GASTRIC LESIONS IN PORTAL HYPERTENSION IN BILHARZIAL HEPATIC FIBROSIS

THESIS

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BY

MAGDY ABD EL-RAZEK HASAN
M.B., B.Ch.

Under The Supervision Of :

Prof. Dr.
MOHAMED ABO EL-ENIN
Prof. of Internal Medicine,
Benha Faculty of Medicine.

Dr.
IKRAM AL ASSIOUTY
Assist. Prof. of Internal Medicine,
Benha Faculty of Medicine.

Dr. MOHAMED ABD EL-WAHAB
Lecturer of General Surgery,
Faculty of Medicine,
Mansoura University.

BENHA FACULTY OF MEDICINE
ZAGAZIG UNIVERSITY
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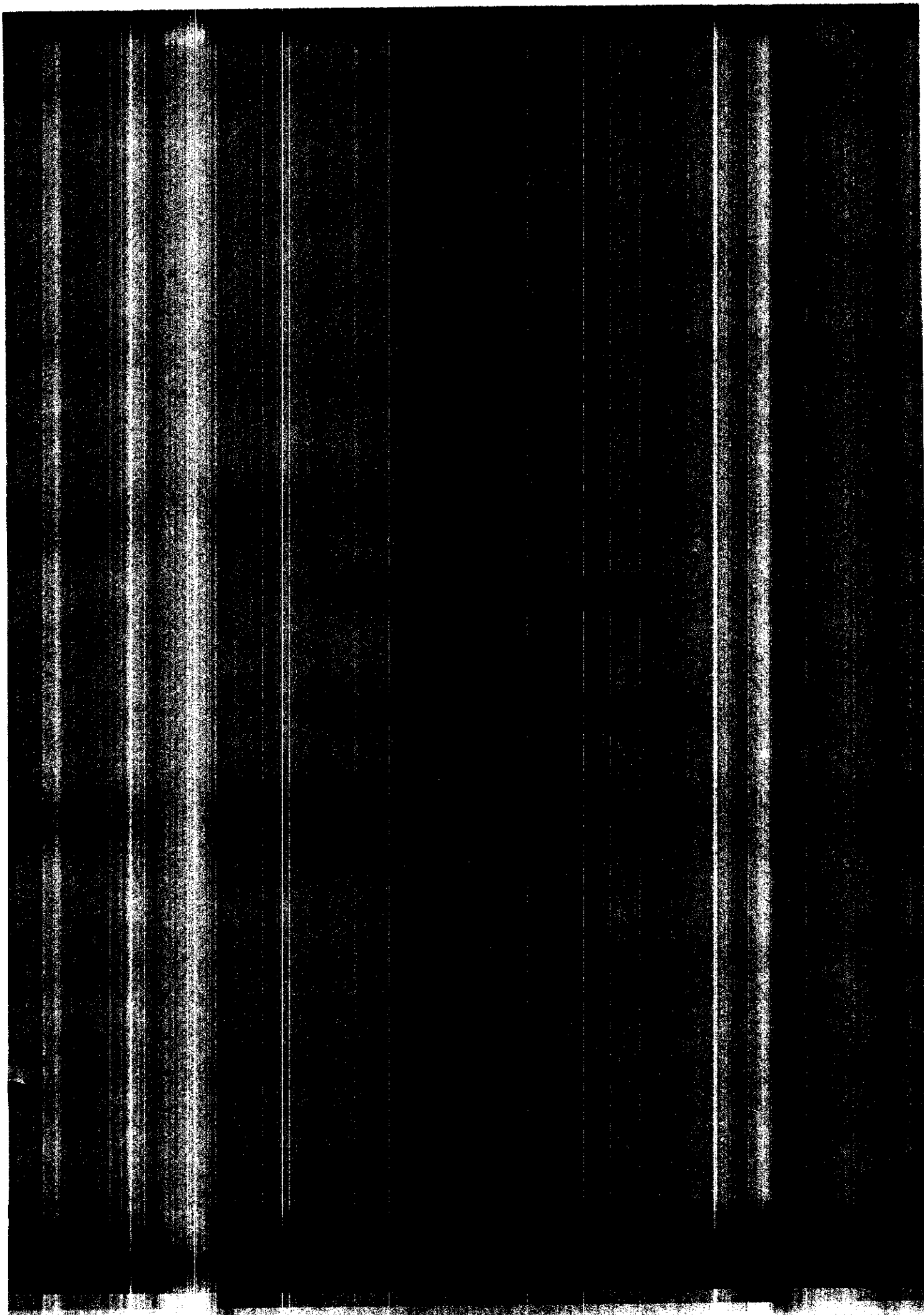


Introduction:

Portal hypertension in Bilharzial hepatic fibrosis is a national problem which has to be ingentily solved. The use of scelerotherapy of bleeding oesophageal varices, combined with regular endoscopic follow up has provided a unique opportunity to study the progression of changes occurring in the gasteric mucosa (Mc Cormack et al.,1985).

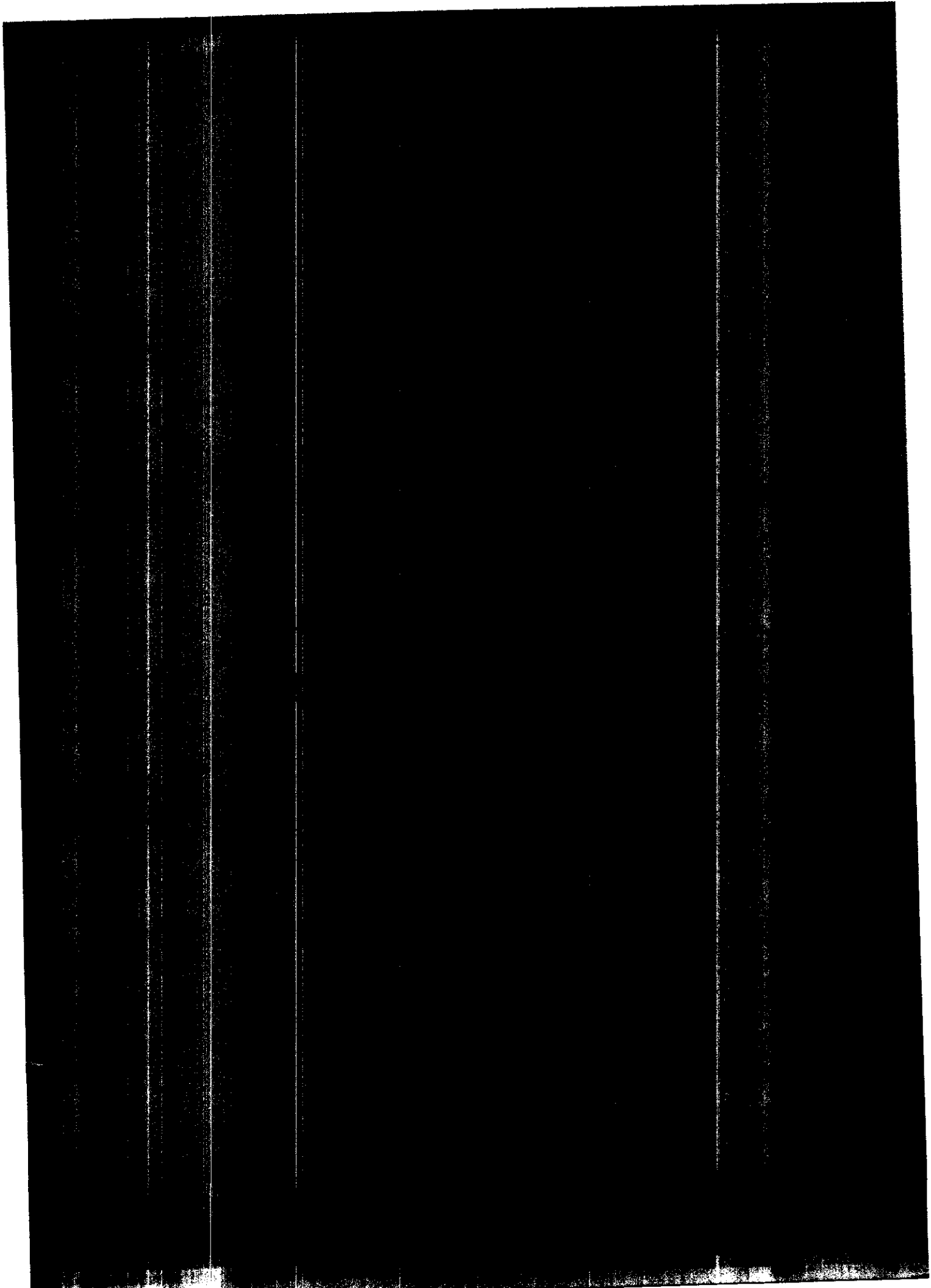
It was concluded that hemorrhagic gastritis in patients with varices should be viewed as portal hypertension bleeding and that the basis for therapy must be the early institution of measures which effect a reduction of portal pressure (Sarfeh et al., 1982).

The severity of hemorrhagic gastritis depends up on the prescence of gastroesophageal varices and hence portal hypertension (Sarfeh et al., 1984).

There are many causes of high incidence of gastric mucosal lesion in cirrhotic patients as gastric acid hypersecretion, damage to gastric mucosal Barrier & upper gastrointestinal bleeding often occur, from gastric mucosal lesion insted of rupture of esophageal varices (Sato et al., 1985).

Aim of the Work :

A trial to identify the gastric lesions in bilharzial hepatic fibrosis either inflammatory gastritis or congestive gastropathy and the gastric mucosal changes after sclerotherapy, using endoscopy and histopathological study.



BILHARZIASIS OF THE UPPER G.I.T.

Bilharziasis of the oesophagus and stomach occuring as a primary affection due to oviposition in these sites, are rare the reason for this is not clear, conversely involvement of the oesophagus and stomach in bilharzial hepatic fibrosis is quite common (El Roby, 1973).

The effects of portal hypertension on the upper gastro-intestinal tract frequently causes dearrangement of structure and function. This usually takes the form of oesophago-gastric varices, gastritis, gastro-oesophageal reflux and oesophagitis have been described (El-Robey 1973). The earliest report of bilharzial ova in the stomach is probably that of May (1908).

Kadry and Hashem (1957) express their belief that gastric bilharziasis may not be so rare as used to be believed.

Kadry and Hashem (1957) reported a case of gastric bilharziasis of nodular fibrotic type.

This case was fresh when examined 2 h. after death, from a severe attack of haematemesis. The mucosa was oedematous and congested with scattered pale, firm, slightly elevated nodules, and with 3 superficial peptic ulcers near the pyloric end.

Khairy et al. (1967) studied gastric biopsies from pyloric canal in 34 cases of bilharzial hepatic fibrosis with and without varices ova were found in 5 cases with oesophageal varices only. They concluded that bilharziasis of stomach and oesophagus was mainly due to distrubed haemodynamics.

El-Sharkawy et al., (1969) stated that patients with bleeding varices, tended to have a normal gastric mucosa, a high acid peptic activity and many of them had gastro-oesophageal reflux, and oesophagitis. Non bleeders on the other hand had atrophic gastric mucosa and depressed peptic activity.

El-Sharkawy et al. (1969) studied the oesophagus and stomach of 90 patients with bilharzial hepatic

fibrosis of various degrees. They recorded bilharzial ova in 3 cases of with minimal reaction around.

Ata and Abdllatif (1971) stated that when there is portal hypertension and in the presence of collaterals. It will be easy to find bilharzial ova in the stomach.

El-Sharkawy et al. (1977) stated that in the superficial part of the gastric mucosa, many bilharzial ova could be seen scattered either single or in groups.

Hunter and El-Rooby (1979) were studying the histopathologic findings of the stomach in cases of bilharzial colonic polyposis, they found that 3 cases showed bilharzial ova and granulation tissue in upper gastro-intestinal tract.

El-Fayomy et al. (1981) studied the histopathological picture in patients with bilharzial hepatic fibrosis and they found bilharzial ova in 10 % of cases. As in the case of oesophagus and

stomach, bilharziasis may affect the small intestine "including duodenum" either directly by oviposition, or indirectly through reflection of portal hypertension.

Gelfeind (1967) recorded an incidence of 15 % schistosoma mansoni ova in the small intestine.

Farag et al. (1980) studied endoscopically and histopathologically 180 patients having oesophageal varices with history of haematemesis they found duodenitis in 80 % of cases, bilharzial nature of duodenitis is proved in 18 %.

Farag et al., (1980) studied the endoscopic piccure in 30 cases of liver cirrhosis and portal hypertension. They found that there were changes in the gastric mucosa in 93.33 % in the form of chronic superficial gastritis, diagnosed endoscopically in 90 % among the 30 cases they also found oesophageal varices in 27 cases, 19 cases only were of bilharzial origin i.e. 67 %.

Farag et al. (1980) studied the endoscopic picture of 180 patients having oesophageal varices. They found that eight of them had chronic duodenal ulcer 4.4 %.

El-Fayomy et al. (1981) studied the endoscopic picture in patients with bilharzial hepatic fibrosis. They found oesophageal varices in 55 % reflux oesophagitis in 10 %, and normal picture in 35 %. They also found that there were prepyloric polyps in 15 %, gastritis in 10%, pale mucosa in 10 % and normal gastric mucosa in 65 %.

El-Fayomy et al. (1981), found active duodenal ulcer in 5 % of cases and deformed stenotic pyloric end which was impossoble in 5 %, and normal duodenal mucosa in 85 % .

Microscopic picture :

Kadry and Hashem (1957) studied the microscopic picture of gastric bilharziasis of nodular fibrotic type. They found thickening of the mucosa in some parts with nodular formation. There were many

bilharzial ova with bilharzial granulation tissue around. The mucous glands in these areas were degenerated where as in the neight bouring parts they were proliferated.

Hashem (1962) explained the variation in incidence among the variation organs on the basis of the richness of the part with venules. The more the venous supply in the part, the more will be the chance for the female worms to get in and lie ova.

Gefland (1967) studied the distribution of ova of S. mansoni and S. haematobium in gastro-intestinal tract in 41 cases. The following rates were given

Organ	Mansoni	Haematobium
Stomach	10 %	12 %
Small intestine	15 %	10 %
Appendix	12.5 %	37 %
Colon	58.5 %	56 %
Rectum	78 %	70 %
Liver	37 %	30 %

Sharkawy et al. (1969) studied the oesophagus and stomach of 90 patients with bilharzial hepatic fibrosis of various grades. Biopsies revealed bilharzial ova in 3 cases with minimal reaction around.

Souidan et al. (1971) showed diminution of basal gastric secretions in bilharzial hepatic fibrosis associated with hypoproteinaemia this was attributed to devitalization or atrophy of the gastric glands. The oxyntic cells were found less than normal in some cases.

Ata et al. (1971) recognized bilharzial ova in the gastric mucosa with minimal round cell infiltration around them. In the colse neighbourhood of the ova the gastric glands were normal. This with eosinaphilic infiltration and abscence of evidences of superficial gastritis, but atrophic mucosa constitutes a pathological entity of bilharzial lesion of the antrum.

Ata and Abdel Latif (1971) found that gastric biopsy from the stomach of a case of bleeding varices with active intestinal bilharziasis, showed presence

of avery vascular mucosa, with many dilated vessels, distributed through out the whole mucosal depth.

El-Sharkawy et al. (1977) stated that in the superficial part of the gastric mucosa, many bilharzial ova could be seen scattered either single or in groups. Giant cell engulfing parts of the ova were occasionally seen, there in minimal cellular reaction around the ova, the neighbouring gastric glands were normal.

El-Fayomy et al. (1981) found that there is no mucosal histopathological changes in oesophagus in patients with bilharzial hepatic fibrosis. As regards stomach they found bilharzial ova in 10 % of cases, atrophic gastritis in 50 %, superficial gastritis in 25 %.

Khairy et al. (1967) suggested that term bilharzial antral gastritis. This term discribes a pathological entity consisting of atrophic mucosa, submucosal fibrosis, with oesinophilic infiltration,

together with chronic inflammatory cells, but lacking bilharzial ova.

Khairy et al. (1967) stated that the histopathologic picture of the mucosa of the body of the stomach in case of bilharzial gastritis is similar to the picture of chronic gastritis wheather superficial or atrophic. But the parasitic nature is proved by the presence of eosinophil in 63.6 % . The antral mucosa shows specific changes which is called diffuse submucosal fibrosis 82.6 %.

El-Roby (1966) studied the function and structure of small intestine of 50 bilharzial patients by biopsies taken from duodenum and jejenum only 3 cases retatined the normal mucosa, all the others presented combination of leaves, ridges, and convolutions but non of them showed flat mucosa.

El-Roby (1966) found bilharzial ova in the submucosa just beneath the muscularis mucosa, in the mucosa, the crypts of lieberkuhn, or in both,

They were usually surrounded by forigen body gaint cells and other constituents of bilharzial grnuloma. The cytology of the lamina propria often showed a noticable eosinophilia but the villi were apparently normal so were also the mucosal cells. Examination of biopsies by transparency techniques revealed bilharzial ova, some of them were viable, always of mansoni species.

Other findings were reduction of total mucosal thickness, blunting of villi with diminution of their absorptive surface, partial villous atrophy, reduction in adult cell, crypt cell ratio, heavy round cell infiltration of the lamina propria and thickening of the muscularis mucosa.

VENOUS ANATOMY OF THE LOWER OESOPHAGUS IN PORTAL HYPERTENSION

The cause of oesophageal variceal bleeding remains an enigma, although most workers accept that varices probably bleed by rupturing rather than by being eroded from the oesophageal lumen. Few delailed anatomical studies of the venous drainage of the lower oesophagus have been undertaken largely because of the lack of suitable techniques. The first major investigation was undertaken by Kegaries and published in 1934. He was the first to suggest that the veins in the oesophagus immediately obove the cardia differed from the remainder of the oesophagus. The most comprehensive study was the of Butler. He studied mainly human fetuses injected with indian ink and classified oesophageal veins into three groups. These were:

A. Intrinsic veins including subepithelial and submucosal and submucosal veins which join the gastric veins below, and perforating veins which pierce the muscular wall to join the extrinsic veins;

B. extrinsic veins formed by the union of groups of perforating veins which join the left gastric vein below and the systemic veins above and

C. The venace comitantes of the vagus nerves which run longitudinally in the adventitia of the oesophagus. This distribution of veins has been confirmed by others The studies of Spence and McCormack were published after the present study had been completed, but were taken into account in the interpretation of the data.

The venous anatomy of the lower oesophagus and upper stomach in man. It extended a previous preliminary in vestigation of a separate set of normal oesophageal casts which demonstrated some aspects of the vascular anatomy. It is not surprising that all oesophageal and gastric venous channels enlarge in patients withe portal hypertension. However, it is the deep intrinsic veins that become massively enlarged and develop into tortuous variceal channels. The resin casting technique demonstrated this clearly for the first time. The large deep intrinsic veins or varices have virtually no direct cross-connections but link with the superficial plexus of veins which have a rich intercommunication. Varices are therefore interconnected but mainly via an indirect route The meshwork of the superficial venous plexus in normal patients is replaced by a more longitudinal arrangement of veins in patients with varices. This could indicate that they are acting as collateral channels.

On the other hand minor variceal bleeds that stop spontaneously may occur from a branch of the superficial venous plexus without a close connection to a large varix or even possibly from dilated intra-epithelial channels. Spence et al.,(1984) have suggested that the intra-epithelial channels that they demonstrated histologically may represent the endoscopic(1983) findings of cherry-red spots and red wate markings of the Japanese group and the varices upon varices noted by others. In our view these endoscopic findings are more likely to represent the subepithelial bloodfilled channels which (Spence et al.), demonstrated to erode into the epithelium.

McCormack and colleagues have demonstrated bidirectional flow in varices in the lower oesophagus and have attributed this to perforating veins between the submucosal (intrinsic) and adventitial vessels. These perforators were first demonstrated by Butler and have been clearly confirmed by the studies of McCormack et al. (1983).

VASCULAR SYSTEM

EXTRAHEPATIC VESSELS

The extrahepatic portion of the hepatic vessels has been studied extensively on radiological and anatomical basis. It is of interest in this review to discus the important facts concerning the portal and hepatic venous systems.

1. The portal vein:

The portal vein is formed by the confluence of the mesenteric and splenic veins behind the neck of pancreas. The portal trunk is about 5.5 to 8 cm. Long and about 2 cm in diameter. It receives the rootlets of the superior pancreaticoduodenal vein, some accessory pancreatic veins, the pyloric vein, the left gastric and the cystic vein. The upper Segment " 5 cm " is usually devoid of major trib utaries. The left gastric vein runs upwards along the lesser curvature of the stomach where it receives some esophageal veins.

The portal vein divides before entering the portal fissure into two lobar branches. The right branch is short and thick. The left branch is Longer and smaller and is joined by the umbilical

vein and the associated para-umbilical veins. There is no anastomosis between the macroscopic branches, but aboundant intercommunications exist at the sinusoidal Level.(Bilbey, 1960)

2. The splenic vein:

The splenic vein is formed by the confluence of two to six major trunks with an average of three use of a splenic tributary rathar than of the splenic vein should there fore be alternative in performing a splenorenal anastomosis(Dougloss et al., 1950). The point of confluence of the splenic tributaries to form the splenic trunk varies considerably. It is commonly between the hilum and the tail of the pancreas, about 3.4 cm from the midpoint of the hilum. However, con fluence may occure behind the pancreas. In 7.7% of eases, the superior polar vein is excessively long and joins the splenic trunk relatively distant from the hilum and proximal to the point of confluence of the other splenic tributaries(Garber et al., 1951).

The splenic vein regularly lies in adistinct groove on the posterior surface of the pancreas below the level of the splenic artery in 75% of cases and

sometimes may actually by completely embedded in pancreatic tissue? Draining into it are an average of seven small pancreatic veins.

The splenic vein receives the left gastroepiploic, short gastric, small pancreatic and the inferior mesenteric veins. The Left gastroepiploic terminates either in the trunk of the Splenic vein (83%) or in the lower splenic tributaries (17%)(Douglosbe et al,,1950)

The majority of the short gastric veins regularly enter the spleen directly, thus anastomosing with the splenic veins within the spleen. The short gastric veins usually enter the upper part of the hilum or the upper pole of the spleen, and in about 27% They enter both the upper and Lower poles, However, it is not unusual to find one or more short gastric veins emptying directly into asplenic tributary of the splenic trunk.

The inferior mesenteric vein joins the splenic vein approximately 3.4 cm distal to the site of formation of the portal vein in 38%. In 32.7% it joins the angle of junction between superior mesenteric and splenic veins. It may join as well the superior mesenteric vein directly. in 29.3% of cases.

3. The Hepatic veins:

The hepatic veins empty into the inferior vena cava. There are three major hepatic veins, right, middle and Left. The middle vein Sometimes unites with the left hepatic to form ashort common trunk. These three hepatic veins drain the three hepatic venous Segments, which interdigitate and overlap the posterior Segments(Rappocport, 1980).

PORTAL HYPERTENSION

Portal hypertension refers strictly to an increase in the portal venous pressure (> 5 mmHg), but clinically the term is used to refer to the syndrome associated with an increased portal venous pressure which is characterised by splenomegaly and the development of abnormal portalsystemic venous anastomoses. Increased resistance to blood flow in the portal venous system is the most important cause, though increased portal blood flow may contribute in a few cases. The causes of increased portal venous resistance may lie in the presinusoidal vessels outside the liver or in the intrahepatic vessels at presinusoidal, sinusoidal or postsinusoidal levels. (Sherlock 1978).

Causes of portal hypertension:

I. Suprahepatic as long-standing right ventricular failure, constrictive pericarditis, pericardial effusion, tricuspid valve disease, and high inferior vena caval thrombosis.

II. Intraphepatic causes: These are the commonest causes of portal hypertension. Obstruction may be postsinusoidal sinusoidal or presinusoidal.

A. Postsinusoidal obstruction due to Budd-Chiari syndrome or veno-occlusive disease.

B. Sinusoidal obstruction occurs in portal cirrhosis This causes hypertension through:

1. Mechanical obstruction of portal blood flow by fibrosis.

2. Pressure of regenerating nodules on smaller hepatic radicles of portal vein, and

3. Transmission of hepatic arterial pressure to portal venous pressure due to opening of arterio-portal venous anastomoses.

C. Presinusoidal obstruction of the portal vessels: Intrahepatic obstruction of the terminal branches of the portal vein occurs in bilharziasis and congenital hepatic fibrosis.

III. Infrahepatic portal hypertension may result from:

A. Portal vein obstruction, e.g.:

1. Portal vein thrombosis, the commonest cause is liver cirrhosis. Also it may occur after splenectomy due to rebound increase in blood platelets, and after portacaval anastomosis.

Pyelophlebitis or septic thrombosis of the portal vein may complicate umbilical sepsis, acute appendicitis, cancer colon, infected piles, etc. Rare causes include invasion of the portal vein by malignant tumours, polycythaemia vera, and dehydrating diseases.

2. Lesions in the wall of the portal vein as angiomatous malformation.

3. Compression of the portal vein from outside by tumours- as cancer head of pancrease, enlorged lymph nodes.

COLLATERAL CIRCULATION

Anatomical Pattern :

The anatomical sites of portosystemic communication are an important factor regarding the hemodynamics of portal hypertension before and after surgical mangement. Pre-surgically , there are five main locations :

1- Oesophageal anastomosis between the left gastric vein and the azygos and hemiazygos veins.

2- Through pancreatic veins from the splenic to the renal vein.

3- Connections between duodenal , pancreatic , and colic veins to perirenal veins that drain into the renal and lumbar veins. These veins are situated on the left side in the splenorenocolic area and on the right side in the pancreaticoduodenorenal area .

4- Veins of Retzius connecting the colic veins to lumber veins at the ascending and descending colon.

5. The rectal plexus joining the superior rectal vein to middle and inferior rectal veins. However , esophageal anastomosis is the most freqently occuring occuring collateral. It is clinically important because of the development of submucosal gastric and esophageal varices that may rupture and lead to massive hemorrhage (Salam, A.A. and Warren, W.D., 1974).

The standard distal splenorenal shunt creates a pressure gradient between two systems in one coelemic cavity. Therefore, with long term follow up all of the patients develop a unique pattern of collateral venous pathways between the high pressure portomesenteric compartement (25 - 35 mmHg) and the low pressure gastroeplenic area (5 - 10 mmHg). These distinguishing portaprival collateral tcutes after DSRS are the transpancreatic, the gastrosplenic, and the colosplenic (Maillcard, J.N. et al., 1979; Hutson, E.E. et al., 1982).

The development of these pathways follows the potential anatomic connections between the two compartments

which enlarge over time to provide signidicant collateral drainge routes. These collaterals originate at the junction of the superior mesenteric and portal vein over a 4 - 6 cm. distance, often from the right side of the vessele. The final common pathway of these routes is into the splenic vein through its pancreatic segment" pancreatic stump", short gastric vessels "gastric collaterals" and directly to the inferior ° ramus colosplenic collaterals" (Henderson, J.M. et al., 1985).

Quantitative Estimation :

Most studies that have measured portosystemic collateral flow utilized the injection of an indicator into the splenic pulp and calculated the proportion of splenic injectate that appears in the hepatic vein. This technique can provide information only about splenic collateral flow , however, retention of indicator at the site of injection complicates the calculation. Selective percutaneous catheterization of the portal system allows separate angiographic or isotopic evaluation of collateral flow originating from the splenic and mesenteric portal inflow (Eber, F.L. et al., 1960 ; Schwortz, S.I. and Greenlaw, R.W., 1961).

The crucial clinical importance of gastroesophageal collaterals compelled Groszman and his associates to undertake the measurement of flow in this area. Since blood flow through the coronary "gastroesophageal" collaterals drains into the azygos venous system, they postulated that measurement of the azygos venous blood flow in portal hypertensive patients would at least allow an estimation of gastroesophageal collateral blood flow. They assumed that at rest , blood flow from the structures normally draining into the azygos venous system represents a relatively low fixed fraction of the azygos venous flow in these patients. Using a thermal dilutiontechnique , they in fact found a close relationship between portal pressure and azygos flow (Britton, R.C., 1963).

In patients with prominent cephalad collaterals and suggested that measurement of azygos blood flow could be a sensitive method in assessing the hemodynamics after selective decompression of the gastro-esophageal varices , e.g. distal splenorenal and coronocaval shunts (Bosch, J. and Eroszmocnn, R.J.,1984).

BILHARZIAL HEPATIC FIBROSIS AND RUPTURED OESOPHAGOGASTRIC VARICES

Oesophageal varices are frequent and dangerous sequalae of bilharzial hepatic fibrosis with portal hypertension normally the blood is carried through the splenoportal veins and is not forced into the other tributaries of the portal system, unless there is an increase of pressure. In portal hypertension there is usually a backflow along the tributaries of the portal vein, with filling and distentention of the gastric and oesophageal vein. In bilharzial hepatic fibrosis, the granulomatous portal tract with the effect of fibrosis on the walls of the portal ramifications is the cause of the raised portal tension . Its sustainment and flactuations open up porto-systemic collaterals and congest the spleen. Oesophageal submucous varices develop. Dilated veins in the distal few centimeters of the oesophagus, the main veins, and consequently the varices run right underneath the epithelium, (Stelzner and Lierse, 1968).

Gastric varices occur in portal hypertension just as frequently as do oesophageal varices , (Yasumoto, 1971). Most of gastric varices are located in the cardia and fundus because of the predilection of collaterals but sometimes they are found distal to the fundus. Roentgenologically, gastric varices produce a characteristic polyoid type of multiple filling defects, much like a bunch of graps or a group of bubbles projecting into the lumen of the stomach (Evans and Delany, 1953).

Computed Transaxial Tomography :

Gastric varices are known to minic polypoidal intra luminal masses on upper gastro-ingestinal barium examination despite their intramural and subserosal origin although the effects of portal hypertension have been well demonstrated by C.T. of the abdomen-gastric submucosal and subserosal varices can appear as intra luminal masses on C.T. images (Baer-J.W ; Smorzaniu , K.E. , 1985).

Incidence of Oesophageal Varices in Bilharzial Hepatic Fibrosis :

Hepatic bilharziasis is mainly a vascular affection. It has been estimated that more than two thirds of patients get portal hypertension and may get haemorrhage from varices (Child, 1954 and Rodriguez et al., 1955). In bilharzial liver fibrosis, the incidence of oesophageal varices by x-ray was found to be 21.6% by Arafa et al. (1957) and Khalil and Fadali (1962) in 100 cases studied with barium swallow, oesophagoscopy and venography found an incidence of 77%.

Kamel et al. (1965) found that varices occured in 52% when searched for, by oesophagoscopy and oesophagoradiography.

Sabour and Salib (1966) found that oesophagoscopy demonstrated the presence of varices in 66 out of 129 patients with hepatic schistosomiasis, while oesophagoradioscopy detected varices in 28 patients.

El-Hawey (1978) found that oesophageal varices were present in 50% of patients with shrunken liver, huge splenomegaly and ascites, and 33.3% of patients with early hepatomegally.

Degree of Varicosity :

The "severity" or diameter of the venous channels is more difficult to evaluate, because diameter varies from point to point along the cause of a varix and because varices of different sizes are often found.

A simple ruler was fashioned for precise transesophagoscopic measurement of varix diameter, but it has been found simple to make accurate visual estimations (Palmer, 1953 and Palmer and Brick, 1956).

Three categories are classified by Palmer, (1953).

Mild : Less than three millimeters.

Moderate : Three to six millimeters.

Severe : In the oesophageal segment most severely involved, most of the varices measure six or more millimeters in diameter.

Criteria for grading the severity of the esophageal varices were as follow :-

0 = no definite varices visible.

1+ = one or more varices under 4 mm in diameter, and under 4 cm in extent.

2+ = multiple varices, 4 to 10 cm in extent.

3+ = multiple varices over 10 cm in extent.

(Baker et al., 1959).

Willoughby et al. (1964) measured oesophageal varices directly with a 5 cm. ruler inserted through the esophagoscope. However, with experience visual estimation proved quite accurate. There was a variability in size of the oesophageal veins, but the range of size of the veins fell into 3 groups; veins 0.5 to 1 mm. in diameter were classified as small, those between 1 and 3 mm. in diameter as moderate, and those over 3 mm. as large.

Extent of Esophagus Affected :-

The extent was categorized as involvement of 1/6 , 1/4, 1/3, 1/2, 2/3 and 100 per cent of the esphagus. This was measured from the length of the esophagoscope which protruted beyond the alveolar ridge at the point, the first was encountered (Palmer, 1953 and Palmer and Brick, 1956).

Criteria for grading the severity of the oesophageal varices (Baker et al., 1959).

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Pathogenesis of Haemorrhage from Oesophageal Varices :

Preble : in 1900, found that any sudden increase in obstruction to the return flow of blood would casue rupture of the varix. The haemorrhage was preceded by

sudden extreme muscular exertion and in others by thrombosis of one or more of the main portal veins. Many cases remained in which the haemorrhage appeared to have been spontaneous.

Snell (1931) found that bleeding began in many patients when they were asleep ; a fact that may be explained on the basis of venous stasis and congestion.

Catch (1950) suggested that dilation of oesophageal veins was not opposed by any pressure in the thorax comparable to intra-abdominal pressure and though that this accounted for the tendency of such varices to bleed.

Compression of the abdominal contents by the abdominal musculature takes place during the act of vomiting, finally, the stomamch itself actively contracts. These changes are sufficient to cause an increase in pressure in oesophageal varices.

Other workers have indicated that erosion by gastric acids considered as important a factor in haemorrhage from varices as the hydrostatic factor. Longmire (1952) suggested that ulceration produced by regurgitated gastric juice is the most important factor.

Beswick and Butler (1951) emphasized two main factors in bleeding from esophageal varices. (1) overdistention of the exposed thin-walled veins in the lamina propria and the submucosa because of great pressure in the portal vein and (2) the fact that many of the dilated thin-walled veins are seperated from the lumen of the esophagus only by the epithelium; the latter is 0.2 mm. in thickness , which may be reduced by stretching caused by underlying varices. Erosion or ulceration of the epithelium over the varices produced by trauma from particles of food.

Baronofsky and Wangesteen (1949) have shown that impairment of the nutrition of the mucous membrane of the stomach, duodenum and esophagus

because of venous pooling lowers the resistance of these regions, which become an easy to prey to the gastric digestive juices.

Chiles et al. (1953) found that once a varix ruptures spontaneously and bleeds, the tear may be the starting point for an ulcer. The anemia that occurs after the haemorrhage, the lowered resistance to infection and the action of gastric juice all may lead to the rapid development of esophageal ulcers. Increased hydrostatic pressure within the varix may result in impaired circulation and devitalization of the surface epithelium. The latter is then less resistant to regurgitated gastric juice or to trauma from particles of food; the complicating factor of ulceration then develop, which in turn causes further destruction of esophageal tissue and the wall of the varix.

So, considerable attention has been given to other factors in the pathogenesis of haemorrhage from esophageal varices. Acid regurgitation, atrophy

of the mucosa, perivascular tissue support, peptic ulceration and esophagitis have all been given consideration in this connection.

The oesophageal varices are a serious complication of portal hypertension; in liver cirrhosis. Hematemesis occurs in approximately 25 per cent. The bleeding in cirrhosis arises from varicose oesophageal veins in approximately 30 per cent of the cases (Bockus, 1944). The problem as to why oesophageal veins bleed has been investigated by clinical, pathologic and experimental methods. Some clinical observations suggest that hydrostatic pressure is the important factor.

During the past 40 years two main theories of the pathogenesis of rupture have arisen, namely: "erosion from without", that is, rupture due to overlying oesophagitis eroding the mucosa and 'explosion from within, that is, rupture due to a sudden rise in portal pressure.

The early evidence for the oesophagitis hypothesis arose from animal experiments and autopsy studies. However, more recent evidence from oesophageal function studies (Heil et al., 1980) and histological examination (Spence et al., 1983a) has indicated that "erosion from without" is not a major factor.

Although portal hypertension is essential for the development of varices, raised hydrostatic pressure alone is not the sole factor. Evidence for a direct relationship between bleeding and pressure is conflicting, depending on the method of measuring portal pressure. Over 30 years age Palmer (1953) measured pressure by direct needle puncture at oesophagoscopy and found no corredlation between portal pressure and the severity of varices. Using wedged hepatic pressure measurements the King's College Hospital, London group found a positive correlation between the height of pressure and the frequency of subsequent rebleeding.

However, French workers measuring wedged hepa-

tic venous pressure found no significant relationship between the risk of gastrointestinal bleeding and the degree of portal hypertension. There is some evidence that there is an increased risk of rebleeding from varices in those patients with larger varices (Lebrec et al., 1980) although there have been conflioting reports.

Although varices may develop throughout the entire length of the oesophagus, many experienced endoscopists have noted that varices usually bleed in the distal few contimetres of the oesophagus. We believe that the difference in the venous anatomy between the distal and more proximal oesophagus may account for the prevalence of bleeding in the lower oesophagus. Using a computerised image analysis system to study the venous anatomy of the stomach and oesophagus in both normal subjects and variceal specimens, an abrupt change in the venous pattern at the oesophagogastric junction is noted (Spence, 1985). In normal subjects the veins of the lower 3-5 cm of the oesophagus lie chiefly in the

lamina propria, that is, between the muscularis mucosa and the basement membrane of the epithelium. In the stomach and more proximal oesophagus the veins lie mainly in the submucosa. There is a sevenfold increase in the area occupied by veins in the lamina propria of the lower oesophagus compared to the corresponding area of the stomach. A similar pattern was found in the variceal specimens. In portal hypertension it is the vessels in the lamina propria of the lowest 3-5 cm of the oesophagus which become varicose, while in the stomach and more proximal oesophagus it is mainly the veius in the submucosa which become dilated. The reason for this arrangement may be related to function of the physiological lower oesophageal sphincter mechanism. Since the veins in this area are closer to the oesophageal lumen they are probaply at most risk of rupture.

Histological studies of oesophageal transection rings have revealed dilated intraeptheliol blood - filled channels which on serial sectioning communicate with these larnge veins in the lamina propria via the

epithelial papillae. These channels frequently separated from the oesophageal lumen by only a few cells and their rupture perhaps precipitated by a sudden rise in pressure such as occurs during a Muller manoeuvre may be the initiating event in variceal haemorrhage (Spence et al., 1983 b).

The Japanese workers have described small vessels on the surface of varices endoscopy "varices upon varices". They have classified these vascular markings in cherry red spots, red whole markings, haematocystic spots and diffuse redness (Inokochi, 1980). It has been suggested that the presence of these cherry red spots, among other features, may correlate with imminent variceal haemorrhage. It is likely the intraepithelial channels seen in the transection rings correspond to the cherry red spots viewed at endoscopy and may be the actual source of haemorrhage.

HAEMORRHAGE FROM ACUTE GASTRIC EROSIONS

(Haemorrhagic gastritis) in Patients with Portal Hypertension :

Haemorrhagic gastritis has recently received attention as an important source of bleeding in patients with portal hypertension Lebrec et al., (1980).

One our Logically ask : Does portal Hypertension predispose to haemorrhagic gastritis and if so by what mechanism ? secondly is bleeding from haemorrhagic gastritis more sever in patients with portal hypertension ?

Does Portal Hypertension Predispose to Haemorrhagic Gastritis ?

A small body of experimental evidence supports such a conclusion wangensteen (1945) produced gastric venous hypertension in dogs by gastroepiploic or portal vein ligation and stimulated acid production

in half of them with histamine. Sham-operated dogs and dogs not stimulated with histamine developed no lesions. Where as the histamine-stimulated dogs developed oesophageal and gastric erosions and duodenal ulcers. More recently.

Sarfeh et al., (1983) demonstrated that rats with portal hypertension produced by partial portal vein ligation had significantly higher gastric luminal pH. increased H^+ back-diffusion, lower mucosal potential difference and increased submucosal oedema compared to sham-operated control rats. Moreover these mucosal abnormalities were markedly enhenced by topical ethanol. The data of these two studies suggest that portal hypertension facilitates gastric mucosal injury by increasing mucosal permeability and acid back diffusion. Effects that are similar to and potentiated by those of ethanol. Clinical data on whether portal hypertension predispose to development of haemorrhagic gastritis are less clear-cut. Flexible fibreoptic

endoscopy has nct demonstrated a difference in the frequency of haemorrhagic gastritis in patients with upper gastrointestinal bleeding with and without portal hypertension. These studies probably did not under diagnose haemorrhagic gastritis in those patients with portal hypertension since the figure of 38 % agrees closely with the complied frequency of haemorrhagic gastritis in surveys of bleeding patients with portal hypertension : 52/154 or 30 % (Mc Cray et al., 1969 : Bonanno et al., 1972 : Mobarhan et al., 1972 : Waldram et al., 1974 : Teres et al., 1976 ; Franco et al., 1977).

Recurrent bleeding was virtually eleminated by the operation. Suggestion that portal hypertension can cause haemorrhagic gastritis and that relieving portal hypertension can relieve it. Our own controlled trial of portacaval shunt for variceal bleeding produced similar results (Reynolds et al., 1981). Despite continued alcoholism in about two-third of shunted patients the incidence

of gastrointestinal haemorrhage was markedly reduced. There is evidence to suggest that haemorrhagic gastritis is more common in certain subgroups of patients with portal hypertension than others. Terés et al., (1976) recognized atendency for the diagnosis of haemorrhagic gastritis to be made more frequently in patients with advanced liver disease. Franco et al., (1977) extended these observations. Reporting that haemorrhagic gastritis was significantly more common in patients with either sever liver failure-defined as the presence of weight loss, ascitis, hyperbilirubinaemia ($>$ 68 u mol/L). Hypoalbuminaemia ($<$30 g/L) and poor prothrombin time or sever stress defined as the occurrence of respiratory or renal failure or bacterial sepesis. An unusual feature of this study was the relatively high frequency of prior exposure to non-steroidal anti-inflammatory agents (40 % of patients with haemorrhagic gastritis). Stress is accepted as a cause of haemorrhagic gastric (Skillman and Silen, 1976) and it is plausible that

patients with portal hypertension are as prone or more prone to this complication than patients without liver disease. The reason that patients with liver failure may be predisposed to develop haemorrhagic gastritis is less clear; perhaps sever impairment of liver function constitutes a form of stress Lebrec et al. (1980) have recently reported that haemorrhagic gastritis is more frequent in patients with large varices than patients with small varices. Standard Liver tests were not reported the reason for their observation is not known. But it may be related to more sever liver disease or to an increased volume of collateral blood flow in patients with large varices.

Experimental and clinical evidence support the concept that portal hypertension predisposes to development of haemorrhagic gastritis. Patients with advanced liver disease. Sever stress and large varices may be particularly predisposed to this complication.

Is Haemorrhagic Gastritis More Sever in Patients With Portal Hypertension ?

Several reports have attempted to ansower this question. Khodadoost et al., (1971) found that the mortality of cirrhotic patients bleeding from gastritis was one - quarter that of patients bleeding from varices.

Similarly Mobarhan et al., (1972) reported that patients with cirrhosis bleeding from varices did so for a mean of 4.8 days with a mortality of 53 % compared to 2.4 % days and 9 % for cirrhotic patients with haemorrhagic gastritis. Recently however Sarfeh et al., (1981) concluded on the basis of a retrospective review that portal hypertension conferred additional risk in patients bleeding from gastritis. Mean blood loss was similar in these patients to that of patients bleeding from varices and markedly higher than that of patients with haemorrhagic gastritis alone. Moreover, mortality was much higher in the former two groups compared to the latter (51 % and 35 % versus 0 %). Standard liver tests were not

mentioned in these communications but it is reasonable to believe that patients with advanced liver disease would experience a higher mortality from any bleeding lesion than patients with no liver disease.

The most important element causing gastritis may be the raised portal pressure itself. Obstruction of the venous drainage from the stomach can induce changes in the gastric mucosa (Palmer, in 1957) induced portal hypertension in dogs by portal vein ligation and found that both the mucosal and submucosal veins in the stomach wall became dilated. Alternatively gastritis might be because of gastric mucosal ischaemia secondary to arteriovenous shunting which can be demonstrated in the stomach of both animals (Manabe et al. 1978) and humans (Hashizume, et al., 1983) with portal hypertension.

The histological changes are entirely consistent with an increase in venous pressure producing a congested gastric mucosa. The occurrance and severity

of this congestive gastropathy may depend, however, not only on the total level of portal pressure but also on local blood flow characteristics which may or may not transmit this increased pressure to the gastric mucosal and submucosal veins. Differences in local blood flow patterns may explain why some patients develop gastropathy and other do not. The greater incidence of the gastric lesion in the longer survivors may be related either to the progression of disease or to the greater number of sclerotherapy treatments in these patients. Our experience suggests that while gastropathy may develop after sclerotherapy in some individuals the converse is true in others. In theory obstruction of blood flow at the gastro-oesophageal junction may increase congestion by blood vessels flowing from the stomach. This effect is likely to be very variable as recent studies using Doppler ultra sound (Mc Cormack et al 1983) have shown that blood flow in oesophageal varices may sometimes be towards the stomach and therefore thrombosis of these varices

would reduce and certainly not induce congestion in the gastric mucosa. In the majority of patients, therefore, the relationship between sclerotherapy and gastropathy is not straight forward and the presence of gastric lesion is probably independent of the patency of oesophageal varices.

Terblanche et al (1979) have described the disappearance of gastric varices after sclerosis of distal oesophageal varices, presumably as a result of retrograde propagation of thrombus, but in the present series there were 2 patients in whom oesophageal varices were satisfactorily oblitrated who were subsequently admitted to hospital with bleeding from gastric varices. In both patients gastric varices were present before sclerotherapy, and there are some patients with portal hypertension and oesophageal varices in whom bleeding occurs only from gastric varices. We have not seen gastric varices developing after the sclerosis of distal oesophageal varices, and the possibility that obliteration of the oesophageal varices results

in a rise in portal pressure sufficient to increase the risk of bleeding from gastric varices seems most unlikely.

The problem of postsclerosis rebleeding is complex and hinges on such factors as persistence, obliteration, recurrence, and ulceration of varices and on the occurrence of gastric varices and portal hypertensive gastritis. In this report, we have not fully evaluated these factors. Nevertheless, some points seem clear. First, recurrent esophageal variceal rebleeding was usually controlled by further injection. Second, those who failed sclerosis predominantly bled from gastric varices or portal hypertensive gastritis, Warren et al. (1986).

In the kings college study Clark et al (1980) bleeding from gastric varices has been seen at a time when the esophageal varices were "abliteroted". The problem of bleeding from gastric varices is often disregarded, and indeed Terblanche et al (1979, 1981) are quoted as showing that gastric

varices also disappear with esophageal sclerotherapy. Data were not presented to support. A second point devarving special emphesis is the peristent portal hypertension in the stomach, since bleeding from erosive gastritis is a major cause of death in those patients. The possible reasons for this are discussed by Reynolds et al (1981) in their randomized study of the portocaval shunt at the university of Southern California. Bleeding from gastritis must be considered a preventable complication of portal hypertension similar to gastric or oesophageal varices and can not be disregarded as a cause of failure of sclerotherapy. Warren (1983) . Bleeding from gastric varices and gastritis has been reported to be precipitated by oesophageal variceal sclerosis. Henderson and Warren (1983).

MEASUREMENT OF PORTAL VENOUS PRESSURE

The most widely accepted methods for measurement of portal venous pressure include :-

(1) Percutaneous Trans-splenic Measurement :

The pressure can be measured by passing a needle percutaneously into the spleen. The external zero reference point is 5 cm. below the sternal angle of a supine patient. Atkinson and Sherlock found a normal range of 3 - 17 mmHg with a slight overstimulation of portal pressure with this technique . Presently , this method is rarely utilized because of splenic hemorrhagic complications in 1 % of the cases. It is reserved for those patients in whom direct splenoportography is indicated as in cases of splenic or portal vein thrombosis (Boyer et al., 1977) .

(2) Percutaneous Transhepatic Measurement :

Direct measurement of the portal venous pressure can be achieved through either percutaneous

transhepatic catheterization of the portal vein or percutaneous insertion of a skinny needle under fluoroscopic guidance into an intrahepatic portal branch. The normal portal venous pressure by this method ranges from 5 to 10 mmHg (Boyer et al., 1977).

(3) Umbilical Vein Catheterization :

This technique also allows the direct measurement of portal venous pressure. The portal venous pressure by this technique is similar to the percutaneous technique. However, the rate of failure of umbilical vein catheterization in cirrhotics ranges from 10 to 40 % usually from creation of a false passage. Potential complications include hemorrhage, wound infection, ascitic fluid leakage and portal vein thrombosis. For those reasons and also because of the advantage of percutaneous transhepatic catheterization , this technique is rarely used Reynolds et al., (1970).

(4) Hepatic Vein Wedged Pressure Measurement :

This technique was introduced by Myers and Taylor in 1951. A wedged catheter in a normal liver

produces asatic column of blood that extends down to the sinusoids where multiple intersinusoidal communications will decompress the pressure column. Hepatic vein wedged pressure is therefore slightly less than portal pressure in the normal liver. However , dye infection before pressure measurement may give falsely elevated levels. The most important advantage of this technique is the use of a dependable internal zero reference point, either free hepatic venous or inferior vena cava pressure (Myers, J.D. and Taylor, W.J., 1951). Groszmann and associates have recently developed a new technique based on the use of aballon catheter that allows the measurement of occulded and free hepatic venous pressure without moving the catheter.

The normal wedged hepatic venous pressure (WHVP) is from 8 to 12 mmHg with approximately a 5 mmHg differential between WHVP and the free hepatic vein or inferior vena cava pressure. This gradient is called corrected sinusoidal pressure (CSP) with less than 5 mmHg being a normal measurement (Tygstrup et al., 1962) . Pressure readings vary with values greater than 30 mmHg denoting a markedly hypertensive state. Good correlation has been shown between indirect methods of obtaining

portal pressure and measurements taken directly at the time of sugery

Corrected sinusoidal pressure allows differentiation of truly elevated sinusoidal pressure from those secondary to an elevated systemic venous pressure

Clinical Significance :

Evaluation of the portal venous pressure established the diagnosis of portal hypertension and confirms the prescence of chronic liver disease in some patients. The degree of elevation may also provide an objective assessement of the exact extent of the disease. Measurement of portal pressure by a combination of different techniques at different sites can help determine the etiology of portal hypertension . Unfortunately , no strong correlation has been found between the degree of portal venous pressure elevation and the intiation of a variceal bleeding episode - Viallet et al., however have indicated that variceal bleeding rarely occurs when the pressure gradient is less than 12 mmHg (Viocllet et al., 1975).

HEPATIC BLOOD FLOW

MEASUREMENT OF HEPATIC BLOOD FLOW

Since 1864, many techniques have been developed in the search for accurate quantitation of hepatic blood flow. Bradley classified these techniques into direct and indirect methods (Bradley, S.E., 1966).

I- Direct Methods :

The methods that have been used for direct measurement of hepatic blood flow include :

1. Electromagnetic Flowmeter :

The electromagnetic flowmeter has been employed for measuring hepatic blood flow in patients during the surgical procedure. It is calibrated for the measurement of absolute flow. However, the associated surgical trauma and the low reproducibility in measuring venous flow limit its use in the clinical field (Denison,Jr, A.B. et al., 1955 and Nordlinger , B.M. et al., 1980).

2. Angiographic Techniques :

Evaluation of portal venous hemodynamics by angiography is a complex and invasive technique which is subjected to significant mechanical and pharmacologic manipulations by the radiologist. However , standardized visceral artery angiography , carried through to the venous phase and supplemented at times by splenoportography or direct transhepatic venous portography has evolved as the most widely implemented method. In addition a cineangiographic techniques has been used by some investigators (Viammonte, Jr. M, et al., 1970).

. Hepatic Angiography :

This technique is a semiquantitative estimation of portal blood flow.

. Cineangiography :

This technique requires biplane fluoroscopy and catheterization of the umbilical vein. Measurement of the velocity of 2 - 3 mm lipoidal droplets represent true flow velocity within the range of medical interest. However , this method may be

too-invasive or complex for routine use (Ohnishi.K. et al., 1985 and Roichle, F.A. et al., 1972).

II- Indirect Methods :

The available techniques for indirect measurement of hepatic blood flow include :

1. Isotopic Techniques :

With these techniques different isotopic materials have been used which are either completely removed by the phagocytic activity of reticulo endcthelial (RE) cells of the liver, or rapidly diffused with instantaneous equalibrium between blood and tissue concenteration. However, there are two different techniques :-

a- Single Injection :

A single dose of radioactive materials is injected intravenously. These substances such as labeled chromium phosphate , radio active gold and heat denatured plasma albumin labeled with ^{131}I are completely cleared only by reticubenothelial cells in the liver.

b- Dilution "Washout" Technique :

The indicator - dilution technique is based on the hydraulic principle that the volume flow within a moving stream may be determined by its ability to dilute a known amount of indicator measured with respect to time.

Various routes for administration of ^{85}Kr and ^{133}Xe have been used. In recent years, these tracers have been injected into the portal vein via catheterization of the umbilical vein (Shoemaker, W.C. et al., 1961). Matie and associates assessed hepatic tissue perfusion during the surgical procedure both before and after DSRS. Using the krypton washout technique , they showed a mean perfusion of 116 ml/minute/100 gm of liver before DSRS and 108 ml/minute/100 hm of liver after DSRS . (Mathie, R.T. et al., 1980).

2. Clearance and Extraction Techniques :

Elimination of a substance by the liver depends on a number of factors. These include the total hepatic

blood flow, binding of substance to blood constituents , up take and transport across the hepatocyte plasma memberane , distribution of flow (Shunting) within the liver, intracellular transport and metabolism and excretion or secretion of the substance. In an attempt to simplify these complex biological events, hepatic substance removal has been conceptualized as a function of both flow and the capacity of the liver to remove the substance in the absence of flow limitations (intrinsic clearance).

a- Galactose Clearance :

In the 1950's galactose clearance was proposed as a method of measuring hepatic blood flow because of the high enzymatic activity in the hepatocytes for galactose metabolism. However lack of accurate methods of analysis for the quantitation of low plasma galactose levels was an obstacle in the border acceptance of the test substance (Greinier, A. ; Laberge, 1973).

In 1980, Henderson et al., modified Mason's assay using galactose oxidase to effectively measure galactose concentration in the rang of 0 - 100 mg/L.

(Mason et al., 1977). With these plasma galactose concentrations , Henderson et al., utilized the original concept of galactose clearance to measure hepatic blood flow in liver disease - continuous intravenous infusion of galactose at rates varying from 25 - 100 mg/min was shown to be the most practical and accurate method of measuring distribution and elimination kinetics for estimation of hepatic blood flour.

They recently demonstrated that galactose clearance and extraction were significantly different when the hepatocytes were challenged with a two fold increase in galactose infusion.

Also, hepatic extraction was independent of liver size and galactose clearance but dependent on the hepatocyte functional reserve (Henderson, J.M. et al., 1986).

b- Indocyanine Green Clearance :

In cirrhotics , a wide range of hepatic blood flow was observed using indocyanine green clearance with either single injection or continous infusion

technique. However, this technique is most accurate when a sizable extraction for the dye is present (Villeneuve, J.P. et al., 1982 and Bosch, J. et al., 1981).

3. Imaging Techniques :

Recently , the advanced imaging techniques have been utilized to measure blood flow. The pulsed doppler flowmater has been used in the last two years to measure portal blood flow. The magnetic resonance imaging (MRI) is another new technique which may be used in the near future for estimation of portal flow (Bernardino, M.E. et al., 1986). The pulsed doppler flowmeter has been combined with an ultrasound sector scanner to measure blood flow. It can be calculated from the blood flow velocity and the cross - sectional area of the vessel. The blood flow velocity can be determined from the doppler shift frequency. The cross sectional area of a blood vessel can be measured on the B-made tomogram (Moriyocsuf et al., 1984). The obvious advantages of this

system are its simplicity , non invasiveness , lack of irradiation and its ability to quantitatively calculate portal flow.

The portal system is a favorable object for pulsed doppler flowmeter use for the following reasons :

a- There is little phasic change in flow velocity.

b- The blood vessele is relatively large with long stright segment with little branching..

c- The blood vessele is relatively close to the body surface (Ohnishi, K. et al., 1985).

Ohnishi et al. used this technique for hemodynamic evaluation of portal system in patients with chronic liver disease and portal hypertension. It can be used for the diagnosis of portal vein thrombosis, reversal of portal or splenic flow and arterioportal shunts. Inokuchi et al., used the pulsed doppler flowmeter for the assessment of hemodynamic changes after selective shunt operations. This technique may be helpful in the near future in selecting patients for surgical treatment (Ohnishi, K. et al., 1985 and Inokuchi et al., 1984).

THE PATHOGENESIS OF PORTAL-SYSTEMIC COLLATERALS

The term portal hypertension is synonymous with portal-systemic collaterals, the mechanism of development of these collaterals is not at all understood Recent investigations have implicated prostaglandins and have shed some light on the pathogenesis of new vessel formation. Prostacyclin (PGI_2) is a potent vasodilator and inhibitor of platelet aggregation. Activity has been shown to be increased in the aortae of spontaneously hypertensive rats (Moncoadoa At al.,1977) and in the portal vein wall of rats after partial ligation of the portal vein. as portal-systemic collaterals developed. Sham-operated rats showed , there was a close correlation between the portal vemous pressure and the degree of PGI_2 activity.

Prostacyclin may be produced by the wall of hypertensive vessels. If released into the bloodstream, such vasodilatory substances might stimulate the development of collaterals. Such an inhibitor of platelet aggregation may prevent thrombosis. They extrapolate this reasoning to the human condition and suggest that in sustained

portal hypertension prostacyclin might induce the development of esophageal varices (and other collaterals) by its vasodilator effect and might cause bleeding by inhibiting platelet aggregation . (Moncada et al., 1977).

McCormack and Co-workers (McCor marck et al., 1983) studied the direction of blood flow within esophageal varices by ultrasound and by injection radiography. Using a Doppler probe that was passed through the biopsy channel of a fiberoptic endoscope, they demonstrated that the flow in varices was usually cephalad but was sometimes caudad and, occasionally, bidirectional in the same varix. They found that the direction of flow was determined by the phase of respiration, and noted in a patient with bidirectional flow at two sites 2 cm apart in a single varix that blood flow reversed direction with respiration. They concluded that this pattern occurred at the site of a perforating vein, i.e., a vein leading from the subserosal to the submucosal vessels, and could be most easily explained by reversal of the direction of flow through the communicating vein. They demonstrated such flow by

direct injection of a sclerosant-contrast mixture. The implication of these findings is that esophageal varices represent a backwater in which blood ebbs and flows with changes in pressure and with respiration ... in the periesophageal varices, which are , by contrast the real blood-carrying varices. This provides some insight into how and why varices bleed. It has been suspected that patients with large varices are at the greatest risk of bleeding(Lebrec et al ., 1980).

LeBrec and his colleagues have indicated that bleeding from acute gastric erosions (AGE) in patients with esophageal varices (EV) has the same pathophysiologic significance as bleeding from EV. (Lebree et al .,1980). They believe that the reduction of portal pressure by beta-blockade (Lebrec et al.,1981). or by portal-systemic anastomosis reduces the risks of recurrent hemorrhage. (Ruff 1976), and their co-workers have reported that hemorrhage from AGE is rare after surgical portal decompression . Sarfeh has suggested that the prognosis of beeding in patients with AGE is associated with high rates of recurrent hemorrhage and death (Sarfeh et al.,1981). In his most recent retrospective evaluation, Sarfeh found that patients with EV who were bleedding from AGE respon-

ded better to portal decompressive procedures than to surgical therapy designed to treat the AGE .

(Lebrec et al, 1981) Lebrec and his associates have included 25% of patients who bled from acute gastric erosions in their investigation of propranolol in the management of patients who bled from esophagogastric varices (Lebrec et al 1981). They use as evidence to support their concept the fact that portacaval shunts diminish the risk of bleeding from both esophageal varices and acute gastric erosions.

ENDOSCOPY OF UPPER G.I.T.

Indications:

The development of fibroscope simplified the technique of direct intragastric observation, reduced the patient's discomfort and at the same time overcome most of the observational blind spots in the stomach. The usefulness of gastric endoscopy in diagnosis of stomach diseases was further enhanced. At the same time fibrogastroscopic observation has made it possible to study diseases which in many cases had been considered to be beyond capabilities of gastroscopic examination. Katon (1981).

Specific Indication of Oesophagoscopy:

1. Diagnosis of oesophageal varices:

   In cases of portal hypertension, pan endoscopy is specifically indicated.

2. Diagnosis and assessment of oesophagitis.
3. Confirmation of suspected neoplasms.
4. Investigation of oesophageal obstruction.
5. Operative procedures.
6. Diagnosis and management of upper gastro intestinal haemorrhage.

7. Investigations of all cases of dysphagia.
8. Substernal or subxiphoid pain of non ischemic nature.

Peterson et al. (1981).

Indications of Gastroscopy :

1. Persistant dyspepsia without X-ray finding.
2. Differential diagnosis of gastric ulcers.
3. Differential diagnosis of other gastric lesions:

   Gastroscopy can easly and directly differentiate between any radiological deformity which can not be diagnosed radiologically e.g., antral gastritis, giant rugeal folds, polyps, filling defect and infiltrative lesion.
4. Evaluation of the extent of the lesions.
5. In cases of peptic ulceration it is the most accurate method of follow up.
6. Investigation of the cause of haematemesis or melena.
7. Evaluation of post operative stomach.
8. Study of gastric mucosa in various diseases e.g. pernicious anaemia, achlorhydria and dry gastritis.
9. Direct study of the tone of the stomach. Also, the effect of various drugs on the stomach, also it is used as a research tool.

10. In evaluation of the inflammatory lesion and its differential diagnosis.

Contra-indications :

Absolute contraindications:

1. Uncooperative patient.
2. Psychopathics are unable to understand the significance of an examination.

However there are no absolute contra-indication for upper G.I.T. endoscopy.

Relative contraindications :

1. Deformity of the spine: It is only impossible in cases of marked deformity of the thoracic spine due to rickets or tuberculosis of the spine.

2. In cases of : cardiac failure, coronary disease, dyspnea, disturbance of consciousness, acute infection.

3. In cases: where there is marked deviation of the oesophagus, due to the presence of an aortic aneurysm, or mediastinal tumor.

4. In cases of: oesophageal diverticula, zenkers diverticula.

5. Forcible insertion: is contra indicated in cases of oesophageal stenosis due to oesophageal cancer or cardiac cancer.

6. In cases of corrosive gastritis and phelgmonous gastritis.

7. Endoscopy immediately after an abdominal operation.

8. Jaundiced patient should not undergo endoscopy until evidence has been obtained that their serum does not contain hepatitis associated antigen, bacause there is no particular method for sterilizing the endoscope.

9. Cervical arthritis.

10. A combination of a short "Bullneck" a small mouth, and long teeth.

11. Sinus infection.

12. Suspecion of recent gastric perforation.

Care of the patient :

History and physical examination, informations on a patients hypersensitivity to drugs, complications and presence or absence of a disturbance of passage through the oesophagus and cardia are especially important before a gastroscopic examination.

I. Investigations prior to endoscopy :

A. Barium meal or swallow: It is important to reveal any absolute contra indications.

B. E.C.G. for cardiovascular patient.

C. Tests for bleeding tendency.

2. Preparation and medication :

Solid food should not be taken for 6 hours prior to endoscopy, but clear fluids is allowed upto 3 hours before examination. Atropin is given as a pre-medication. It protect against dysrhythmia which occure during any form of intubation. It also, reduces the excessive salivation which is sometimes associated with the use of Diazepam

(Zbinden & Randal, 1967). General anaesthesia is indicated in children, psychopathics, heavy alcoholics very nervous subjects when difficulty is encountered in passing the endoscope through the oesophagus and where gastric washout is indicated.

Care during endoscopy:

The patient should be maintained at the level of consicusness to tell about any unusual pain or discomfort which are the first worning of trauma and to cooperate, to obey simple commands as to swallow during the passage of the endoscope.

Post endoscopy care :

The patient must be put under medical observation at least until he is completely recovered from the diazepam, he has to be kept in the left lateral position as further aspiration may occur.

Hazards :

With the development of fibrogastroscope the hazards of endoscopy are limited.

1. Complication of medication :

The major hazards of local anaesthesia are acute rhythm changes, failure and even sudden death which may be contributed to pulmonary aspiration.

2. Perforation :

It is the most serious complication of oesophagoscopy, it is more likely to cause perforation than gastroscopy. With the use of F.G.S. as with the rigid endoscope it may occur as a result of over inflation, pain is a good warning.

3. Pulmonary aspiration :

It may lead to the development of pneumonia and its complications and may be the cause of dath (Schiller et al., 1972).

Aspiration pneumonia is responsible for most post endoscopy morbidity.

Cardiovascular Complications:

Cardiovascular complications may be due to local anaesthesia or to intubation.

Haemorrhage :

Post endoscopy haemorrhage is not a common complication.

Other complications include :

1. Impaction.
2. Trauma from unplanned withdrawal.
3. Mucosal burn (in the old endoscope).
4. Swallowed mouth guard.
5. Rupture of the stomach.
6. Over distention simulating perforation.
7. Volvulous of the small bowel.
8. Subparotid painless swelling.
9. Inappropriate intubation.
10. Infection.
11. Transport problems.

Normal Endoscopic Picture :

1. Oesophagus.

It discribes a gentle curve which, altered by the spinal and diaphragmatic movement. It deviates to the left in the neck and upper thorax and then it

deviates to the right. It reaches the mid line behind the arch of aorta. The normal oesophageal mucosa appears pale pink in colour, and glistening, moisted by mucus. In the proximal oesophagus there are broad longitudinal folds the change from oesophageal squamous to the gastric columnar mucosa is indicated by the change of pale and glistening oesophageal mucosa to the deep red or orange gastric mucosa. The junctional border may be sharply defined giving a dentate appearance. However, there are considerable variations in the position, prominence and pattern of this boundary zone in different individuals.

2. Stomach :

The most proximal landmark situated in the stomach is the lake of mucus which is made up of gastric juice.

Swallowed saliva and bile stained fluid. It is found on the posterior wall in supine position, and on the greater curve at the fundus when the

patient is lying in the left lateral position. This mucus should be aspirated to seek out any lesion and to minimize the danger of regurgitation.

The rugae of the stomach are most prominent on the lesser curve and the posterior wall aspect of the body of the stomach. The lesser curve aspect of the stomach is comparatively smooth and marked by shallow lengitudinal folds. The most persistent land mark of the mid stomach is the angulus, situated about half way down the lesser curve. Beyond the angulus there is a change in the direction of the lumen, of the stomach to the right, posteriorly, also the fold on the greater curve becomeslongitudinal and is directed into the antrum, the distal antrum is often rather featureless. The viewed tangentially which indicates a change in the direction of the lumen.

In certain phases of peristalses,the shape of the pylorus changes with the phase of peristalsis.

Reflux of bloody mucus may be seen or sometimes bile stained fluid regurgitates across the pylorus.

INJECTION SCLEROTHERAPY

The first report of the injection of sclerosant into esophageal varices for the control of bleeding came in 1939 from Crafoord and Frenkener two Swedish Surgeons.

Moersch considered that splenectomy should procede any attempt at injection sclerotherapy. Also he noted that the majority of the patients with recurrent varic-eal bleeding had gastric fundal varices which where not seen in the other patients in whom therapy was regarded as successful. Moersch considered that the presence of fundal vaxices is a contraindication to injection sclerotherapy.

Wodak, an Austrian surgeon, in 1960, he belived that the esophageal varices were useful colleterale helping to decompress the portal system, and there fore, to obliterate them by injection of sclerosant into the veins was unwise. Wodak modified the tech-nique of injection sclerotherapy by injection not into the varix but alongside it, the aim being to create a layer of fibrosis to cover the va ices leaving them as functioning collaterals.

AIM OF INJECTION SCLEROTHERAPY

Endoscopic Sclerotherapy can be formed in three different ways aiming at either:

1. Thrombosing obliterating, and eradicating varices by intra variceal injection.

2. Covering the varices with a fibrous layer by para-variceal injection and.

3. Achieving sclerosis of the varices and the inner esophageal wall by the combined peri-and intravascular injection technique (Soehendra N., et al., 1983).

Rose(1983) provided an evidence for the safety of intravariceal sclerosant injection, when two millileters of sclerosant contrast mixture was injected into the largest varix under radiological control in an old woman with cryptogenic cirrhosis, duodenal and gastric ulcers and three esophageal varices. Histological study of her esophagus showed incomplete thrombosis of the injected varix without ulceration or inflammation at the injection site.

So in the absence of compression, distention and blanching at the site of injection suggest that Scler-

osant is extravasating, regardless of the lack of resistence to injection (Rose, et al., 1983).

Sclerotherapy is usually effective and safe but injection should be intravariceal for maximum effect and to avoid ulcer or stricture formation, (Yassin et al., 1983).

PARAVARICEAL INJECTION

Wodak, in 1960 reported that the esophageal varices were useful collaterals helping to decompress the portal system, and therefore, to obliterate them by injection of sclerosant into the veins was unwise. Wodak modified the technique of injection sclerotherapy by injecting alongside the varix aiming to create a layer of fibrosis to cover the varices leaving them as functioning collaterals.

The treatment must be carried out in stages. On average, 6 months are necessary in order to adequately selerose the inner wall of the esophagus, a process requiring more than ten sessions. During the relatively long period of treatment it is not uncommon for recurrent bleeding to occur. This is often hemorrhage due to erosion of the varices as a result of necrosis

of the mucosa caused by the sclerotherapy which account about 45% (Soehendra. N., et al., 1983).

SITE FOR INJECTION

Crafoord and Frenkner, 1939 their first injection was undertaken in the bleed-free interval into three varices in the proximal esophagus which was associated with very little bleeding.

Stelzner and lierse 1981, demonstrated that the source of bleeding will be found in the lower part of the esophagus in more than 90%. Therefore, a direct attack of the lower esophagus varices is a reasonable concept.

Recently, almost all studies emphasize that injections into the varices should be placed distally as near to the esophagogastric junction as possible

Terblanche,1979; Rose et al., 1983; Clark et al., 1980; and Yassin et al., 1983). After the ring of varices at gastro-esophageal junction has been injected, a further injection is given at a more proximal level in the case of large varices extending high in the esophagus (Clark et al., 1980).

Even those who are using the extravascular tachnique, sclerotherapy is initiated at the cardiac and of the esophoqus.

Multiple superficial pricks are done circumferentially involving the lower 3-5 cm of the esophagus (Paquet, 1981, 1983).

FREQUENCY OF RE-INJECTION

For definitive treatment of esophageal varices, sclerotherapy couress are needed to be repeated. In the original article by Crafoord and Frenckner (1939) the procedure was repeated every other day during the space of a month.

For initial obliteration of varices, more spaced courses have been also suggested. Johnson (1977)repeated the injections every one month and clark et al.(1980) every 3-4 weeks. Lewis et al. (1980) carried out the second injection after 7-10 days and hence forward they repeated at one month intervals. In the studies of Barsoum et al. (1982) reinjection was performed 2 weeks after the emergency session and repeated 6 week later or earlier if rebleeding occured. Rose et al., (1983) Performed generally injections at weekly intervals untile

the varices were thrombosed or markedly reduced in size. There after treatment took, place monthly until varices had disappeared.

However , Westably et al, (1984) made a comparative study of the efficacy and complications of injection sclerotherapy carried out at intervals of one week and three weeks up to the time of varices obliteration and found that the number of courses of injection required for obliteration of the varices was not different in the two groups and despite a shorter time scale for obliteration in the weekly treated patients the frequency with which further episodes of bleeding occurred before that was less but the difference was not statistically significant.

Rose et al., (1984) made up his new regime is to give a maximum of three courses at this interval and then to allow a period of three weeks before reassessing. by endoscopy, the need for further injection.

FOLLOW-UP IN CHRONIC SCLEROTHERAPY

Most authorities firmly emphasize that after the varices have been controlled, the patients should be re-examined every 3 to 12 months.

INDICATIONS OF INJECTION SCLEROTHERAPY

Endoscopic variceal sclerotherapy is contemplated as an emergency and elective therapeutic procedure for bleeding esophageal varices or as a prophylactic measure of the silent cases. (Paquet, 1982).

Terblanche (1983) reported that definitive control of variceal hemarrhage was achieved in 95% of hospital admissions, usually with a single intravariseal injection (70%), while Paquet (1983), stated, that the success rate of stopping acute and massive hemorrhage from varices by means of paravariceal injection is 93% with a rigid instrument and 81% with a flexible one, and added that sclerotherapy is the treatment of choice of acute variceal hemorrhage.

Once the bleeding is controlled, chronic aclerotherapy is a possible long-term treatment in place of medical support or surgical operation (Terblanche et al., 1979).

On the other hand, Shields, (1979), stated that after the haemorrhage has been controlled, there are several therapeutic options, only one of which is injection sclerapeutic options, only one of which is injection sclerotherapy. Actually, the technique is indicated in the following.

Pinel et al., (1976) hoped that sclerotherapy should be used as a prophylactic measure to prevent haemorrhage, as soon as the presence of varices is realised. Kronberger et al, (1976) claimed that the best result are obtained when varices are just begining to form and when the esophageal mucosa is still intact. Prophylactic injections were used by Soehendra (1980): with no primary mortality.

SCLEROTHERAPY IN COMBINATION WITH SPLENECTOMY AND DEVASCULARISATION OPERATION

Moersch (1947) considered that splenectomy should precede any attempt at injection sclerotherapy. So did Fearon and Sass-Kortsak, 1959. Macbeth (1955) agreed with them as he noticed that the varices were much more controllable after splenectomy. However, he advised that in those recently bled, it is best to inject first then to have a splenectomy and finally to complete the injection course.

Recently, terblanche et al., (1979) remined of the possible value of sclerotherapy complementary to splenectomy and devascularization operations. They raised the question of whether or not injection sclerotherapy, together with a devascularization procedure, might be the method of choice in patients in whom eradication of the varices proves difficult.

SCLEROSING SOLUTIONS

Ethanolamine oleate has remained popular in Great Britain and South Africa for intravariceal injection,

whereas polidocanol is the most popular in Austria and west Germany for paravariceal injection.

The important considerations in choosing sclerosing aqents are:

1. Safety and efficacy.
2. Whether an intravariceal approach will be used or paravariceal.
3. Availability and status with the federal drug Administration.
4. Case of injection through small gauge needles.
5. Frequency of anaphylaxis.
6. Side effects such as fever, pleural effusion and chest pain and.
7. Stability and half life of agent (Jensen, 1983).

The Problem of the Gastric Varices :

There are conflicting views regarding the effect of the oesophageal varices on the gastric varices which, themselves, are a matter of great controversy concerning their incidence and rupture in portal hypertention.

In the Lancet editorial letter (1979), it has been suggested that the preservation of perioesophageal veins patent, may explain why no gastric varices appear or enlarge after injection.

On the other hand, Williams and Clark (1981) are of the opinion of increased gastric variceal bleeding after oesophageal varices eradication, but they presumed that the incidence is probably less than 5%. They added that, since there have been no reports of increased portal pressure after injection sclerotherapy, it might be the increased survival after the procedure which gives time for the gastric varices to manifest.

Moersch (1947), in a series of 22 patients, reported that in 6 out of 10 unsuccessfully treated patients, gastric varices were found on gastroscopic examination.

At two necropsies, he confirmed the gastric origin of the bleeding. He went to the conclusion that the presence of varices in the cardiac end of the stomach is a contradication to the injection type of treatment of oesophageal varices.

On the contrary, Johnston and Rodgers (1973) after 15 years of sclerotherapy experience on 117 patients with acute variceal bleeding reported that it was extremely rare to find bleeding originating in varices below the cardia. Terblanche et al. (1979) and Lewis et al. (1980) had the same result.

Bleeding gastric varices after sclerotherapy of the oesophageal varices was reported as a complication in 6.7% (Raschke and Paquet,1973) of the cases and in 8.3% (Clark et al., 1980). However, the latter found no evidence that gastric varices had increased in size after sclerotherapy.

Terblanche et al. (1979) and Lewis et al. (1980) claimed that gastric varices that were seen occasionally prior to sclerotherapy have disappeared completely in several patients.

However, in 1978, Barsoum et al. carried out a pilot study to follow the process of variceal injection sclerotherapy by radiolography. They indicated that, even with a wide oesophagoscope inside the oesophagus compressing the upward flow, the sclerosant did not pass downwards across the cardia to fill the gastric varices.

Brunner (1980) claimed that through the recently developed fibrescopes, sclerosing of varices of the fundus can be achieved. But Printen (1980) found it too difficult to bend the scope around and spear vessels with any creat degree of certainty.

MATERIAL AND METHODS

Material and methods of this study consists of forty patients with recurrant gastro-intestinal bleeding due to oesophageal varices in bilharzial hepatic fibrosis.

Our cases were selected from Benha University Hospital, two groups of patients were included in this study:

1. 20 patients with bilharzial hepatic fibrosis without sclerotherapy (19 males and one female) Their age ranged from 22-65 years with the mean of 39.5 S.D ± 12.9.

2. 20 patients with bilharzial hepatic fibrosis after sclerotherapy by intravariceal injection of ethanol amine aleate (all of them were males) their age ranged from 28-64 years, with the mean of 46.5 S.D ± 10.5.

All patients were submitted to through history taking and clinical examination

With especial stress on history of :

1. History of precipitating factors of bleeding as ulcerogenic drugs, sudden straing, chest infection, previous operations, and injection sclerotherapy.

2. The frequency of previous bleeding.

3. Jaundice.

4. Fever.

Examination for :-

1. Pulse, blood pressure, temperature, pallor, Jaundice, pellagric rash, Herpes, Candidasis Oedema Lewer limbs.

2. Examination of chest, Heart, Nervous systems.

3. Abdominal Examination for : dilated veins, liver, spleen, kidney, Ascites, Masses.

The following investigation were done for all patient :-

1. Urine analysis, stool analysis, blood picture, HB %.

2. Endoscopy (Fibropticendoscopy).

3. Liver biopsy.

4. Gastric biopsy.

Upper G.I.T. Endoscopy :

Fibroscopy was done for all patients using olympus G.L.F type K.

Preparation and Medication :

All the patients were fasted for at least 10 hours before examination :

* Immediately before examination each of them was injected intravenously by 5-10 mg Diazepam. According to his state of consciousness which was kept at a point where he was just able to answer comands sloowly.

* Buscopan was given only where there was pyloric spasm.

Passing the instrument :

The patient lies in his left side, his mouth opend, a mouth guard is fixed into it :-

* A forward viewing endoscope is connected and checked, It's distal 20 cc were lubricated by olubricating jell.
* The endoscope then introduced into the mouth close to the midline advanced slowly.
* The patient asked to swallow.
* Deep inspiration was encouraged.
* The tip was slightly bent during introduction and maintained by fixing knob.
* As soon as the tip has been swallowed, the fixing knob was pushed into the free position.
* The instrument then advanced in the oesophagus under vision.
* The air insuflation and fluid aspiration, controles, used to obtain a good view and to remove the fluid in the oesophagus.
* The oesophagogastric junction is about 40 cm from the incisor teath and is normally visible as a distinct change in the colour of mucosa.
* The oesophageal mucosa is whitish while that of the stomach in pink.

* Inflation of the stomach with air and aspiration of fluids is essential to examination, It was examined in a systemic manouver using the angling mechanism, The state of gastric mucosa, the presence of any mucus besides, the presence or absence of biliary reflux and the motor activity of the stomach were noted.
* The pyloric ring was examined then the 1st and 2nd parts of the duodenum.

Post Endoscopy Care :

* The patient should be kept in the left lateral position till he is fully concious.
* He should not take any thing by mouth for at least 3 hours, or to undertake any responsible work within 24 hours after receiving Diazepam.

Endoscopical diagnosis of gastritis:

The endoscopic changes which occur in the gastric mucosa were classified according to the description of Taor et al. (1975).

(i) a fine pink speckling or'scarlatina' type rash.

(ii) a superficial reddening, particularly on the surface of the rugae giving a striped appearance.

(iii) a fine while reticular pattern separating areas of raised red oedematous mucosa resembling a 'snake skin'.

(iv) discrete red spots analogus to the chemy red spots described in the oesophagus. These spots can become confluent giving a local area of severe gastritis which may bleed.

(v) a diffuse haemorrhagic gastritis.

Liver biopsy :

Needle liver biopsy and Histopathological examination. This was done using Menghini technique by using true cut needle as described by Sherlock (1981).

Histopathological examination :

Endoscopic biopsies were taken from stomach, one from the fundus of the stomach, and one from

the pyloric antrum. and fixed in 10% formalin, embeded in parafin and stained with hematoxyline and eosin, Alcian P.A.S. (periodic acid Schiff) and Reticulin stains.

Diagnosis of Gastritis :

The principles of classification of the inflammatory changes in the fundal or antral area have been based on the scoring planned by Kekki et al., (1983).

Score 0 : Normal epithelium.

1 : Chronic superficial gastritis (accumulation of round cells without loss of normal glands).

2 : Slight atrophic gastritis (slight loss of normal glands).

3 : Moderate atrophic gastritis (Moderate loss of normal glands).

4 : Severe atrophic gastritis (Severe loss of normal glands).

* Congestion (Ectasia) :

The lamine propria is the seat of many dilated blood spaces, most of these spaces are filled with blood.

Blood picture : Table No.(3)

- Haemoglobin % :

  all cases showed low haemoglobin % before treatment, the mean value was (67.85%) group I & (75.8%) group II.

- RBC's count :

  The mean value was (3,792,500/C.ML in group I SD ± 287904) & (4,537,500/C.ML in group II SD ± 486023).

- WBC's were not changed significantly the mean value being (6580/C.ML in group I + SD ± 1132) & (6385/C.ML in group II & S.D ± 1092).

Liver biopsy :

All cases show schistosomal hepatic fibrosis. Some of them show mixed lesions. We failed to obtain samples from two patients, because of marked shrinkage of the liver.

Endoscopic results of the stomach :

* group I : 4 cases (20%) showed gastritis one of them was combined with gastric varies one case (5%) gastric varices and free in 16 cases (80%) Table No.(5)

* group II: (with sclerotherapy): 7 cases (35%) showed gastritis and free in 19 cases (65%) Table No (6).

Histopathological results of group one :

* Fundus : Table No (7).
* Epithelial changes : 8 cases (40%) showed columinar metaplasia, 4 cases (20%) showed chronic superficial gastritis and one case (5%) showed slight atrophic gastritis. Fig. No (6).
* Lamina proparia: 8 cases (40%) showed congestion. Fig. NO (1) 2 cases (10%) showed oedema, 5 cases (25%) showed cellular infilteration and 3 cases (15%) showed fibrosis.

* Antrum :
* Epithelial changes : 3 cases (15%) showed columinar metaplasia.
* Lamina proparia: no case of congestion, 2 cases (10%) showed oedema, 2 cases (10%) showed cellular infilteration and one case (5%) showed fibrosis.

Histopathological results of group two (with sclerotherapy):

* Fundus

* Epithelial changes : 10 cases (50%) shoed columinar metaplasia, 3 cases (15%) chronic superficial gastritis and 1 case (5%) slight atrophic gastritis.

. Lumina proparia changes : 15 cases (75%) showed congestion, 6 cases (30%) showed oedema, 4 cases (20%) showed cellular infilteration and 5 cases (25%) showed fibrosis.

* Antrum :

. Epithelial changes: 3 cases (15%) showed columinar metoplasia.

. Laminar proparia changes: no case of congestion, 3 cases (15%) showed oedema, 3 cases (15%) showed cellular infilteration and 2 cases (10%) showed fibrosis.

* Metaplastic changes :

In the present study, the metaplastic changes were in the form of columnar metaplasia i.e. changing the character of the columnar secretory cells into columnar cells resembling those of the small or large intestine. The goblet cells were completely absent throughout all the work as prooved by the negative alcian blue staining.

Table (1): History and clinical picture without sclerotherapy

NO.	Age	Sex	Abd. Dis-Comf.	Airophagia	Dysphagia	Nausia	Melena Haemat-emesis.	Diarrhea Dysentry	Past. Hist. Jaundice	Resp. Tract. infection	Urinary Tract infection	Anti-B - Treatment	Spleen	Liver	Ascitis	Tenderepi.
1	50	male	+	+	−	−	+	−	+	−	−	+	+	sh	−	+
2	50	male	+	−	−	−	−	−	−	+	−	−	+	sh	−	+
3	40	male	+	−	−	−	−	+	−	+	+	+	+	sh	−	+
4	26	male	+	−	−	−	+	+	+	−	−	−	splen.	sh	−	+
5	57	male	+	−	−	−	−	+	−	+	−	+	−	sh	+	+
6	65	male	+	−	+	−	+	−	−	−	+	−	+	sh	−	+
7	57	male	+	+	−	+	−	+	+	+	−	+	+	sh	−	+
8	40	male	+	−	−	−	+	+	−	−	+	−	+	not asse.	+	+
9	35	male	+	−	−	−	−	−	−	+	−	+	+	not asse.	−	+
10	32	male	+	−	−	−	+	+	+	−	+	−	+	enlarged	+	+
11	23	female	+	−	−	+	−	−	−	+	−	+	+	sh	−	+
12	30	male	+	−	−	+	−	+	+	−	−	+	+	sh	−	+
13	61	male	+	−	−	+	+	+	−	+	+	−	+	sh	−	+
14	22	male	+	−	−	−	−	−	+	−	−	+	splen.	sh	−	+
15	28	male	+	−	−	−	−	−	−	+	+	−	+	sh	−	+
16	35	male	+	−	−	−	−	−	−	+	−	+	+	sh	−	+
17	42	male	+	−	−	−	−	−	−	+	−	+	+	sh	−	+
18	24	male	+	−	−	−	−	−	−	+	−	+	+	sh	−	+
19	37	male	+	−	−	−	−	−	−	+	−	+	+	sh	−	+
20	45	male	+	−	−	−	−	−	−	−	−	+	splen.	enlarged	−	+
Mean = 39.55 SD ± 10.56																

Past History = Past Hist. Respiratory Tract = Resp. Tract Abdominal discomfort = Abd. discomf.
splen = splenectomy not asse. = not assessed sh. = shrunken

Table (2): History and clinical picture after sclerotherapy group II

NO.	Age	Sex	Abd. Dis-Comf.	Airophagia	Dysphagia	Nausia	Melena Haemat-emesis.	Diarrhea Dysentry	Past. Hist. Jaundice	Resp. Tract. infection	Urinary Tract infection	Anti- β - Treatment	Spleen	Liver	Ascitis	Tenderepi.
21	50	male	+	−	+	−	+	−	−	−	−	+	splen.	sh	−	+
22	56	male	+	−	+	−	++++	+	−	−	−	+	splen.	sh	−	−
23	32	male	+	−	+	−	+	+	−	+	−	+	+	enlarged	−	+
24	48	male	+	+	+	−	++ +	−	+	+	−	+	splen.	sh	−	++
25	57	male	+	−	−	−	+ +	−	−	−	−	+	+	sh	−	+
26	35	male	+	+	+	−	− − −	−	+	−	−	+	+	sh	−	+
27	40	male	+	+	+	−	− − −	+	+	−	−	+	+	sh	−	+
28	38	male	+	−	+	−	+	−	+	+	−	+	+	sh	−	+
29	52	male	+	+	+	−	+	+	+	+	−	+	+	sh	−	+
30	40	male	+	−	+	−	+	+	+	−	−	+	+	not ass.	−	+
31	60	male	+	−	+	−	+	+	+	+	−	+	+	enlarged	−	+
32	64	male	+	−	+	−	+	+	+	−	−	+	+	sh	−	+
33	30	male	+	−	+	−	+	+	+	−	−	+	+	sh	−	+
34	28	male	+	−	+	−	+	+	+	−	−	+	+	sh	−	+
35	50	male	+	−	+	−	+	+	+	−	−	+	+	sh	−	+
36	45	male	+	−	+	−	+	+	+	−	−	+	+	sh	−	+
37	63	male	+	−	+	−	+	+	+	−	−	+	+	sh	−	+
38	40	male	+	−	+	−	+	+	+	−	−	+	+	sh	−	+
39	50	male	+	−	+	−	+	+	+	−	−	+	splen.	sh	−	+
40	53	male	+	−	+	−	+	+	+	−	−	+	+	enlarged	−	+
Mean = 46.55 SD ± 10.56																

Past History = Past Hist.. Respiratory Tract = Resp. Tract. Abdominal discomfort = Abd. disconf.

Shrunken = Sh. splen = splenectomy not ass. = not assessed

HB %		R. B. C's		W. B C s	
without	ésclerotherapy	without	ésclerotherapy	without	ésclerotherapy
62 %	71 %	3500000	4000000	4900	4700
60 %	70 %	3480000	4000000	6600	6600
70 %	78 %	3800000	4700000	8400	8200
65 %	70 %	3500000	4200000	6900	6800
80 %	82 %	3630000	4200000	5700	5600
62 %	74 %	3600000	3900000	5500	5400
60 %	65 %	4400000	5650000	5000	4800
82 %	86 %	4400000	5150000	5500	5400
60 %	68 %	3540000	3900000	7200	7000
80 %	84 %	4200000	5300000	7000	6800
71 %	80 %	3900000	4900000	7700	6800
69 %	78 %	3800000	4700000	7000	6800
65 %	77 %	3400000	4150000	6600	6400
67 %	77 %	3800000	4800000	6400	6300
62 %	74 %	3500000	4550000	8100	7900
72 %	76 %	4000000	4800000	8400	8200
65 %	78 %	4000000	4550000	7400	7200
68 %	72 %	3700000	4400000	7200	7000
65 %	79 %	3900000	4900000	5500	5400
72 %	77 %	3800000	4000000	4600	4400
67.85% ±6.75	75.8% ±5.32	3792500 ±295383	4537500 ±498649	6580 ±1161.5	6385 ±1130.8

table No (3): Show changes in blood picture

No.	Without Sclerotherapy	With Sclerotherapy
1	xx	x
2	xx	-
3	xxxx	xx
4	xxx	x
5	xx	xx
6	xxxx	x
7	xxx	-
8	xxx	xx diverticulum
9	xx	x
10	x	-
11	xx	xx
12	x	xx active bleeding
13	x	xx
14	x	-
15	x	x
16	x	xx
17	xxxx	xx
18	xxx	xx Hyperaemic mucosa
19	xx	xx
20	x	xxx

- = Free
x = early oesophageal varices.
xx = mild oesophageal varices.
xxx = moderate oesophageal varices.
xxx = advanced oesophageal varices.

table No. (4) show results of endoscopy of oesophagus of group one and two.

No.	Endoscopy of stomach group one
1	Free
2	Free
3	Small submucosal vein +Gastritis
4	Free
5	Free
6	Free
7	Free
8	Free
9	Gastritis
10	Free
11	Free
12	Free
13	Gastritis
14	Free
15	Free
16	Free
17	Free
18	Free
19	Gastritis
20	Free

Table No. (5) show results of endoscopy of stomach group one

No.	Endoscopy of stomach group two
1	Free
2	Free
3	Gastritis
4	Free
5	Free
6	Free
7	Free
8	Free
9	Free
10	Free
11	Free
12	Gastritis
13	Gastritis
14	Free
15	Gastritis
16	Gastritis
17	Free
18	Gastritis
19	Gastritis
20	Free

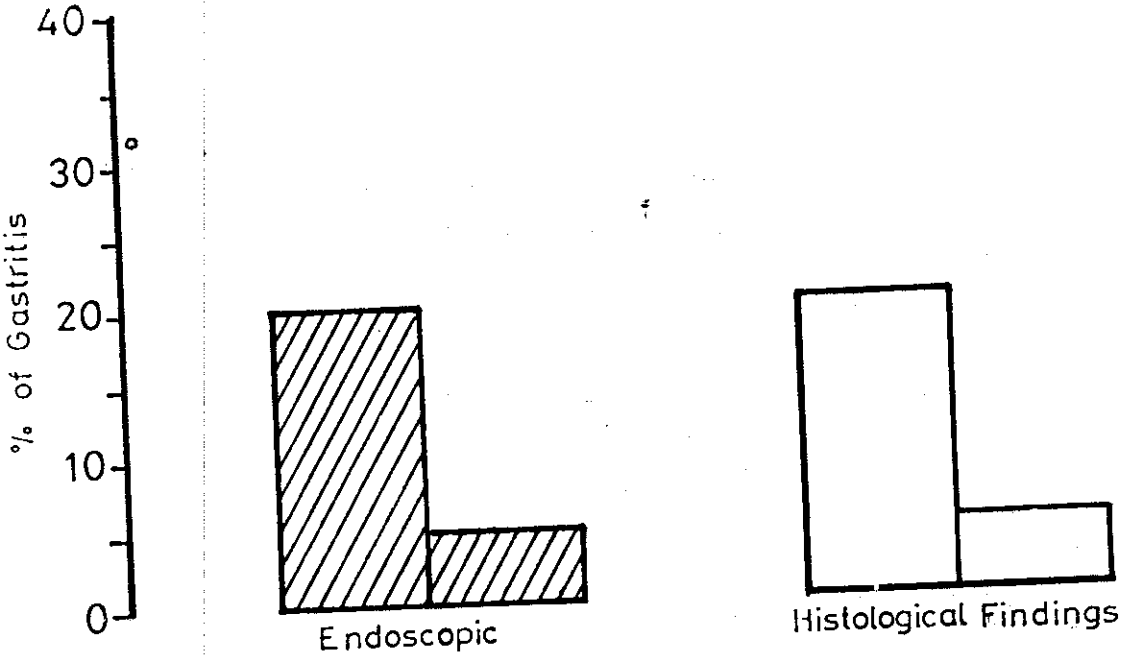
Table No. (6) show results of endoscopy of stomach group two.

Site		No. of cases	Epithelial changes						lamina propria changes							
			Hyperplasia	Columnar metaplosia		Gastritis			Congestion (ectasia)		oedema		Cellular infiltration.		fibrosis	
			-	No.	%	No.		%	No.	%	No.	%	No.	%	No.	%
Fundus	I	20	-	8	40%	4 cases show score 1 1 case show score 2		20% 5 %	8	40%	2	10%	5	25 %	3	15%
	II	20	-	10	50%	3 cases show score 1 1 cases show score 2		15% 5%	15	75%	6	30%	4	20%	5	25%
Pylorus	I	20	-	3	15%	-		-	-	-	2	10%	2	10%	1	5%
	II	20	-	3	15%	-		-	-	-	3	15%	3	15%	2	10%

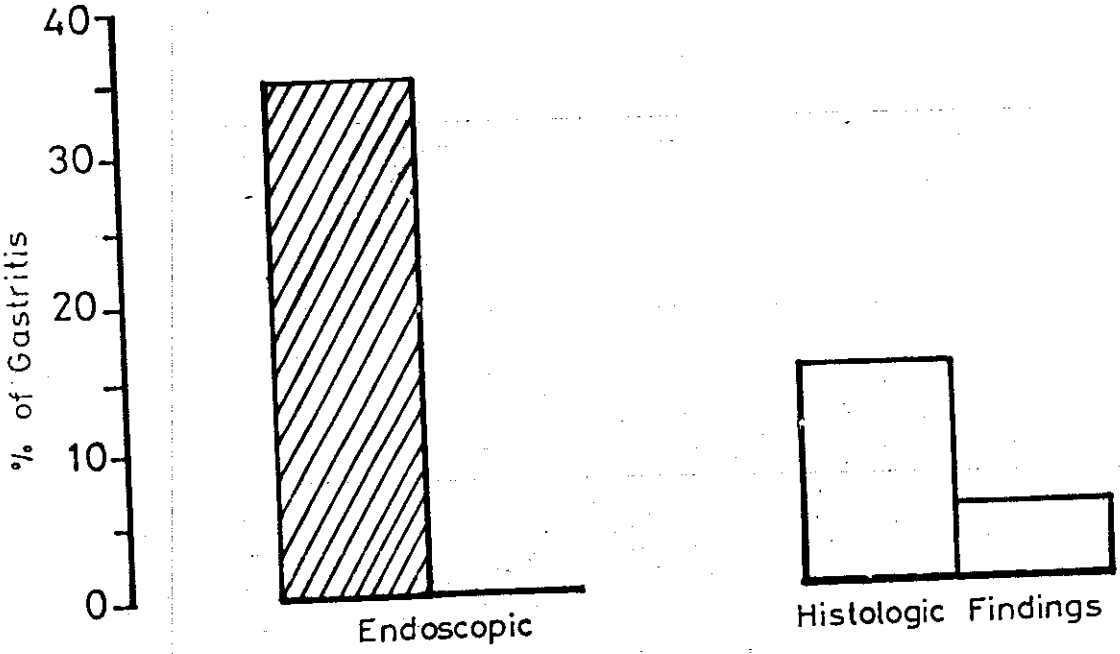
table (7): Pathological changes in group I,II

group NO.	Total NO.	Macroscopic appearance	Microscopic appearance
I	20	. gastritis 4 cases gastric varices 1	. mucosal ectasia 8 cases . oedema 2 cases . Cellular infiltration 5 cases
II	20	gastritis 7 cases	. Mucosal ectasia 15 cases . Oedema 6 cases . Cellular infiltration 4 cases

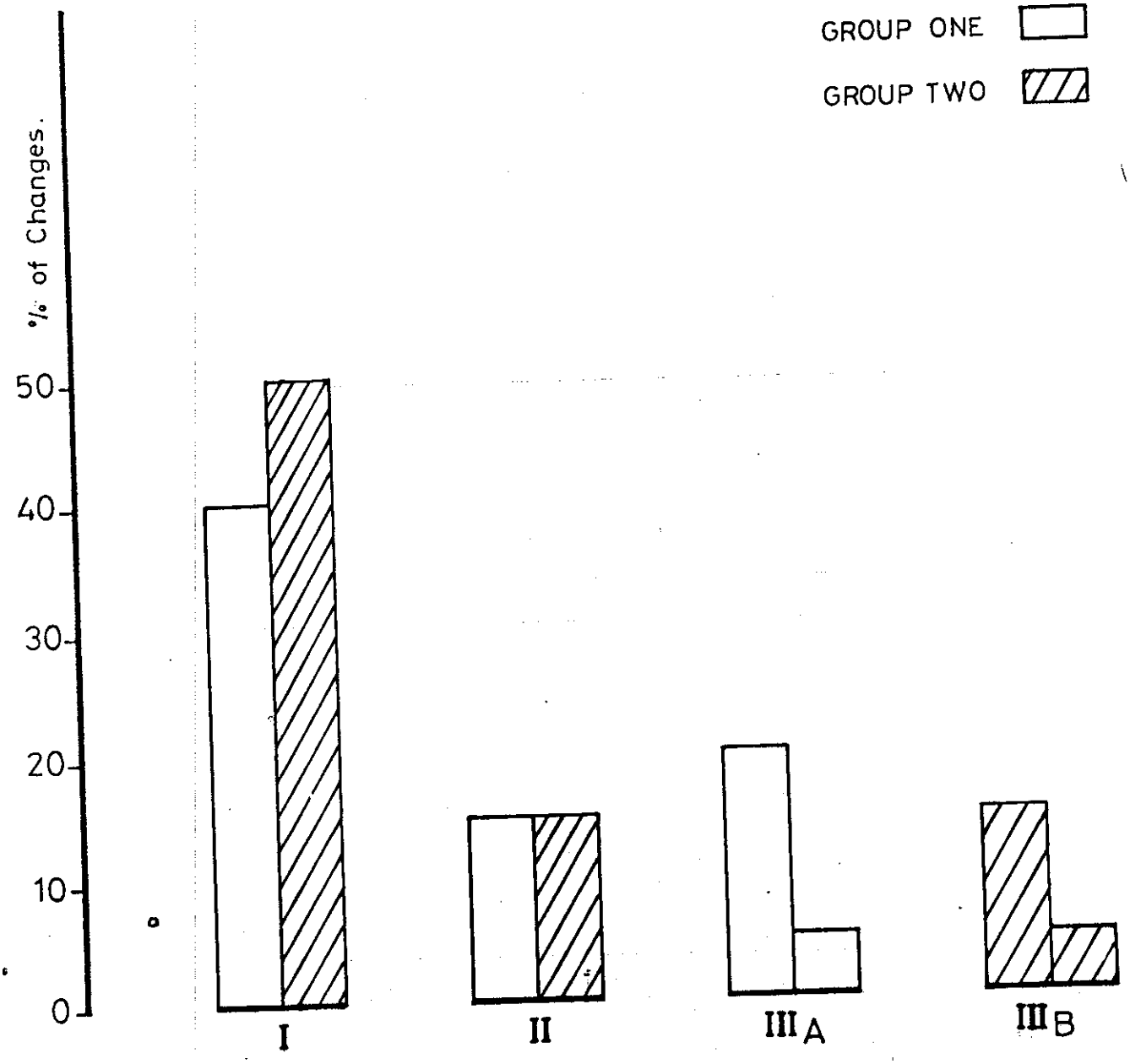
table no (8) showing macroscopic & Microscopic findings in gastric specimens.



Histogram 1: ENDOSCOPIC & HISTOLOGICAL FINDINGS IN GROUP ONE



Histogram 2: ENDOSCOPIC & HISTOLOGICAL FINDINGS IN GROUP TWO.



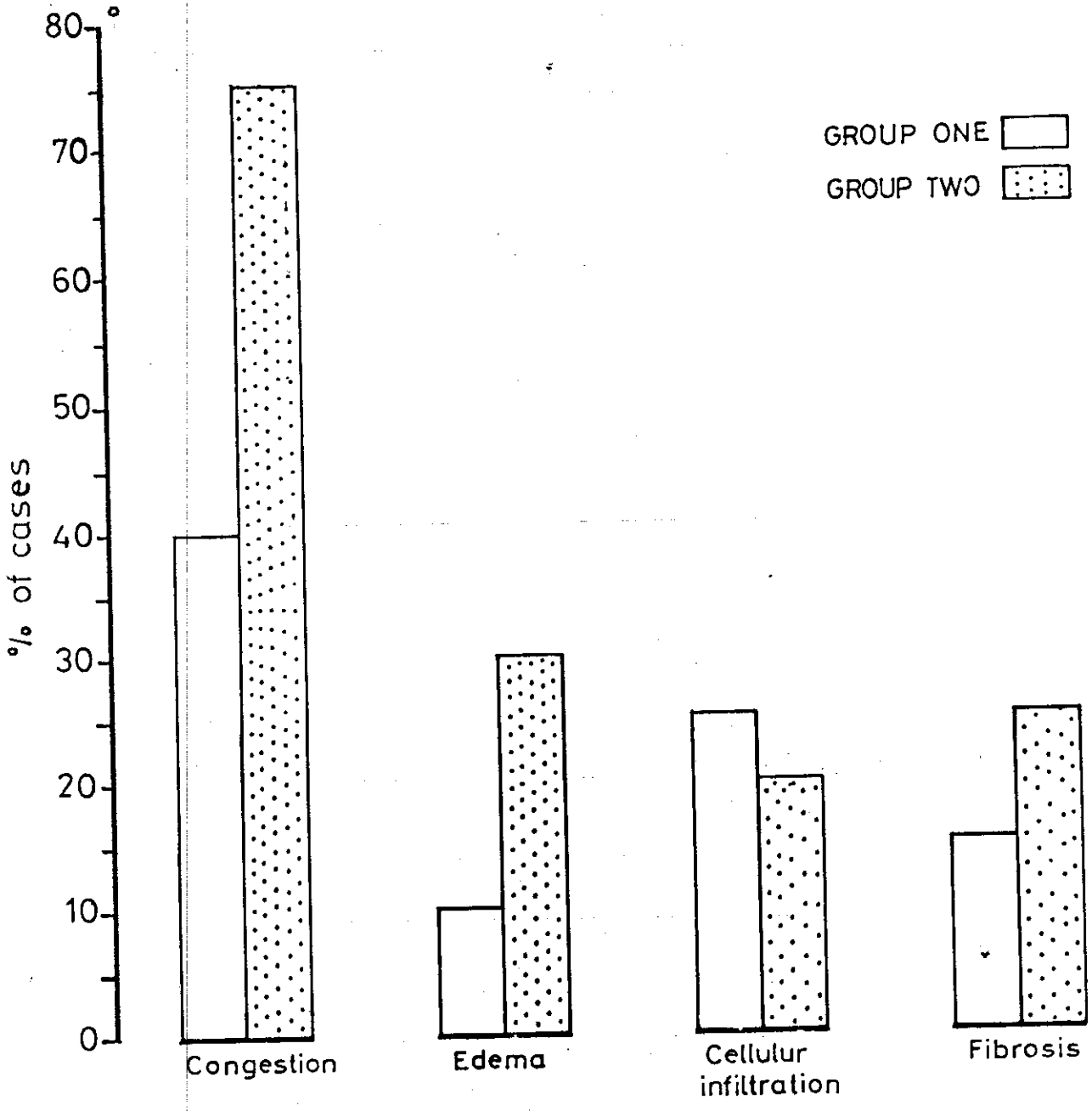
Histogram 3: PATHOLOGICAL CHANGES IN FUNDUS & ANTRUM.

I : Columinar Metaplasia in Fundus.

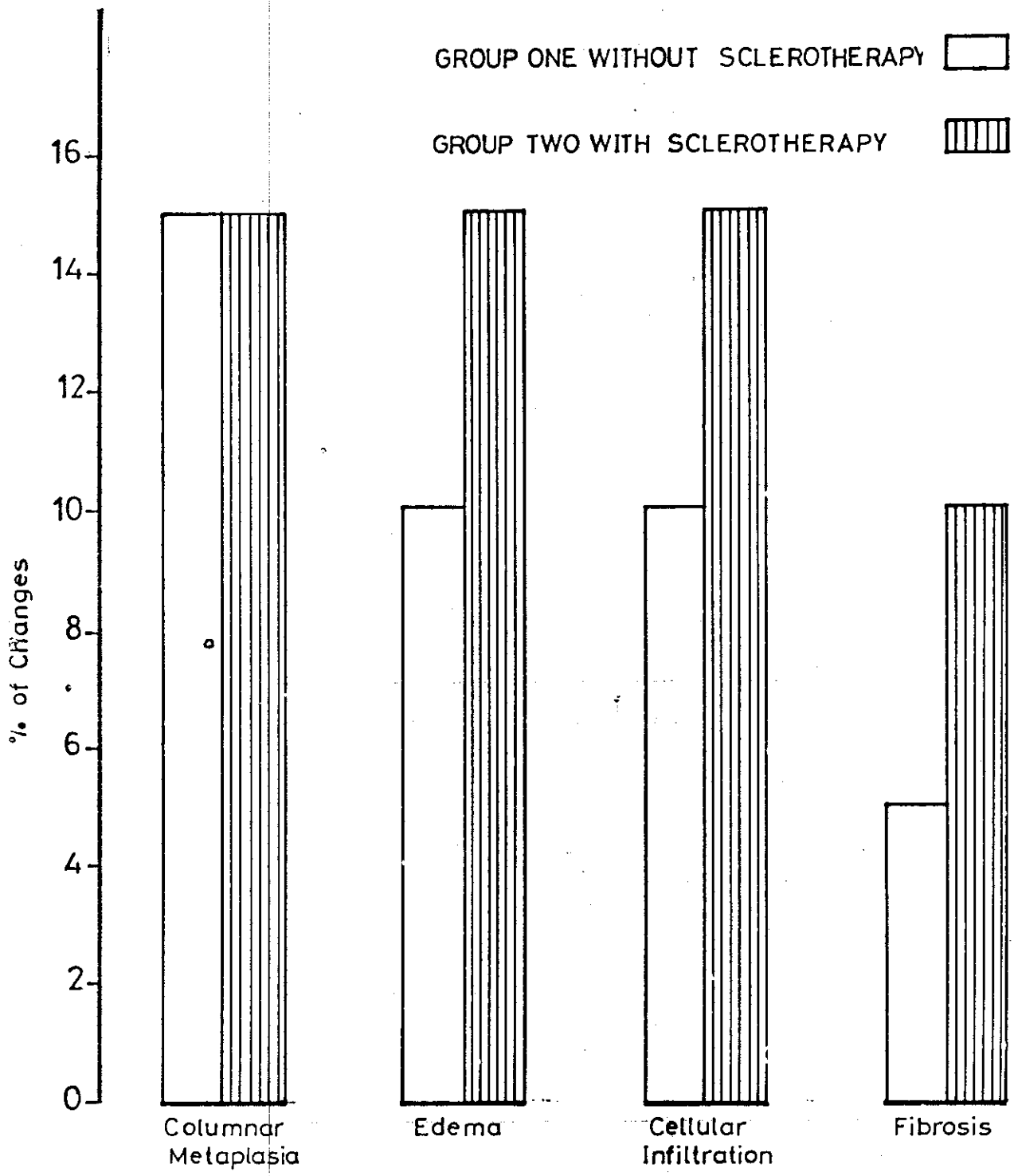
II : Columinar Metaplasia in Antrum.

III_A : Gastritis Score 1, 2.

III_B : Gastritis Score 1, 2.



Histogram4: LUMINA PROPRIA CHANGES IN FUNDUS.



Histogram 5: EPITHELIAL & LAMINA PROPRIA CHANGES IN ANTRUM.

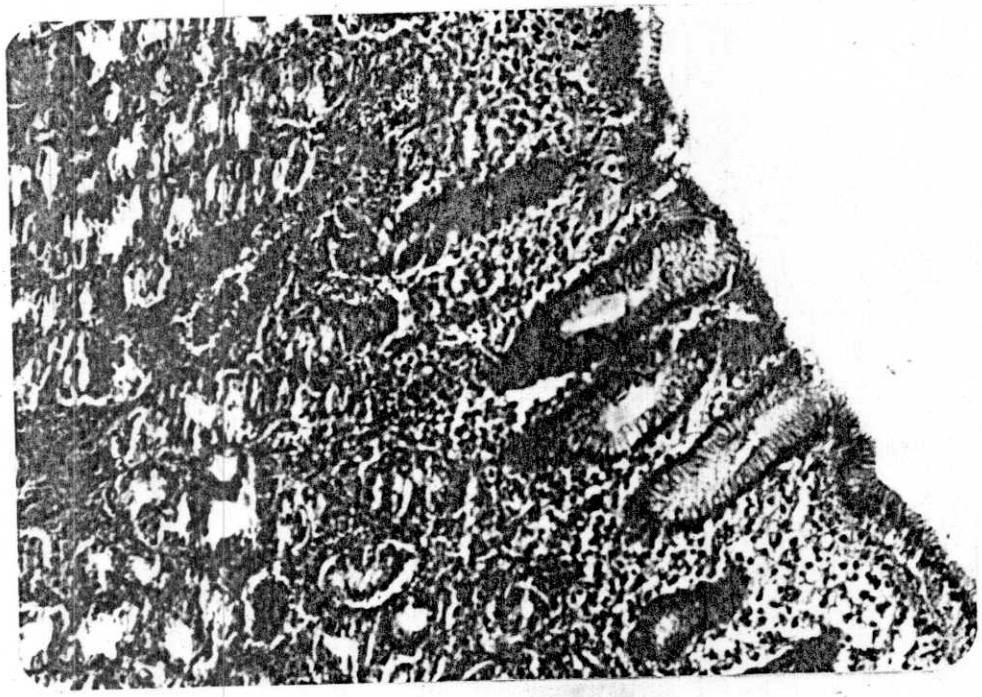


Fig.(1): Gastric mucosa with prominent ectasia Hx & E (x200).

Fig.(2): Gastric mucosa showing regenerating glands. and ectosia. Hx & E (x200).

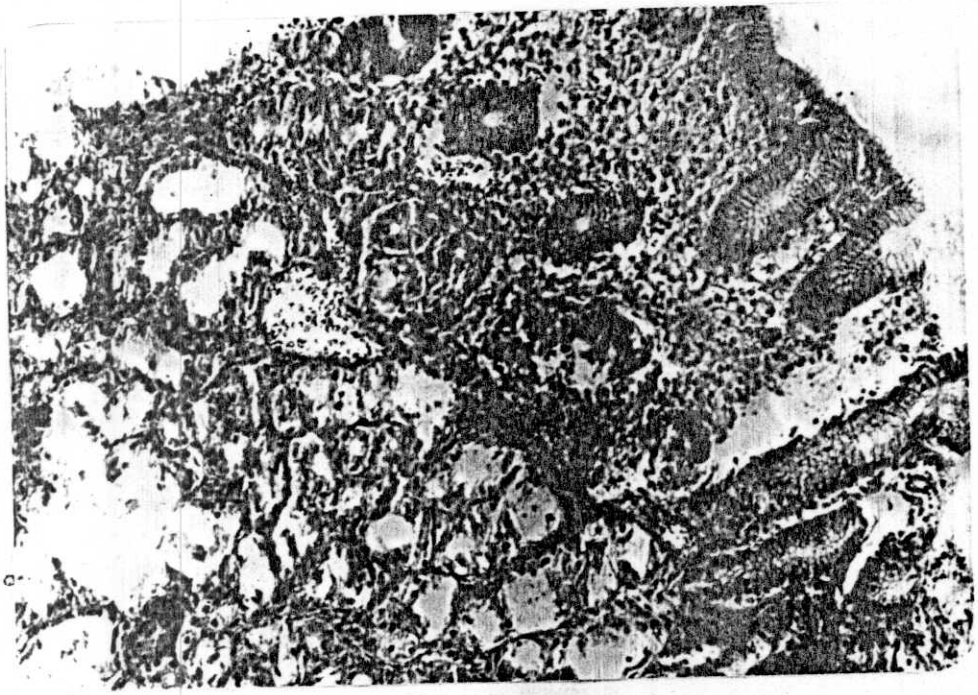


Fig.(3): Gastric mucosa showing chronic superficial gastritis, diffuse cellular infilteration and ectasia. Hx & E (x200).

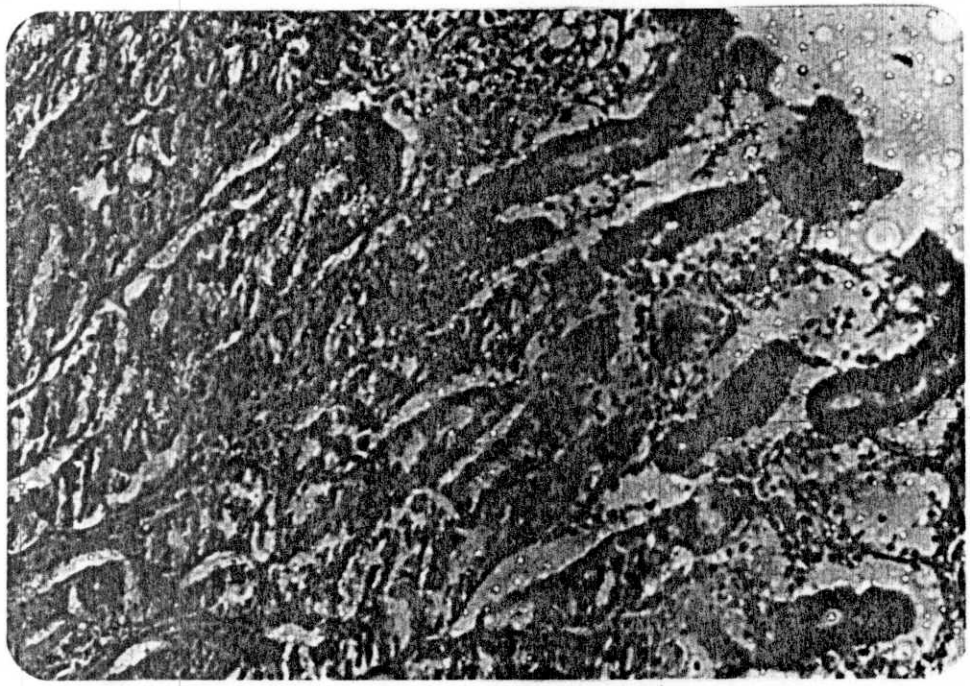


Fig. (4): Gastric mucosa showing chronic superficial gastritis. Hx. & E. (x200).

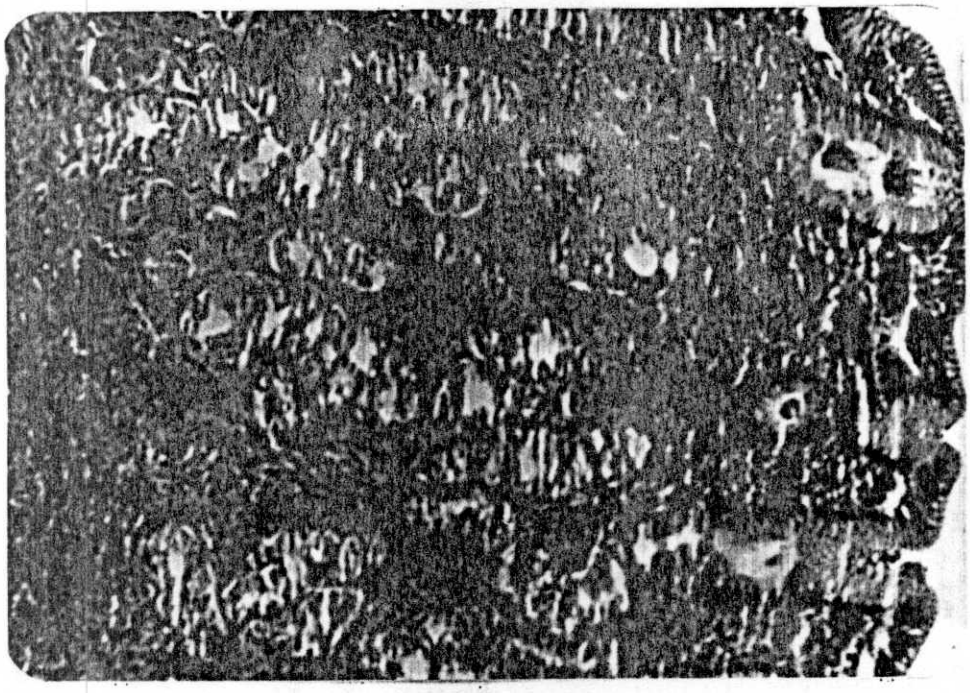


Fig. (5): Gastric mucosa showing chronic superficial gastritis and focal cellular infilteration. Hx. & E (x200).

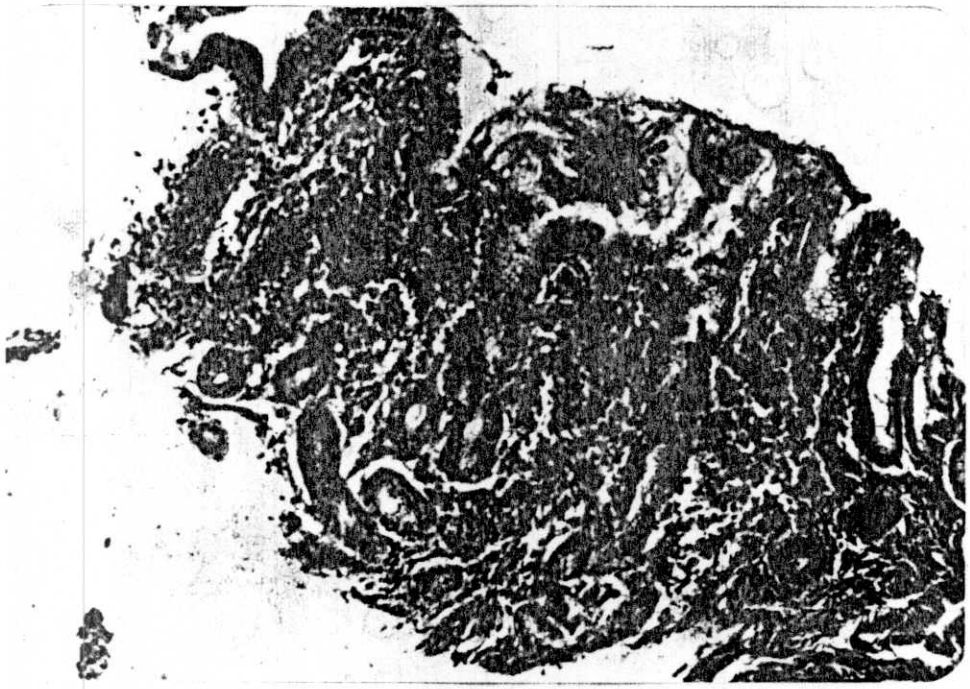


Fig.(6): Gastric mucosa showing chronic superficial gastritis and aggressive focal cellular infilteration. Hx & E (x40).

Fig.(7): Castric mucosa showing slight atrophic gastritis and focal cellular infilteration Hx & E (x 200).

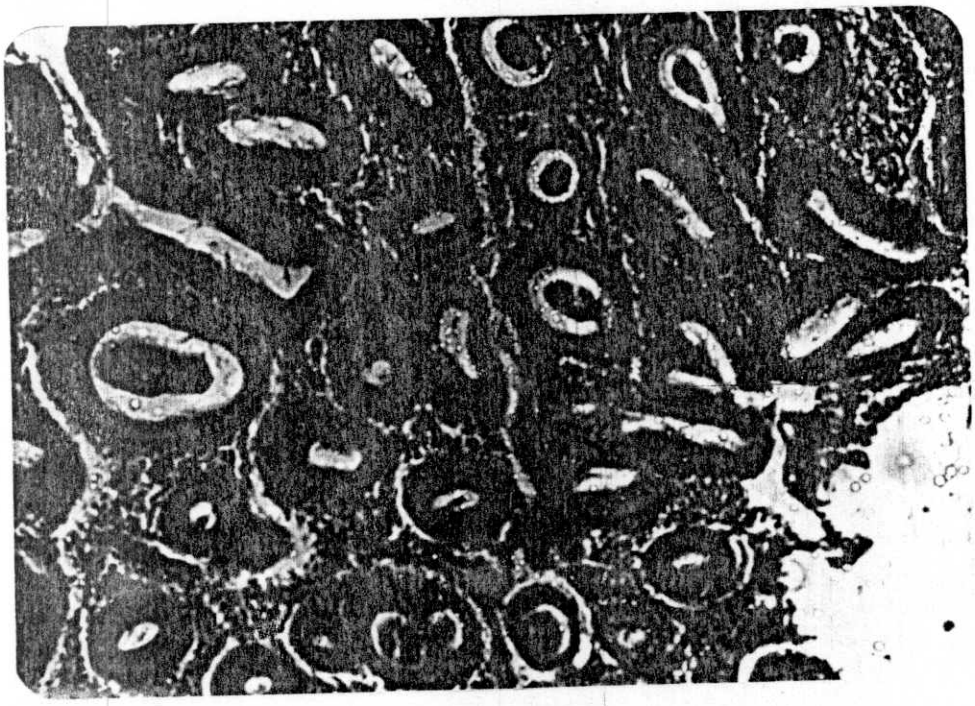


Fig.(8): Gastric glands showing strong positive PAS staining. PAS (x200).

DISCUSSION

DISCUSSION

Bilharziasis is a common endemic disease in our locality. Hepatosplenic affection may be associated with portal hypertension, ascites and oesophagogastric varices. Haematemesis and/or melena are one of the most serious emergencies in our locality and bleeding oesophagogastric varices is the most common cause of upper gastro-intestinal haemorrhage among patient with bilharzial liver cirrhosis (Sheir et al., 1980).

Bilharziasis reduces the total economic production by 30 %, Mousa et al.(1969).

Schistosoma ova were first noticed in the stomach by May (1908).

Kadry and Hashem (1957) stated that gastric bilharziasis was apparently rare because of the rapid postmortem auto digestion of the stomach.

Zaki and Hashem (1962) found that gastric bilharziasis represents 0.08 % of the gastro-

intestinal bilharziasis, while bilharziasis of the small intestine represents 41 % Hashem (1962) explained the variation in incidence among the various organs on the basis of the richness of the part with venules. The more the venous supply in the part the more will be the chance for the female worms to get in and lie ova.

Khairy et al., (1967) stated that bilharziasis of the oesophagus and stomach was mainly due to disturbed haemodynamics.

Ata and Abdellatif, (1971) attributed the finding of bilharzial ova in the stomach to portal hypertension and to the presence of collaterals.

Hunter and El Rooby, (1979) found bilharzial ova in the stomach in cases of colonic polyposis.

El Fayomy et al. (1981) found bilharzial ova in the stomach in 10% of patients with bilharzial hepatic fibrosis.

Kadry and Hashem (1954) reported a case of gastric bilharziasis of nodular fibrotic type, the mucosa was oedematous, and congested with scattered pale, firm, slightly elevated nodules and with 3 superficial peptic ulcers.

El Sharkawy et al. (1969) stated that patients with bleeding varices tend to have normal gastric mucosa and many of them had gastro-oesophageal reflux, and oesophagitis. Non bleeders had atrophic gastric mucosa.

Farag et al. (1980) found that there was changes in the gastric mucosa in 93.33 % in cases of bilharzial hepatic fibrosis. In the form of chronic superficial gastritis, and oesophageal varices in 27 cases out of 30 but 19 cases only were of bilharzial origin.

El Fayomy et al. (1981) found oesophageal varices in 55 %, reflux oesophagitis in 10 %, and normal picture in 35 % they also found that there were prepyloric polyps in 15 %, gastritis in 10 % ,

pale mucosa in 10%, and normal gastric mucosa in 65% of patients with bilharzial hepatic fibrosis.

In our work we found normal gastric endoscopic picture in 16 cases 80 %, Gastritis in 4 cases (20%) and gastric varices in one case (5%) in group one.

Also we found normal gastric endoscopic picture in 13 cases (65%) and Gastritis in 7 cases (35%) in group two (with sclerotherapy).

As regards the microscopic picture of gastric bilharziasis, Kadry and Hashem, (1957) found thickening of mucosa in some parts, with nodular formation, and many bilharzial ova with bilharzial granulation tissue around. The mucosal glands in these areas were degenerated, where as in the neighbouring parts they were proliferated.

Khairy et al., (1967) suggested the term bilharzia antral gastritis, and they stated that the histopathological picture of the mucosa of the body of the stomach in cases of bilharzial

gastritis is similar to the picture of chronic gastritis, wheather superficial or atrophic.

Souidan et al., (1971) found atrophic gastric glands in some cases of bilharzial hepatic fibrosis.

Ata et al. (1971) recognized bilharzial ova in the gastric mucosa with minimal round cell infilteration, around. This, with eosinophilic infiltertion, and absence of evidences of superficial gastritis, but atrophic mucosa constitutes a pathological entity of bilharzial lesion of the antrum.

Ata and Abdellatif (1971) found very vascular mucosa with many dilated vessels.

El Sharkawy et al., (1977) found many bilharzial ova scattered either single or in groups in the superficial part of the gastric mucosa and giant cells engulfing parts of the ova with normal gastric glands.

Hunter and El Rooby, (1979) found bilharzial

ova and granulation tissue in the stomach of 3 cases of bilharzial colonic polyposis, the other cases revealed non specific inflammatory reaction.

El Fayomy et al. (1981) found that there is no histopathological changes in the oesophagus. They found bilharzial ova in 10 %, atrophic gastritis in 50 %, superficial gastritis in 25 %, and normal mucosa in 25 %.

In our study, histopathological examination did not detect ova in any case in the fundus and antral mucosa or submucosa.

But we found in the fundus of group (1) one columnar metaplasia in 8 cases (40 %), chronic superficial gastritis in 4 cases (20 %) slight atrophic gastritis in one case (5 %) congestion (ectasia) in 3 cases (40 %), oedema in 2 cases (10 %), cellular infilteration in 5 cases (25 %) and fibrosis in 3 cases (15 %). In the antrum of the same group we found metaplasia in 3 cases (15 %), no case of congestion, oedema in 2 cases (10%),

cellular infilteration in 2 cases (10 %) and fibrosis in one case (5 %).

Also as regards the group 2 we found in the funuds columnar metaplasia in 10 cases (50 %), chronic superficial gastritis in 3 cases (15 %), slight atrophic gastritis in one cases(5%),congestion in 15 cases (75 %), oedema in 6 cases (30 %), cellular infilteration in 4 cases (20 %) and fibrosis in 5 cases (25 %).In the antrum of the same group we found columinar metaplasia in 3 cases (15 %), no case of congestion, oedema in 3 cases (15 %), cellular infilteration in 3 cases (15 %) and fibrosis in 2 cases (10 %).

McCormack et al. (1985) found that classic histological features of chronic inflammatory gastritis were seen in 4 of 14 patients, while the other 10 had vascular ectasia with little or no inflammatory infiltrate. Six of the nine biopsies from macroscopically normal mucosa were histologically normal, the remainder showing vascular ectasia.

Also found that successful sclerotherapy of oesophageal varices may induce local changes in blood flow patterns and if this results in an increased venous pressure in area proximal to the site of thrombosis, congestive gastropathy might be predicted. The mean number of sclerotherapy treatments in patients with gastropathy was significantly greater than in those without it. Sclerotherapy appears to increase long term survival (MacDougall, 1982).

The haemoglobin percentage was significantly increased in all patients of group two (after sclerotherapy) than of patients in group one.(The mean average being 75.8 % after sclerotherqpy and 67.85 % before sclerotherapy.

R.B.C's count was also increased in all patients, the mean average being 4537500/Cmm after sclerotherapy and 3792500/C.ML before sclerotherapy. White B.C.'s count were decreased in all patients. The mean average being 6.385/C.ML after sclerotherapy and 6.580/C.ML before sclerotherapy.

In our work we found that in group two (after sclerotherapy), congestion was detected in 15 cases (75%), oedema 6 cases (30%), cellular infiltration 4 cases (20%) & fibrosis in 5 cases (25%). On the other hand in group one we detect congestion in 8 cases (40%), oedema in 2 cases (10%), cellular infiltration 5 cases (25%) & fibrosis in 3 cases 15% i.e. congestive changes in gastric biopsies in group two (after sclerotherapy) was more than that of group one.

The most important element causing gastritis may be the raised portal pressure itself. Obstruction of the venous drainage from the stomach can induce changes in the gastric mucosa. Palmer, in 1957 induced portal hypertension in dogs by portal vein ligation and found that both the mucosal & submucosal veins in the stomach wall became dilated. Both he and Sandblon (1975) observed similar changes in

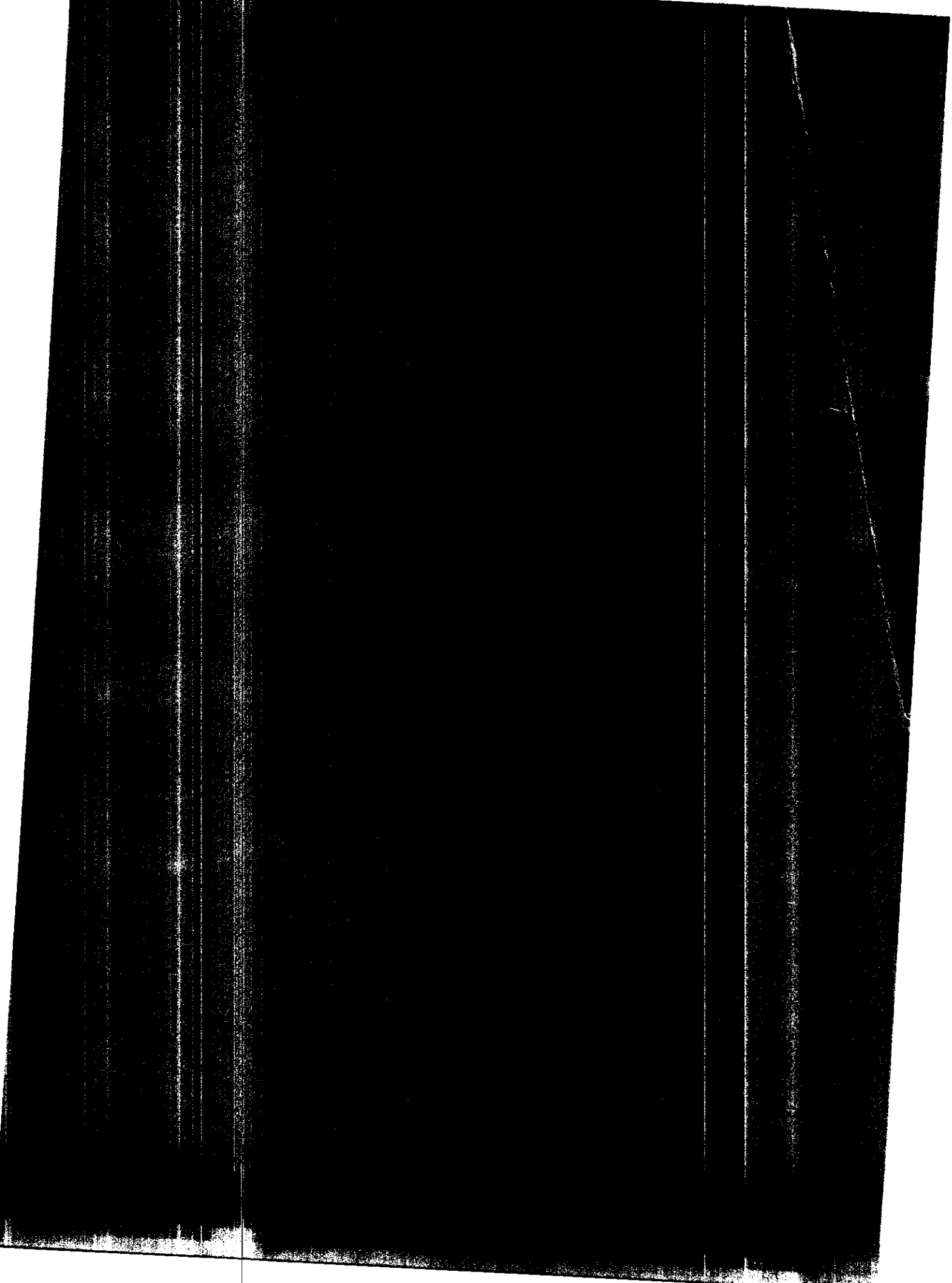
gastric biopsies from patients with portal hypertensions. Alternatively gastritis might be because of gastric mucosal ischaemia secondary to arteriovenous shunting which can be demonastrated in the stomachs of both animals (Manabe et al 1978) and humans (Hashizume et al 1983). with portal hypertension.

The histological changes are entirely consistent with an increase in venous pressure producing a congested gastric mucosa. The occurance and severity of this congestive gastropathy may depend, however, not only on the total level of portal pressure but also on local blood flow characteristics which may or may not transmit this increased pressure to the gastric mucosal & submucosal veins. Differences in local blood flow patterns may explain why some patients develop gastropathy and others do not. Successful sclerotherapy of oesophageal varices may induce local changes in blood flow patterns and if this results in an increased venous pressure in areas proximal to the site of thrombosis, congestive gastropathy might be predicted.

From these results we found that, gastritis was 25% before sclerotherapy, it was 20% after sclerotherapy, & ectasia was 40% before sclerotherapy, & 75% after sclerotherapy. This means that ectasia was more prominent than gastritis as previously diagnosed endoscopically & but now proved histopathologically to be ectasia this difference was more prominent after sclerotherapy.Hence the term of congestive gastropathy appears to be more appropriate.

Sclero-therapy is considered as double ended weapon. While it increases long term survival it increases the incidence of gastropathy.

Most of our patients received H_2 receptors antagonists & antacids. None of these agents had any significant effect upon either the gastritis or the bleeding. This tends to support the hypothesis that it is congestion rather than erosion which is the major factor damaging the gastric mucosa. The rational approach to treatment is therefore a reduction of the portal venous pressure which should thus reduce the congestion in the gastric mucosa.



SUMMARY AND CONCLUSION

We studied 40 cases with recurrant gastrointestinal bleeding due to oesophageal varices in bilharzial hepatic fibrosis. Our cases were selected from Benha University Hospital.

- We divided the cases into two groups:
  1) 20 patients with bilharzial hepatic fibrosis without sclerotherapy (19 male and one female) Their age ranged from 22-65 years with the mean of 39.5 .
  2) 20 patients with bilharzial hepatic fibrosis after sclerotherapy (all of them were males) Their age ranged from 28 - 64 with the mean of 46.5.

- All patients examined clinically and endoscopically, biopsies were taken from their stomach.

- From history and clinical examination, all patients were presenting by upper gastro-intestinal symptoms.

- Our case were subjected to through history taking and medical examination.

- Routin laboratory investigations were performed to overy case and liver biopsy were done.

- Haemoglobin percent was increased in all patients, after sclerotherapy mean value was 67.85 % in group one and 75.6 % in group two.

- RBC'S count was increased also in all patients, after sclerotherapy. mean value was 3.792.500/Cu ml in group one and 4.537.500 in group two.

- Endoscopic examination reveiled oesophageal varices of different degree in group one but in group two we reveiled oesophageal varices of different degree, one case of diverticulum of the lower oesophagus, one case showed active bleeding and one case showed hyperaemis mucosa.

- In the stomach there were gastritis in 35 % of cases 65 % were free and in group two. there were in group one gastric varices in 5 % of cases congested gastric mucosa in 20% of cases and 80 % of cases were free.

- Histopathological examination reveiled in the fundus of group one and group two were columnar metaplasia in 40 %, 50 % of cases, chronic superficial gastritis in 20 %, 15% of cases, slight atrophic gastritis in 5 %, of cases, congestion in 40%, 75% of cases, oedema in 10%, 30% of cases, cellular infilteration in 25%, 20% of cases and fibrosis in 15%, 25% of cases.

- We found in the antrum of group one and group two columnar metaplasia in 15 %, 15 % of cases, no cases of congestion, oedema in 10 %, 15 % of cases, cellular infiltration in 10 %, 15 % of cases and fibrosis in 5% , 10% .

- We concluded that patients with gastric lesions in bilharzial hepatic fibrosis may present by upper gastro-intestinal symptoms which may related to these change. The changes in our work were congestive gastropathy more than inflammatory.

Also we concluded that the use of sclerotherapy for bleeding of oesophageal varices combined with

regular endoscopic follow up has provided a unique apportunity to study the progression of changes occuring in the gastric mucosa.

We conclude that ectasia was more prominent than gastritis, this difference was more prominent after sclerotherapy, hence the term of congestive gastropathy appears to be more appropriate.

Also, we conclude that haemorrhagic congestive gastropathy is so important as gastro-oesophageal varices and the basis of the therapy must be the early instituation of measures which effect a reduction of portal pressure. Sclero-therapy is considered as double ended weapon. While it increases long term survival it increases the incidence of gastropathy.

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الملـخص والاستنتـــاج

تم دراسة اربعـون مريضا بنزيف الجهاز الهضمى المتكـرر بسبب دوالى المرىء فـى مرض تليف الكبـد البلهارسـى • تم اختيار الحالات من المستشفيات الجامعيه ببنهـا ، تم تقسيم الحالات الى مجموعتين •

المجموعه الاولى : عشرون مريضا بدون استخدام العلاج التصلب •

المجموعه الثانيه : عشرون مريضا بعد استعمال العلاج التصلبى لدوالى المرىء بواسطة الحقن المنظارى ب الايثانول أمين أوليات •

والهـدف من البحث هو محاولة للتعرف على أضرار المعده فى تليف الكبد البلهارسى اما التهاب المعده او احتقـان المعـده والتغييرات فى الغشاء المبطن للمعـــده بـعد العلاج التصلب باستخدام المنظار والدراسه الهستوباثولوجيا لعينه من الغشاء المبطـن للمعـــده •

تم فحص جميع المـرضى اكلينيكيـا وبالمنظار الليـفى الضوئـى وقد أخـــذت العينـات من الغشاء المخاطـى المبطـن للمعـــده •

تم صبغ العينات بمادة الهيماتوكسلين والأيوزين والألشن بلص والرتكيولين وكانـــت نتيجـة الفحـوص كـالاتـــى :-

١) نسبة متوسط الهيموجلوبين فى المجموعه الثانيه بـ عد العلاج التصلب لدوالى المـرىء كانت ٨ر٧٤٪ وفى المجموعه الاولى ٨٥ر٦٧٪ •

٢) عدد كرات الدم الحمراء زادت فى جميع المرضى فى المجموعه الثانيه عن المجمـوعـه الاولـــى •

٣) وجد ان المرىء بالفحص المنظارى توجد به دوالى بدرجاته المختلفه فى المجموعه الاولى وفى المجموعه الثانيه أيضا .

٤) بينت صورة المعده بالمنظار الليفى الضوئى وجد احتقان بالغشاء المبطن للمعده فى ٢٠ ٪ ودوالى بالمعده فى ٥٪ من الحالات فى المجموعه الاولى ، احتقان بالغشاء المبطن للمعده فى ٣٥٪ من الحالات فى المجموعه الثانيه .

٥) بالفحص الهيستوباثولوجى لجدار المعده فى الجزء القاعى فى المجموعه الاولى والثانيه على التوالى :-

- تغير معوى فى ٤٠٪ ، ٥٠٪ من الحالات .
- تقرح سطحى مزمن فى ٢٠ ٪ ، ١٥٪ من الحالات .
- التهاب ضمورى فى ٥٪ ، ٥٪ من الحالات .
- كما وجد احتقان بالاوعيه الدمويه فى ٤٠٪ ، ٧٥٪ من الحالات واوديما فى ١٠٪ ، ٣٠٪ من الحالات .
- ارتشاح خلوى فى ٢٥ ٪ ، ٢٠ ٪ من الحالات .
- تليف فى ١٥٪ ، ٢٥ ٪ من الحالات .

وجد ايضا فى الجزء البوابى من المعده فى المجموعه الاولى والثانيه على التوالى :

- تغير معوى فى ١٥٪ ، ١٥٪ من الحالات .
- ارتشاح خلوى فى ١٠٪ ، ١٥٪ من الحالات .
- تليف فى ٥٪ ، ١٠٪ من الحالات .

ومما سبق استنتج أن :-

١- تحسن صورة الدم بعد العلاج المصلب مع زيادة نسبة التغيرات الهيستوباثولوجيه فى جدار المعده .

ب- وجد ان التغيرات فى هذا البحث هى احتقان جدار المعده اكثر من الالتهاب المعدى وقد استخلص ان استعمال العلاج المصلب لعلاج دوالى المرىء النازفه مرتبط بالمتابعه المنتظمه بالمنظار اثبتت انها الطريقه المثلى لدراسة تطورات التغير فى الغشاء المبطن للمعده •

وايضا استنتج انه حالات احتقان المعده النزفى من الاهميه مثل حالات دوالى المرىء المعديه، وان اسس العلاج يجب ان تكون الاجراءات الاوليه والتى تؤدى الى خفض الضغط البابى وليس بمضادات الحموضه كما هو متبع الآن •

يعتبر العلاج المصلب لدوالى المرىء كسلاح ذو حدين بينما يحسن حالة المريض الصحيه فهو يزيد من تضرر الغشاء المخاطى •

ولقد استنتج ايضا ان تمدد الاوعيه الدمويه الاحتقانى كان أكثر وضوحا من الالتهاب المعدى وهذا الفرق كان اكثر وضوحا بعد استعمال الحقن التصلبى لدوالى المرىء وهنا يكون اصطلاح احتقان المعده مناسب •

أضرار المعدة فى إرتفاع الضغط البابى فى تليف الكبد البلهارسى

رسالة مقدمة من الطبيب

مجدى عبد الرازق حسن

بكالوريوس الطب والجراحة

كجزء متمم للحصول على درجة الماجستير فى

الباطنة العامة

تحت إشراف

الأستاذ الدكتور

محمد أبو العينين

أستاذ الأمراض الباطنية

كلية طب بنها

الدكتور

أكرم القديوى

أستاذ مساعد الأمراض الباطنية

كلية طب بنها

الدكتور

محمد عبد الوهاب

مدرس الجراحة العامة

كلية طب المنصورة

كلية طب بنها

جامعة الزقازيق

١٩٨٧