 had the coeliac axis tied. Her portal venous pressure dropped from 260 to 150 mm. of water. Her ascites and peripheral oedema have disappeared, and her general condition has improved, and her blood bilirubin has dropped in six weeks to less than half the pre-operative level. How the improvement is obtained is a matter for speculation. It may be that cutting off the high-pressure arterial blood relieves congestion within the inelastic portal tracts and thereby allows the portal blood, bile, and hepatic lymph to flow more freely. It must be possible for nutrition of the liver to be maintained by portal blood alone, but whether the liver improves in function or not time alone will tell. The stopping of ascites certainly saves wastage of metabolized protein and thereby achieves a relative improvement in liver function. The patient (Case 58) who has been bed-ridden with ascites for two and a half years was operated on only a fortnight before writing by a transpleural approach to the coeliac axis, with division of the origin of the diaphragm. An easier and more direct

There are means of mitigating haemorrhage other than by reducing portal venous pressure, such as resection (following which the two patients in this group died); or by interrupting portal-to-systemic communications, from above the diaphragm by venous ligation (Allison, personal communication), or from below the diaphragm combined with gastric transection (Tanner, 1950). These operations, which aim at simplifying the treatment, are no more than palliative, and both operations failed in Case 51.

A new operation—common hepatic and splenic arterial ligation—has been evolved (Berman *et al.*, 1951). I was

access to the trunk of the vessel was thereby obtained, but she was not materially improved. All these four patients had aureomycin.

Group IV : The Terminal Stages of Cirrhosis (Table IV)

Thus we see that there are great variations in the results of this report must be judged. These 15 cases were regarded as hopeless at their first assessment. The rapid deterioration of most can be seen by a glance at the Table. No treatment, either medical or surgical, was of any avail. The most radical of the therapeutic measures to be tried was the operation labelled here as portal-systemic exchange, devised to drain off the portal blood by anastomosing the portal vein to the inferior vena cava or the right renal vein (after the removal of the kidney), and to replace this source of blood to the liver by anastomosing the right renal artery to the hepatic end of the portal vein (Fig. 6). It was done altogether on four occasions. One patient (Case 13, Table III) was in hepatic coma (following spleno-renal anastomosis) at the time of the operation, and the other three (Cases 16, 17, and 42) had all been through at least one period of partial or complete insensibility due to liver failure. I mention it only to show that there may be, in this operation, a way of permanently reconstructing the severed arterial supply to the liver. Yet the reverse operation, that of arterial ligation, at present seems to hold out most hope for these unfortunate individuals.

Conclusions and Summary

Thus we see that there are great variations in the course of this inconsistent and miscellaneous disease process. We shall therefore have to wait some years before the final results of treatment can be assessed, and before the effects of these apparently unphysiological operations are fully understood. The outlook for many of the patients is still depressing, but the study is in its infancy, and there is much to encourage us.

TABLE IV.—GROUP 4—TERMINAL STAGES OF CIRRHOSIS

CASE NO.	SEX	AGE	CAUSE	BEFORE 1946	1946	1947	1948	1949	1950	1951
19	F	55	?			J-S	LIVER FAILURE, 3 MONTHS			
23	M	51	ALCOHOLIC			H-G-S	J	HAEMORRHAGE AND LIVER FAILURE		
4	F	72	BILIARY				J-S	LIVER FAILURE		
5	F	36	CARDIAC				J-A	PNEUMONIA AND LIVER FAILURE, 27 DAYS		
25	F	53	?				H-A-J	TM	LIVER FAILURE, 3 DAYS	
17	M	43	1938 J (S) INFECTIVE HEPATITIS				J-A	P-S	LIVER FAILURE, 4 DAYS	
16	M	51	?				H-E	J-A	LIVER FAILURE AND SHOCK, 24 HOURS	
33	F	50	J INFECTIVE HEPATITIS					H-A	LIVER FAILURE	
37	F	60	P ALCOHOLIC				J-A	B TH	LIVER FAILURE	
40	M	6	SINCE BIRTH J BILIARY ATRESIA				H	A	H	HAEMORRHAGE AND LIVER FAILURE
42	M	44	?					J-A	P-S	HAEMORRHAGE (HEPARIN), 2 DAYS
46	M	52	ALCOHOLIC			A	TM	A	H-H	HAEMORRHAGE AND LIVER FAILURE
39	F	63	?					A-J	LIVER FAILURE	
57	M	3	SINCE BIRTH J BILIARY ATRESIA				B	B	H	LIVER FAILURE
68	F	61	STARVATION						H-A	HAEMORRHAGE AND LIVER FAILURE

KEY TO SYMPTOMS

A • ASCITES
H • HAEMORRHAGE
J • JAUNDICE

KEY TO OPERATIONS

B • BILIARY
BTH • CROSSIE-CUNNEY BUTTON
E • EXPLORATORY LAPAROTOMY
F • FASCITOMY
P-C • PORTA-CAVAL ANASTOMOSIS
P-S • PORTAL-SYSTEMIC EXCHANGE
S • SPLENECTOMY
TM • TALMA MORISON OMENTOPEXY

FIG. 6.—Diagram of the operation of portal-systemic exchange.

The immediate cause of Banti's syndrome is generally thought to be a damming back of the blood in the portal venous tree.

The portal-vein obstruction may be extrahepatic (12 cases) or intrahepatic as a result of cirrhosis (56 cases).

A cirrhotic liver is not necessarily associated with Banti's syndrome, but the conditions overlap to such an extent that it is advisable, in the present state of our knowledge, to consider them as one group.

The indications for operation are: (a) gastro-oesophageal haemorrhage; (b) hypersplenism; (c) ascites, after medical treatment has failed; and (d) associated conditions, such as biliary obstruction.

When the liver is normal or only mildly cirrhotic the operation of splenectomy (to remove the overactive spleen) and spleno-renal anastomosis (to reduce the portal venous pressure) gives excellent results, provided that (a) the splenic vein is patent, and (b) the veins are of sufficient size.

Portal venography is of great value in demonstrating the exact state of the portal venous tree.

In the case of one patient with extrahepatic obstruction who had no available splenic vein, resection of the lower 4 in. (10 cm.) of the oesophagus and proximal two-thirds of the stomach has proved successful.

Porta-caval anastomosis provides an alternative and most efficient method of reducing portal venous pressure, and is clearly indicated for gastro-oesophageal haemorrhages in the absence of hypersplenism.

Portal-systemic venous anastomosis should be done early in the course of the disease because it is hazardous in cases of advanced cirrhosis. Careful clinical and biochemical assessment is necessary to select cases suitable for this operation. Those that survive are much improved.

All venous anastomoses should be done by the suture method, with intima-to-intima approximation.

For the advanced cirrhotic patients who are considered unsuitable for a shunt the operation of common hepatic and splenic arterial ligation offers hope.

The case histories of a complete consecutive series of 68 patients are set out in tables, in which an attempt has been made to relate the underlying cause, the symptoms, and the duration of the disease to the treatment and the result.

Cirrhosis in the terminal stages has not responded to any form of treatment. A method of replacing the portal blood flow to the liver is briefly described, but this operation failed in four cases.

I would like to thank all those who have worked with me and the physicians and surgeons who have referred patients. Miss W. B. Cattle and Mr. N. K. Harrison, A.R.P.S., drew up the tables and the graph and provided the illustrations.

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SYMMETRICAL ARTERIAL OCCLUSION OF UPPER EXTREMITIES, HEAD, AND NECK: A RARE SYNDROME

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We have recently had under our observation two almost identical cases of a very unusual circulatory anomaly. The main effect of the anomaly is to cause profound restriction, in symmetrical fashion, of the blood supply to the upper extremities, head, and neck, with the production of certain symptoms. Our interest in these patients was aroused by noting, first, that the radial pulse was absent at both wrists, although the radial arteries themselves could be felt in the normal position; and, secondly, that pulsation was virtually absent in all the arteries of the upper limbs, head and neck.

Apart from embolism, trauma, and anatomical variations in the course of the radial artery, unilateral absence of the arterial pulse in an upper extremity is a rare phenomenon, and has been found chiefly in syphilitic aneurysm of the aorta and in dissecting aneurysm. Bilateral absence of the pulse is rarer still, and we have been able to collect only 16 cases from the literature. In examining the records of these we were at once struck with their close resemblance to our own patients. Thus in the great majority the arterial pulse was lacking not only at the wrist but in the upper extremities, and in the head and neck; indeed, the symptoms and clinical findings in the series of cases as a whole formed a remarkably constant pattern. The purpose of this paper is to discuss this syndrome, as it appears to be little known. Our own cases are first described.