

PORTAL HYPERTENSION: AETIOLOGY
AND MANAGEMENT

AT

KENYATTA NATIONAL HOSPITAL,
NAIROBI,
KENYA.

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A DISSERTATION SUBMITTED IN
PART FULLFILMENT FOR THE
DEGREE OF MASTER OF
MEDICINE (SURGERY) AT THE
UNIVERSITY OF NAIROBI.

NOVEMBER 1986.

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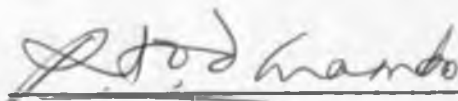
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ACKNOWLEDGEMENTS

I would like to extend my most sincere gratitude and appreciation to the following:

- (1) My supervisor Mr. P. A. Odhiambo of the Department of Surgery University of Nairobi, who assisted and guided me throughout this work.
- (2) Mr. Bodo, the Deputy Director of Kenyatta National Hospital for granting me permission to use the Records Department for data collection.
- (3) The staff of the Records Department Kenyatta National Hospital especially Miss Pamela Akoth for their assistance in data collection.
- (4) Express Services Bureau Limited for the obvious excellent secretarial work.

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SUMMARY

One hundred and ninety three patients with portal hypertension were studied at the Kenyatta National Hospital during the period June 1976 to June 1986. There were 130 males and 63 females, aged between one year and sixty years, the average age being 26 years.

The commonest presenting features were haematemesis, occurring in 154 (80%) cases, abdominal swelling, in 83 (43%) cases. Splenomegally occurring in 180 (93%) cases, hepatomegally 79 (41%), and ascites 76 (39%) were the commonest clinical signs.

On the basis mainly of the liver function tests, stool examination, rectal snip, liver biopsy and splenic venogram the various aetiological groups were identified. Schistosomiasis was the major cause of portal hypertension accounting for 93 (48%) cases, followed by liver cirrhosis, 40 (21%). Portal vein thrombosis was noted in 18 (9%) of the patients. In 42 (22%) patients the cause of portal hypertension was not established.

93 (48%) of the patients studied were from the Kamba tribe and 49 (26%) were from the Kikuyu tribe, representing 74% of all the patients studied, largely because of their geographical closeness to the Kenyatta National Hospital. 22 (11%) of the patients were from the Luo tribe, while 15 (8%) were from the Luhya tribe. It is noteworthy that of the 93 patients from the Kamba tribe 56 (60%) of them had schistosomiasis. This correlates well with the high incidence of portal hypertension and schistosomiasis seen in this tribe. It is also of interest that of the 22 patients studied from the Luo tribe 14 (60%) of them had schistosomiasis.

146 patients were treated medically, and 47 were treated surgically. The surgical means of treatment included splenectomy alone, stapling of oesophageal varices, mesocaval shunt, portacaval shunt, and splenorenal shunt. The poorest results were obtained with stapling of oesophageal varices. All the three patients who had this procedure died from recurrent haematemesis. Of the

three shunts, the splenorenal shunt appeared to have the least complications.

INTRODUCTION:

Portal hypertension and variceal haemorrhage are frequent problems in the hospitals of the developing world. A patient with portal hypertension may easily be dismissed as yet another cause of splenomegally (Decock 1983) and yet present later with exsanguinating haemorrhage from oesophageal varices.

Schistosomal portal fibrosis, cirrhosis of the liver and portal vein thrombosis are the main causes of portal hypertension in the tropics (R.H. Caruthers et al 1970, J.R. Miller et al 1972, Wicks et al 1972). On the other hand, alcoholic liver cirrhosis is the most important cause of portal hypertension and variceal haemorrhage in the Industrialized West (Lieber C.S. 1978).

It is obvious that there is no simple answer to the problem of portal hypertension and bleeding oesophageal varices because,

1. Bleeding from varices can be massive and repeated and often fatal.
2. Medical treatment is often ineffective.
3. Operations which are effective in preventing recurrent haemorrhage are technically difficult and associated with serious long term complications.

The increased attention paid to the disadvantages of portasystemic shunt operations has made some surgeons hesitant and caused others to advocate the abandonment of the use of traditional shunt procedures (Bengmark et al 1980). Over the past few years a number of different types of portasystemic venous shunts have been reported in the literature (Inokuchi, Warren, Linton); each an attempt to prevent the postshunt complications of the direct portacaval type. So far the distal splenorenal shunt as advocated by Warren appears to have the least complications but as to whether this will stand the test of time remains to be seen.

This study was undertaken to establish the prevalence and the causes of portal hypertension in this country, to compare this with previous published work and to find out the current surgical management of these patients at the Kenyatta National Hospital.

A review of literature on portal hypertension, and physiology and pathophysiology of the hepatic circulation has been included.

REVIEW OF LITERATURE:

The liver is supplied by both the hepatic arterial and portal venous circulation, the latter comprising 75% of total hepatic blood volume with an oxygen saturation of 85%. (Maingot). The portal veins primarily carry nutritional material from the gastrointestinal tract to the liver for metabolism and detoxification. Ligature of the portal vein in man is followed by survival but the pressure in the portal trunk rises with the establishment of collateral circulation. Total diversion of the portal blood in man for example, portacaval shunt is followed by the development of portasystemic encephalopathy and its consequences.

Portal pressure at laparotomy is 5 to 7 cm of saline and via the transplenic route 9 to 13 cm of saline. Wedged hepatic vein pressure is about 3 to 9 cm of saline. Portal hypertension is present when the portal pressure is persistently raised above 25 cm of saline (Maingot).

Portal hypertension produces a collateral circulation communicating with the systemic circulation. These collaterals are for the most part haemodynamically ineffective in reducing the portal pressure. When the portal pressure is consistently raised these collaterals form at the following sites:

1. Oesophagogastric region.
2. Falciform ligament.
3. Anal canal.
4. Retroperitoneal areas where intrabdominal organs come in contact with retroperitoneal structures.

Other channels may be reopened such as umbilical and paraumbilical veins originating from the left intrahepatic portal vein and joining with the deep epigastric and iliac veins. Since some of these communications may be quite large bleeding may never develop in some patients with portal hypertension. Clinically the patient may present as follows:

1. Upper gastrointestinal bleeding or malena.
2. Splenomegally.
3. Ascites.
4. Encephalopathy.

OTHERS:

Jaundice
Peripheral Oedema
Spider naevi
Caput medusae
Liver palms
Finger clubbing

In the presence of prolonged liver damage the following may be present:

Testicular atrophy
Muscle wasting
Gynaecomastia
Alopecia

Portal hypertension is not an entity in itself, rather it is the reflection of a pathological lesion causing haemodynamic changes in the portal system. It is usually caused by obstruction of the portal venous system or the hepatic outflow from the liver. The main causes therefore can be divided into presinusoidal intrahepatic and postsinusoidal, a classification which is of practical value because when the obstruction is presinusoidal hepatic function is relatively unimpaired and liver failure is uncommon when the varices bleed (McDermott Jr). On the other hand where there is parenchymal damage as in hepatic cirrhosis liver failure often accompanies or follows variceal haemorrhage. Summarized below is a simple classification of portal hypertension.

(I) PRESINUSOIDAL:

- (a) Extrahepatic Obstruction of Portal Vein.

The block to outflow of the portal blood may occur in the portal trunk or any of its

tributaries. Liver function is usually normal. Characteristically these conditions are associated with extensive collaterals and at times cavernous transformation of the portal vein owing to recanalization.

The blockage may be:

- (i) Congenital e.g. atresia of the portal vein.
- (ii) Traumatic.
- (iii) Thrombotic.

Thrombosis may be intrinsic such as may occur following:

- ° neonatal sepsis
- ° increased coagulability of the blood
- ° pyelophlebitis resulting from intrabdominal sepsis.

Extrinsic thrombosis may be caused by:

1. Pancreatic tumour by invasion of portal vein.
2. Diseases of lymph nodes.
3. Pancreatitis.
4. Previous Splenectomy.

(b) INTRAHEPATIC OBSTRUCTION OF PORTAL VEIN:

Lesions in portal vein or within sinusoids of the liver caused by:

- (1) Reticuloendothelial disease e.g. Hodgkins disease, leukemias, and myeloid metaplasia.
- (2) Sarcoidosis by granuloma formation.
- (3) congenital hepatic fibrosis associated with polycystic kidneys and cysts of the liver.
- (4) Schistosomiasis.

Here the pathological condition is such that the portal venous pressure is normal. Schistosomiasis is the most

common aetiological factor in the tropics (Hassab. M.A.). In general hepatic function is excellent in these conditions.

(II) POSTSINUSOIDAL BLOCK:

(a) Intrahepatic (occlusion of central veins or hepatic venules). Mainly caused by cirrhosis which produces impedance of blood flow through the liver by fibrosis, thrombosis and nodular regeneration. The cirrhosis may be:

- (i) Portal (Laennec's cirrhosis).
- (ii) Postnecrotic following hepatitis.
- (iii) Biliary.

The relationship between Laennec's cirrhosis and alcoholism are clearly established. Additional causes include:

- ° Wilson's disease
- ° Cystic Fibrosis
- ° Hepatic porphyria
- ° Sickle cell disease
- ° Haemachromatosis
- ° Gauchers disease

(b) Suprahepatic obstruction where there is obstruction of hepatic veins or inferior vena cava, such as in Budd-Chiari syndrome.

(III) INCREASED BLOOD FLOW INTO PORTAL SYSTEM

e.g. Arteriovenous Fistula.

This is an extremely rare cause of portal hypertension. Cases have been described, however, in which a fistula between the hepatic artery and portal vein or between the splenic artery and splenic vein; have resulted in significant portal hypertension and the development of oesophageal varices.

Portal hypertension as a cause of upper gastrointestinal haemorrhage through bleeding oesophageal varices is an important cause of acute admissions throughout the world. Indeed life threatening haemorrhage in patients with portal hypertension is frequently an indication for surgical therapy.

In Kenya the common causes of upper gastrointestinal bleeding are peptic ulcerations, oesophageal varices and gastritis/duodenitis according to the work of Carvalho et al (1974) Hansen et al (1978), and Thomas et al (1983) done at the Kenyatta National Hospital.

In Africa it appears that variceal haemorrhage from portal hypertension is an important cause of upper gastrointestinal bleeding. One study from Zimbabwe by Wicks, Thomas and Clain demonstrated a higher incidence of variceal bleeding among the Africans compared with the Europeans.

The higher incidence of variceal haemorrhage in Africans may be related to hepatic fibrosis caused by Schistosoma mansoni. R.H. Caruthers and P. Sinha carried out a study in Lusaka Zambia on 30 patients admitted with portal hypertension between 1971 and 1976 and found that 70% of the patients studied had schistosomal portal fibrosis while 20% had cirrhosis of the liver.

In Kenya, the situation appears similar. A recent endoscopic study done at Kenyatta National Hospital by S.E. Thomas et al, showed that the major causes of bleeding from the upper gastrointestinal tract were due to duodenal ulceration (51%) oesophageal varices (20%) and gastric ulcer (16%). The rest of the patients studied (20%) had more than one bleeding site. It was noted that 67% of the fifteen cases of oesophageal varices were from the Kamba tribe who represented 28% of the total number of patients. This correlates well with the high incidence of portal hypertension and schistosomiasis seen in this tribe.

The authors however point out that the tribal distribution in their study may be due to the fact that the Kamba (28% of the patients studied) like the Kikuyu (39% of the patients studied) live closer to Kenyatta National Hospital and that the results are unlikely to be an accurate reflection of the overall distribution of this disease. However, Vogel, Muller, Onyango and Odingo reported that schistosoma mansoni infection is prevalent among the Kamba and that there is a high incidence of portal hypertension in this tribe.

It is now generally recognized that alcoholism is a major social problem confronting many societies of the world. In the Industrialized West, alcohol is the most important cause of cirrhosis. In the United States of America 75% of all deaths attributable to alcoholism are due to cirrhosis of the liver (Lieber C.S 1978).

The importance of alcohol as a cause of liver disease in the tropics is difficult to assess. Without doubt alcohol abuse is a neglected problem in the developing world (Edwards G 1979). While no detailed study on use and abuse of alcohol and other drugs has been done in this country up to date, preliminary investigations strongly indicate that the problem is widespread according to a review of literature by S.W. Acuda (1982) of the psychiatry department, University of Nairobi. He points out that the major drugs of abuse are alcohol, cannabis (bhang) miraa (catha edulis) and tranquillizers.

The first investigation was a direct result of frequent claims that Kisii District in Western Kenya seemed to have the worst alcohol and drug problem in this country; consequently the department of psychiatry and community health of the University of Nairobi with the help of medical students decided to carry out a survey in that district (Bittah O. et al 1979). The results were rather alarming. 27% of the males and 24% of the females could be classified as alcoholics according to the criteria specified in the second report of the Alcoholism Committee of World Health Organisation Expert Committee on mental health. A similar study was carried out in Mathare Valley, Nairobi. In this overpopulated slum of Nairobi the problem seemed even more

acute. Up to 46% of the males and 24% of the females studied were alcoholics according to W.H.O. criteria above. The majority of the alcoholics blamed lack of something to do during leisure time (i.e. unemployment and public holidays) for their excessive drinking (Wanjiru F 1979).

In a neighbouring country, Uganda, R. Owor carried out an autopsy study of 40 Ugandan chronic alcoholics and showed that 18 (45%) had significant fatty change and 14 (35%) had cirrhosis of the liver. While it is true that not all cirrhotic patients are alcoholic, alcoholism continues to present a growing problem in most societies and plays a part in liver damage which could eventually lead to portal hypertension.

Until recently liver cirrhosis in alcoholics was attributed entirely to the effects of malnutrition which is often part of chronic alcoholism. Several studies however now show that alcohol unquestionably has direct toxic actions on many tissues particularly the liver (Lieber C.S.).

The demonstration that ethanol is a direct hepatotoxin has important implications for prevention and therapy: since chronic alcoholic intake may lead to cirrhosis even in the presence of an adequate diet it is obvious that dietary supplementation cannot counteract its adverse effects on the liver. Total abstinence is the key to successful prevention; in alcoholics with established cirrhosis abstinence may considerably improve their chances of survival (Lieber C.S.).

There are special problems in the management of portal hypertension because:

- (1) Bleeding from varices can be massive and repeated and often fatal.
- (2) Medical treatment is often ineffective;
- (3) Operations which are effective in preventing recurrent haemorrhage are technically difficult and associated with serious long-term complications.

The history of shunt surgery dates back to 1877 when NICHOLAI ECK described a series of experiments in dogs.

This was done by constructing a lateral portacaval shunt and then converting this into a total diversion by ligation of the portal vein on the hepatic side of an already constructed shunt. Only one dog survived to leave the confines of the laboratory, but at least this marked the beginning of shunt surgery. (McDermott Jr.).

Several efforts were made at construction of portasystemic shunts by various workers but it was not until 1945 that Whipple reported a successful series of portasystemic shunts (McDermott 1977).

That many shunt procedures now exist is a pointer to the fact that none of them is best as definitive treatment for portal hypertension. Indeed as early as 1893, some 16 years after ECK reported that he had shunted the portal vein blood into the inferior vena cava of dogs, Hahn and his associates described the symptoms of hepatic encephalopathy in a group of ECK fistula dogs (McDermott Jr. 1977).

Hepatic encephalopathy is a potential long-term complication in any patient receiving a portacaval shunt and other traditional shunts (End to side portacaval, side to side portacaval, splenorenal and mesocaval shunts are usually regarded as traditional shunts). Such mental changes may negate the value of the operation in preventing recurrence of bleeding and reduce the quality of life. Some workers (Willis Maddrey 1977) believe that severe encephalopathy defined as episodes requiring hospitalization is considerably more frequent in patients who receive portacaval shunts than those who do not.

To operate or not to operate is many a time the dilemma in which the surgeon finds himself. Indeed the disappointing outcome of recent controlled trials of portacaval anastomosis (done by Conn H.O. et al, Jackson F.C. et al, Resnick R.H. et al) may suggest to clinicians that nothing can be done to improve survival in patients with portal hypertension. The above three studies have shown conclusively that survival is no different in the operated group and one showed it to be worse - the decreased frequency of bleeding is offset by the increased frequency of hepatic encephalopathy and liver failure.

There are however many inherent difficulties in setting up such trials that need to be considered before far - reaching conclusions are drawn. Most of these patients had alcoholic liver disease many with grossly abnormal liver function tests and many were in the upper age range for this condition. Despite the results of these recent trials clinicians remain ignorant about the virtues of shunting procedures for younger and non-alcoholic patients with portal hypertension who present with repeated episodes of variceal haemorrhage. Indeed Conn. H.O. expressed the opinion shared by most when he wrote:

"It is clear that we must learn either to select better who should be shunted or shunt better those we select."

Initial work by Davidson et al did suggest that postshunt encephalopathy may be prevented or reduced by selectively shunting blood from the portal system. They compared the blood ammonia levels in dogs in which end to side portacaval anastomosis was performed to those in which splenocaval anastomosis was constructed and found out that the dogs with splenocaval anastomosis maintained normal fasting arterial ammonia levels while those dogs with portacaval anastomosis had a three-fold rise of ammonia and later developed postshunt encephalopathy.

They attempted this procedure (spleno-caval anastomosis) in a patient operated upon for cirrhotic portal hypertension but found an extensive complex of large varices in the retroperitoneal area surrounding the splenic vein. Under this circumstances, it was decided that the time-consuming dissection necessary to identify the true splenic vein would render the procedure unfeasible: therefore a conventional portacaval anastomosis was performed. However it was assumed that in other patients with less severe retroperitoneal varices it would be technically possible to anastomose the distal end of the splenic vein to the left renal vein.

Warren later devised and popularized the technique of the distal splenorenal shunt. Here blood is selectively shunted from the system of vessels supplying the varices

but not from the portal system. Initial reports of this procedure have been encouraging: rates of postoperative bleeding and encephalopathy have reportedly been low.

MATERIALS AND METHODS:

A retrospective study over a ten year period was undertaken on patients with a clinical diagnosis of portal hypertension admitted to both medical and surgical units of the Kenyatta National Hospital Nairobi, from June 1976 to June 1986 by analysing obtainable data contained in their clinical case records kept in the hospital records department. 193 patients were admitted to the study. There were 130 males and 63 females aged between one year and sixty years. The aspects studied included analysis of the patient distribution during the study period, tribal distribution, age, sex, history of alcohol ingestion, presenting features, investigations and management. 146 patients were treated conservatively while the remainder 47 were subjected to surgical means of treatment.

Only those case records with complete clinical, diagnostic and management data were included. However in 22% of the cases (42 patients) the cause of portal hypertension could not be ascertained despite the fact that investigations for these were completed.

RESULTS:

TABLE 1: DISTRIBUTION OF PATIENTS OVER THE PERIOD
OF STUDY JUNE 1976 - JUNE 1986

YEAR	NUMBER OF PATIENTS		
	MEDICALLY TREATED	SURGICALLY TREATED	TOTAL
1976 JUNE - DEC 1976	2	4	6
1977 JAN - DEC 1977	10	2	12
1978 JAN - DEC 1978	13	5	18
1979 JAN - DEC 1979	18	3	21
1980 JAN - DEC 1980	25	3	28
1981 JAN - DEC 1981	13	10	23
1982 JAN - DEC 1982	15	4	19
1983 JAN - DEC 1983	9	5	14
1984 JAN - DEC 1984	21	6	27
1985 JAN - DEC 1985	15	3	18
1986 JAN - JUN 1986	5	2	7
TOTAL	146	47	193

Table 1 shows the distribution of the patients during the study period. The number of patients during the period of June 1976 to December 1976 would have been higher than indicated in the table above, but their case records were incomplete and therefore they could not be included in the study.

TABLE 2: DISTRIBUTION OF TRIBES

TRIBE	NO. OF PATIENTS	PERCENTAGE
KAMBA	93	48
KIKUYU	49	26
LUO	22	11
LUHYIA	15	8
EMBU	8	4
OTHERS: KALENJIN - 1)		
MASAI - 1)		
TAITA - 2)	6	3
MDIGO - 1)		
TANZANIAN- 1)		
TOTAL	193	100

The distribution of the patients according to tribes is shown in table 2. Out of the 193 patients studied 93 (48%) were from the Kamba and 49 (26%) from the Kikuyu tribe. This is perhaps partly explainable by the fact that the two tribes live close to the Kenyatta National Hospital.

TABLE 3: AGE DISTRIBUTION

AGE IN YEARS	NO. OF PATIENTS
0 - 4	5
5 - 9	6
10 - 14	19
15 - 19	27
20 - 24	37
25 - 29	31
30 - 34	24
35 - 39	12
40 - 44	14
45 - 49	11
50 +	7
TOTAL	193

Table 3 depicts the age distribution. The youngest patient studied was 1 year. The child presented with abdominal distension, yellowness of eyes and passage of pale stools and at laparotomy was found to have congenital biliary atresia with portal hypertension and a cirrhotic liver.

The oldest patient was 60 years and the average age 26 years. Mean age was 30½ years. Most of the patients studied were between 10 years and 34 years (71% of total patients). The age distribution is also shown below in figure 1.

There were 130 males and 63 females, a ratio of approximately 2:1.

FIGURE 1
AGE DISTRIBUTION

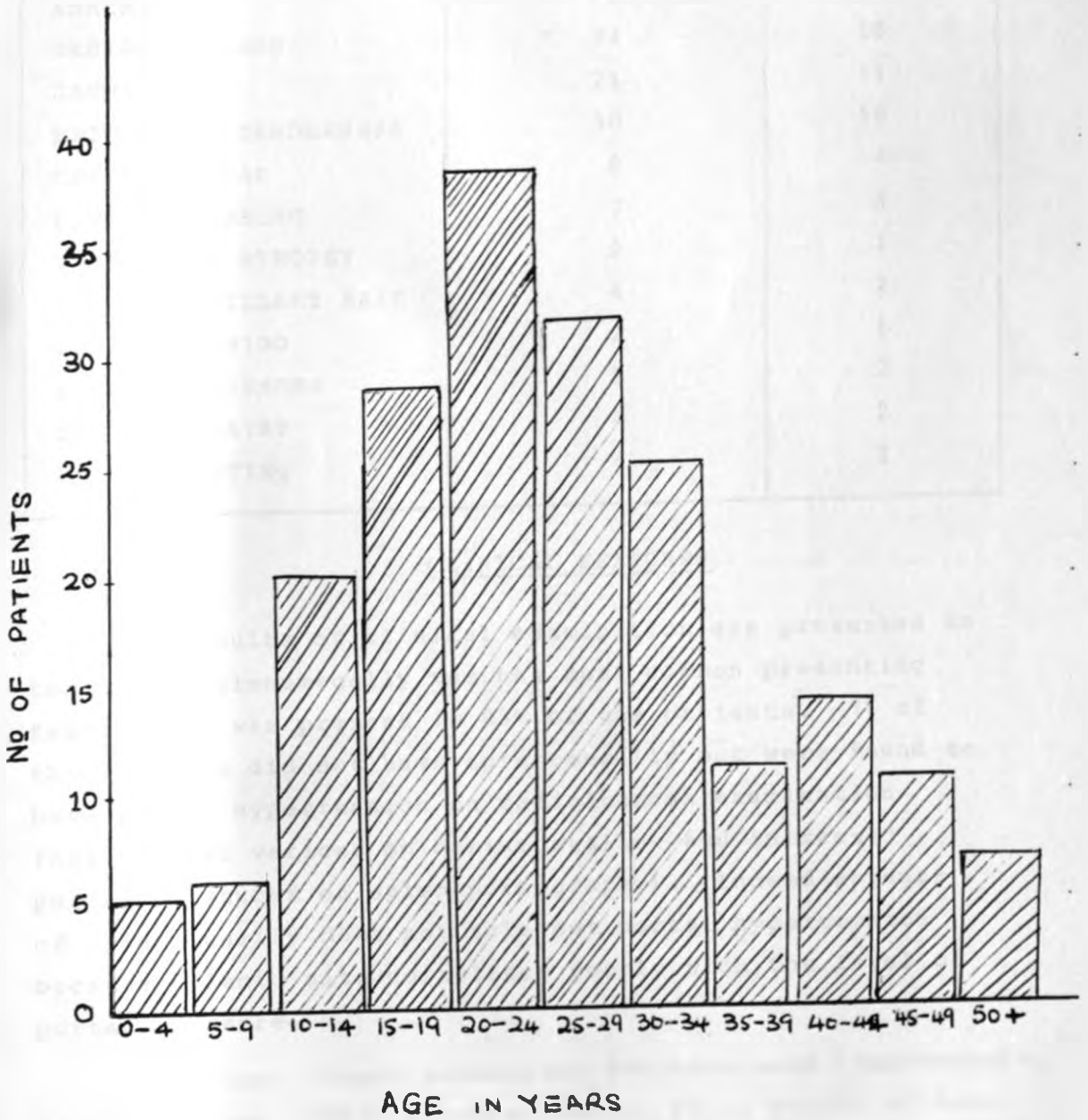


TABLE 5: PHYSICAL FEATURES

PHYSICAL FEATURE	NO. OF PATIENTS	PERCENTAGE
SPLENOMEGALLY	180	93
HEPATOMEGALLY	79	41
ASCITES	76	39
ANAEMIA	74	38
OEDEMA OF LEGS	34	18
JAUNDICE	21	11
EPIGASTRIC TENDERNESS	30	16
CAPUT MEDUSAE	8	4
FINGER CLUBBING	7	4
TESTICULAR ATROPHY	2	1
LOSS OF AXILLARY HAIR	4	2
LOSS OF LIBIDO	2	1
FLAPPING TREMORS	3	2
ENCEPHALOPATHY	4	2
MUSCLE WASTING	4	2

CLINICAL FEATURES

The results of clinical examination are presented in table 5. Splenomegally was the most common presenting feature and was present in 93% of the patients. 7% of the patients did not have splenomegally but were found to have portal hypertension on radiological examination (oesophageal varices on Barium swallow and increased portal pressures on splenoportography). Indeed absence of splenomegally does not rule out portal hypertension because splenic size correlates poorly with the level of portal pressure.

The other common presenting features were hepatomegally (41%), ascites (39%), anaemia (38%). It is worthy of note that 38% of the patients who presented with ascites, their liver function was still quite good, with serum albumin above 35 grams per litre and in 19%; the serum albumin was between 30 to 35 grams per litre which meant that they were still good candidates for surgery in spite of the presence of ascites.

TABLE 4: PRESENTING SYMPTOMS

SYMPTOM	NO. OF PATIENTS	PERCENTAGE
HAEMATEMESIS	154	80
ABDOMINAL SWELLING	83	43
MALENA STOOL	74	38
ABDOMINAL PAIN	51	26
FRANK BLOOD PER RECTUM	15	8
EPISTAXIS	13	7
LEG SWELLING	28	15
LOSS OF WEIGHT	14	7
BREAST SWELLING	5	3
CONFUSION AND LACK OF CONCENTRATION	11	6
YELLOW EYES	11	6
HAEMOPTYSIS	4	2

It is shown in table 4 that haematemesis (occurring in 80% of patients) was the most common symptom followed by abdominal swelling 43%, malena stool 38%, abdominal pain 26% and peripheral oedema 15%. 4 patients presented with haemoptysis and haematemesis, but investigations for tuberculosis were negative. They were later found to have portal hypertension.

TABLE 6: HISTORY OF ALCOHOL INGESTION

POSITIVE HISTORY	NEGATIVE HISTORY	HISTORY NOT AVAILABLE FROM CHART	TOTAL
31	24	138	193

Table 6 shows the data obtained on history of alcohol ingestion. Thirty one (16%) of the patients offered a positive history of alcohol ingestion while twenty four (12%) gave a negative history. It is noteworthy that in one hundred and thirty eight patients (72%) there was no mention of history of alcohol ingestion in the clinical notes; a regrettable point in consideration of aetiological implications. Indeed of the 31 patients who offered a history of alcohol ingestion 25 of them (81%) suffered from cirrhosis of the liver which was a cause of their portal hypertension.

TABLE 7: OTHER ILLNESS IN ASSOCIATION
WITH PORTAL HYPERTENSION

	NO. OF PATIENTS	PERCENTAGE
DUODENAL ULCER	32	17%
KALAAZAR	5	3%
HAEMORRHOIDS	6	3%
GASTRIC ULCER	5	3%
OESOPHAGITIS	13	8%

The above table (No. 7) shows the other conditions the patients had on complete clinical evaluation in the hospital. The diagnosis of duodenal ulcer, gastric ulcer and oesophagitis was made by endoscopy. However, one patient with duodenal ulcer presented with features of acute abdomen and on exploratory laparotomy was found to have perforated duodenal ulcer which was repaired. The liver at laparotomy was noted to be cirrhotic and a biopsy taken from this proved to be cirrhosis. The patient later had haematemesis which was conservatively managed.

Kalaazar was present in 5 patients and haemorrhoids in 6 patients. The patients with kalaazar were all from Machakos District and splenomegally in them was massive.

TABLE 8: CAUSES OF PORTAL HYPERTENSION

CAUSE	NO. OF PATIENTS	PERCENTAGE
SCHISTOSOMIASIS	93	48
LIVER CIRRHOSIS	40	21
PORTAL VEIN THROMBOSIS	18	9
CAUSE NOT ESTABLISHED	42	22
TOTAL	193	100

Table 8 shows the causes of portal hypertension after full investigations of the patients which included:

1. Liver function tests.
2. Stool for ova of schistosoma mansoni.
3. Rectal snip for ova of schistosoma mansoni.
4. Splenic portovenography.
5. Liver biopsy.

Schistosomal liver fibrosis was the commonest cause of portal hypertension and was found in 93 patients (48%). The second commonest cause was liver cirrhosis; occurring in 40 patients (21%). Two patients had biliary cirrhosis.

The third cause of portal hypertension was portal vein thrombosis; 18 patients (9%). Two of these patients had umbilical sepsis which later led to portal vein thrombosis.

The cause of portal hypertension could not be established in 42 patients (22%) in spite of full investigation.

The average age of patients presenting with schistosomiasis was 26 years, and 32 years in those presenting with cirrhosis of the liver. It was found to be 17 years in those presenting with portal vein thrombosis.

CAUSES OF PORTAL HYPERTENSION IN THE VARIOUS
TRIBES STUDIED

TABLE 9 (a)

KAMBA TRIBE		
CAUSE	NO. OF PATIENTS	PERCENTAGE
SCHISTOSOMIASIS	56	60%
LIVER CIRRHOSIS	13	14%
PORTAL VEIN THROMBOSIS	2	2%
CAUSE NOT ESTABLISHED	22	24%
TOTAL	93	100%

TABLE 9 (b)

KIKUYU TRIBE		
CAUSE	NO. OF PATIENTS	PERCENTAGE
SCHISTOSOMIASIS	16	33%
LIVER CIRRHOSIS	20	41%
PORTAL VEIN THROMBOSIS	6	12%
CAUSE NOT ESTABLISHED	7	14%
TOTAL	49	100%

TABLE 9 (c)

LUO TRIBE		
CAUSE	NO. OF PATIENTS	PERCENTAGE
SCHISTOSOMIASIS	14	64%
LIVER CIRRHOSIS	2	9%
PORTAL VEIN THROMBOSIS	1	4%
CAUSE NOT ESTABLISHED	5	23%
TOTAL	22	100%

TABLE 9 (d)

LUHYA TRIBE		
CAUSE	NO. OF PATIENTS	PERCENTAGE
SCHISTOSOMIASIS	1	7%
LIVER CIRRHOSIS	8	53%
PORTAL VEIN THROMBOSIS	2	27%
CAUSE NOT ESTABLISHED	2	13%
TOTAL	15	100%

TABLE 9 (e)

OTHERS		
CAUSE	NO. OF PATIENTS	PERCENTAGE
SCHISTOSOMIASIS	3	21%
LIVER CIRRHOSIS	5	36%
PORTAL VEIN THROMBOSIS	4	29%
CAUSE NOT ESTABLISHED	2	14%
TOTAL	14	100%

Tables 9 (a) to 9 (e) show the causes of portal hypertension in the various tribes studied. It is noted that of the 93 patients studied from the Kamba tribe 60% (56 patients) suffered from schistosomal liver fibrosis which was the cause of portal hypertension. Liver cirrhosis played a lesser role in causation of portal hypertension. A similar trend is noted in the Luo tribe where 64% of the patients studied suffered from schistosomal liver fibrosis.

In the Kikuyu and Luhya tribes liver cirrhosis appeared to be a major cause of portal hypertension. Liver cirrhosis was a cause of portal hypertension of 41% of patients studied in the Kikuyu tribe (20 patients) and 53% in the Luhya tribe (8 patients).

TABLE 10

PRESENCE OF ASCITES IN THE VARIOUS DIAGNOSTIC
GROUPS OF PORTAL HYPERTENSION

DIAGNOSIS	NO. OF PATIENTS	PERCENTAGE
SCHISTOSOMIASIS	32	42%
LIVER CIRRHOSIS	26	34%
PORTAL VEIN THROMBOSIS	4	5%
CAUSE NOT ESTABLISHED	14	19%
TOTAL	76	100%

Table 10 shows the presence of ascites in the various diagnostic groups of portal hypertension. Ascites tended to be mild in those patients with schistosomiasis. Only one patient had gross ascites in this group and this was intractable and difficult to treat by diuretics. Ascites in those patients with cirrhosis was moderate to gross, and especially so in those patients also presenting with jaundice; serum albumin in those particular patients was also low; a reflection of their poor liver function.

TABLE 11

AVERAGE SERUM ALBUMIN AND SERUM BILIRUBIN IN THE
VARIOUS DIAGNOSTIC GROUPS

DIAGNOSIS	SERUM BILIRUBIN (AVERAGE)	SERUM ALBUMIN (AVERAGE)
SCHISTOSOMIASIS	21.3 MILLIMOLS/LITRE	36 GRAMS/LITRE
CIRRHOSIS	53 MILLIMOLS/LITRE	28 GRAMS/LITRE
PORTAL VEIN THROMBOSIS	23 MILLIMOLS/LITRE	34 GRAMS/LITRE
CAUSE NOT ESTABLISHED	24 MILLIMOLS/LITRE	31 GRAMS/LITRE

Table 11 shows the average serum albumin and serum bilirubin in the various diagnostic groups in portal hypertension. On the whole, liver function was noted to be much better in those patients with schistosomiasis and portal vein thrombosis than in those patients with liver cirrhosis. Indeed liver cirrhosis causes destruction of the liver parenchyma and hence poor liver function is expected in these patients especially in advanced disease. On the other hand, there is relatively little liver damage in patients with schistosomal liver fibrosis and liver failure is not a serious problem in these patients.

MODE OF TREATMENT

Of the 193 patients studied 146 patients were treated conservatively while the remainder 47 were subjected to surgical means of treatment. Medical treatment included the following:

- (1) Blood transfusion to replace blood loss
- (2) Antiacids in those patients with features of peptic ulceration;
- (3) Aldactone and Lasix for relief of ascites.
- (4) Nasogastric suction
- (5) Neomycin and restriction of high protein diets in those patients presenting with features of liver failure.
- (6) Pitressin (vasopressin) infusion for control of bleeding.
- (7) Sangestaken Blakemore tube for control of bleeding. This was used in only six of the patients studied and at oesophageal pressures of 25-30 mmHg for 48 hours. There was malena in one patient after removal of the tube.
- (8) Propranolol.
- (9) Oxymaquin and Hycanthone for those patients found to have schistosomiasis.
- (10) Sclerotherapy at oesophagoscopy using ethenolamine.

It is evident from the records of patients studied that follow up was poor and it is therefore difficult to ascertain for how long after the diagnosis was made; the patients survived or stayed relatively symptom free. The longest follow-up period recorded was six years in those treated by conservative means.

A summary in terms of follow up for those patients treated conservatively is given below.

- (1) 50 patients were completely lost to follow up, i.e., were not seen again after discharge from the hospital.
- (2) 23 patients attended the liver clinic twice and were at the time recorded as symptom free, but were later lost to follow-up.
- (3) 52 patients are recorded as stable and still attend the liver clinic.
- (4) 11 patients had severe recurrent haematemesis that necessitated readmission for replacement of blood loss.
- (5) 5 patients died of liver failure while 4 patients died from severe haemorrhage that could not be controlled.

That portal hypertension presents difficult management problems is exemplified by the fact that some of the patients require repeated admissions for purposes of blood transfusion to replace blood loss, which in some situations called for massive blood transfusion. One female patient required a total of 35 pints of blood in a period of 6 months.

The choice of patients for surgery does not appear in some situations to have been entirely based on liver function tests as one hundred and eleven patients treated conservatively would have qualified for surgery and be "moderate to good risk candidates" according to Childs classification in terms of Hepatic reserve. See table 12 below.

TABLE 12. CLASSIFICATION OF CIRRHOTIC PATIENTS
IN TERMS OF HEPATIC RESERVE (CHILD AND TURCOTTE)

	GROUP A	GROUP B	GROUP C
SERUM BILIRUBIN Umole/litre	≤ 34.0	34.0-51.0	≥ 51.0
PLASMA ALBUMIN grams/litre	≥ 35	30-35	≤ 30
ASCITES	NONE	EASILY CONTROLLED	POORLY CONTROLLED
ENCEPHALOPATHY	NONE	MINIMAL	ADVANCED COMA
NUTRITION	EXCELLENT	MODERATE	POOR
RISK OF OPERATION	GOOD	MODERATE	POOR

33 patients of those studied fall under group A while 78 patients would be placed in group B. The remainder of those patients (35 patients) fell under group C. The patients who died from liver failure and severe haematemesis were from this last group.

Of the patients who had surgery for portal hypertension 32 were in group A, 11 in group B and 4 were in group C. 2 of the patients in group C died of haematemesis after surgery.

TREATMENT BY SPLENECTOMY ALONE

A total of 9 patients were treated by the procedure of splenectomy alone. One patient in this group had features of hypersplenism and two patients had thrombosed splenic veins. The other 6 patients in this group could not have shunts constructed because of various reasons including; friable portal and splenic vein, and multiple tributaries of these vessels that were easily bleeding and making the procedures difficult to perform.

Follow up of the above patients is recorded as follows:

- (1) 3 patients were lost to follow up on discharge from the hospital.
- (2) 2 patients were followed up for 2 years and in which period of time were symptom free.
- (3) 3 patients have remained symptom free and still attend the cardiothoracic clinic. The follow up period in one has been 8 years (1978 -1986) while the follow up period in the other has been 3 years.
- (4) One patient died of exsanguinating haemorrhage after splenectomy.

TREATMENT BY STAPLING OF OESOPHAGEAL VARICES:

A total of 3 patients underwent the procedure of stapling of oesophageal varices as an emergency measure to stop bleeding. One patient remained symptom free but died three years later from severe haematemesis. The remainder 2, developed haematemesis again after a period of 2 months and had portacaval shunts constructed. They both died about four months later from severe haematemesis.

TREATMENT BY PORTACAVAL SHUNTS:

8 patients were treated by construction of portacaval shunts. Two of these patients however had, had stapling of oesophageal varices which had failed to control the haematemesis (see above).

In 3 patients haematemesis recurred and death from severe haematemesis followed 2 to 4 months later. 2 patients developed hepatic encephalopathy after the shunting procedure; one patient after 2 months and the other after a period of 2 years.

2 patients were discharged in good condition but were lost to follow up. 1 patient remained symptom free for 2 years, but thereafter was lost to follow-up.

TREATMENT BY MESOCAVAL SHUNTS

A total of ten patients had mesocaval shunts. 2 of them as a repeat operation when other shunts had become thrombosed. 4 patients redeveloped haematemesis after a period of 3 years. Two patients have remained symptom free, but the follow-up period had been short (6 months), while the remaining 2 patients have been lost to follow-up.

TREATMENT BY SPLENORENAL SHUNT

21 patients underwent the procedure of splenorenal shunt. 6 patients were lost to follow-up after discharge from the hospital, while 12 patients are reportedly doing well and have remained symptom-free with an average follow-up period of 3 years. One patient developed haematemesis after a period of 1 year but still attends the surgical clinic.

THE PROBLEM OF RE-BLEEDING AFTER SURGICAL PROCEDURES
FOR PORTAL HYPERTENSION:

3 patients are presented below as case summaries to illustrate the problem of rebleeding after a surgical procedure in portal hypertension.

CASE NO.1

R.N. 25 year old female patient presented to the general surgical wards with haematemesis, epigastric pain and splenomegally. Diagnosis of portal hypertension was made and patient operated on as emergency to stop bleeding. Gastroesophagectomy was performed but the patient continued to bleed and also developed reflux symptoms. Splenectomy and splenorenal shunt were later performed (a month later) but this controlled haematemesis only for a short period, as the patient redeveloped haematemesis a year later.

CASE NO. 2

F.M. 12 year old male patient presented with haematemesis, malena stool, caput medusae and splenomegally. Diagnosis of portal hypertension was made on clinical and radiological examination. He had a splenorenal shunt performed but he redeveloped haematemesis the same month. The shunt was found to be thrombosed.

A Tanners portoazygous disconnection was then done to control bleeding, but the patient had a stormy recovery. He developed bilateral lung collapse post-operatively but later recovered and was discharged. However ten months later the patient was readmitted with bleeding oesophageal varices, necessitating a third operation. A mesocaval shunt was performed and the patient improved. Follow-up period however has been short - 4 months.

CASE NO. 3

W. A. 21 year old male patient presented with features of portal hypertension, (Haematemesis, malena stool and splenomegally). Diagnosis of portal hypertension was confirmed radiologically and the patient admitted for surgery. He had a splenorenal shunt but this underwent thrombosis, resulting in recurrent haematemesis. A mesocaval shunt was then performed but the patient still bled from the oesophageal varices and died from severe blood loss.

TABLE 13: SUMMARY OF THE SURGICAL PROCEDURES AND COMPLICATIONS:

SURGICAL PROCEDURE	NO. OF PATIENTS AND COMPLICATIONS		
		ENCEPHALOPATHY NO. OF PATIENTS	RECURRENT HAEMATEMESIS (NO. OF PATIENTS)
		<u>PERCENTAGE GIVEN IN BRACKETS</u>	
SPLENECTOMY	9	0	1 (11%)
STAPLING OF VARICES	3	0	3 (100%)
MESOCAVAL SHUNT	10	0	4 (40%)
PORTOCAVAL SHUNT	8	2 (25%)	3 (37%)
SPLENORENAL SHUNT	21	0	1 (5%)

As can be seen from table 13 encephalopathy was seen in only those patients who had portacaval shunt, while recurrent haematemesis was noted in all other surgical procedures, but with a higher percentage (100%) in those who had stapling of oesophageal varices. The number of patients studied however is small and should be interpreted with caution.

DISCUSSION

The results of this study showed that schistosomal portal fibrosis (occurring in 48% of the patients studied) and cirrhosis of the liver (21% of the patients studied) are the most important causes of portal hypertension in this country, although the irregular geographical distribution of schistosomiasis must be remembered. This compares well with studies done earlier by J.R. Miller and Bagshawe in 1970 in Nairobi.

In another tropical country, Zambia R.H. Caruthers and P. Sinha in 1971 found that 70% of the patients studied had schistosomal portal fibrosis where 20% had cirrhosis of the liver.

Portal vein thrombosis accounted for 9% of the patients, in contrast to what was reported much earlier on by J.R. Miller in 1967, where he noted that about 50% of the patients he studied had portal vein thrombosis. Indeed umbilical sepsis in infancy is sometimes followed by phlebitis of the umbilical vein which spreads to the portal vein to cause extrahepatic portal obstruction. Perhaps with better standards of maternity delivery services in this country, portal venous thrombosis resulting from umbilical sepsis has declined.

As noted in the review of literature alcohol is the most important cause of cirrhosis in the industrialised West (Lieber CS) and that its importance in the tropics as a cause of liver disease is difficult to assess. However it is significant from this study that of the 31 patients who offered a positive history of alcohol ingestion 25 (81%) of them suffered from cirrhosis of the liver. While it is true that not all alcoholics suffer from liver disease it is equally true that alcohol abuse is a neglected problem in this country and other developing countries of the world.

On the basis mainly of the liver function tests, stool examination, rectal snip and splenic venogram the various aetiological groups were identified. However there are those patients in whom the cause of portal hypertension could not be established. In these patients portal hypertension occurred in the absence of obvious intra or extrahepatic obstruction to portal venous blood flow. In this study this type of patients were grouped under the term "cause not established." This was preferred to "non-cirrhotic portal fibrosis," "idiopathic portal hypertension," "obliterative portal venopathy" terms which have been used in literature elsewhere (M. Decock); because it was felt that the causes of portal hypertension in some patients were probably missed using the available methods of diagnosis.

It is obvious that there is no simple answer to the problem of portal hypertension and bleeding oesophageal varices; and as stated earlier in the review of literature to operate or not to operate is many a time a dilemma in which a surgeon finds himself because of the poor results of medical treatment and serious long term complications associated with the operations which are effective in preventing recurrent haemorrhage. In this study 146 patients were treated medically although one hundred and eleven patients from this group qualified for surgery because of good hepatic reserve and patency of the portal system.

The results of medical treatment and surgical treatment in this study are difficult to compare as most patients were lost to follow up. It is however true that it was distressing to physicians attending to these patients, watch helplessly as some of the patients succumbed to exsanguinating haemorrhage from oesophageal varices.

The number of patients who underwent surgical procedures was small, making it rather difficult for meaningful discussion. It is however noted that the worst results in terms of recurrence of bleeding occurred in the operation of stapling of oesophageal varices. All the three patients who had this procedure done rebled from oesophageal varices.

Hepatic encephalopathy was only noted in those patients who had portacaval shunt. Indeed this procedure was initially favoured by surgeons because in most patients it is the easiest to construct from the technical viewpoint. Unfortunately it does not give long term results because of a much higher incidence of postshunt encephalopathy and a higher early and late mortality rate from liver failure. Although this shunt decompresses the portal venous system more effectively than any other shunt, it does reduce markedly the blood supply to the liver. 2 patients who had portacaval shunt rebled, but it was not established whether this was from oesophageal varices or from a different site.

It does appear from the results (although this should be interpreted with caution because of the small number of patients) that the splenorenal shunt end to side as advocated by Linton (reviewed by Ottinger et al 1982) and distal as advocated by Warren, had the best results as complications in these patients were fewer. It is believed that the main reason that the splenorenal shunt has fewer postshunt complications is that it is constructed with a small anastomosis so that it does not shunt all the portal venous blood as does a direct portacaval anastomosis, yet at the same time, a sufficient amount is released to decompress the portal bed.

Splenectomy alone which decompresses the portal venous system by as much as 20%, was beneficial to all the 9 patients who had it except one of them who later bled to death from oesophageal varices.

In this study the average age of the patients was 26 years. In the study by Caruthers and Sinha, the average age of the patients was 32 years. In the industrialized West however most of the studies have been done on older patients, averaging 50 years and more. The relative youth of many of the patients in developing countries is favourable, as they may withstand better major surgical procedures than older patients. There is also good reason to expect better results from surgery for portal hypertension due to schistosomal fibrosis than from portal hypertension due to cirrhosis of the liver, as there is relatively little liver cell damage, so liver failure is not a serious problem, much to the advantage of the surgeon. In the light of this knowledge, a controlled, prospective study on surgery for portal hypertension in this country is called for.

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APPENDIX

- 1. Name.....
- Unit No.....Age.....Sex.....
- Tribe.....District.....
- Present residence.....
- Day of admission.....Duration of stay.....

2. Presentation

- | (a) Main symptoms | (b) Physical signs |
|-----------------------------------------------------------------------|--------------------|
| 1. | 1. |
| 2. | 2. |
| 3. | 3. |
| 4. | 4. |
| 5. | 5. |
| 6. Significant past medical history e.g. history of alcohol ingestion | |

- 3. Clinical diagnosis.....
- 4. Any other illness e.g. Peptic Ulcer
-
-

5. Investigations

- (a) Laboratory findings:
 - Hb..... - A/K. Phosphatase.....
 - PTI..... - SGOT.....
 - Albumin..... - BUN/Urea.....
 - Total protein..... - Plateletes.....
 - Bilirubrin..... - Occult blood.....
 - Rectal snip.....
 - Stool.....

Coagulation screen

- Bleeding time.....
- Prothrombin time test.....
- Prothrombin time Control.....
- Thrombin time test.....
- Thrombin time Control.....
- KCCT test.....
- KCCT Control.....
- Ba swallow.....
- Endoscopy.....
- Splenic portovenography and any other investigations
.....

6. Treatment

- a) Medical.....
.....
.....
- b) Surgical:
Operative findings and procedure done.....
.....
.....

- 7. Complications at surgery. Post op and late follow up complications.
.....
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.....
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.....
.....

8. Histology report .

Liver.....
.....
Spleen.....
.....

9. Comments