Surgery of Vascular Distortions in Cirrhosis of the Liver *

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IN PREVIOUS STUDIES we have called attention to the presence of an increase in the oxygen saturation of the blood in the portal vein when compared to that in the general systemic venous circulation,[16, 19] an observation that had been made previously by others.[2, 3, 18] We offered evidence that this increase in oxygen saturation of portal venous blood was due to the presence of normally occurring arteriovenous anastomoses in the abdominal viscera, the stomach and spleen being particularly studied in our experiments. These arteriovenous anastomoses would when open, allow highly saturated arterial blood to pass directly into the venous system without flowing through the capillary bed.

While such a phenomenon would certainly explain the increase in the oxygen saturation of the venous blood when such shunts were open, at the same time there should also be lowering of the functional activity of the cells deprived of adequate blood supply.

Experiments were devised to test this hypothesis and such was found to be true. In a series of observations detailed elsewhere [16] the ability of the gastric mucosa to secrete acid could be correlated with the adequacy of its capillary perfusion. The capillary bed could be shown to be open when histamine was administered or the vagus nerves were stimulated. At such times there was an increase in the secretion of hydrochloric acid. Conversely, with the injection of adrenalin the capillary bed was bypassed to a large extent by the blood flow and acid secretion ceased or diminished even though histamine continued to be injected. At such times the oxygen saturation of the venous blood coming from the stomach approached arterial levels.

It seemed proper to interpret these observations as demonstrating that when hydrochloric acid is secreted by the stomach, the energy requirements are so great that increased blood flow through the stomach with an increase in perfusion through the mucosal capillary bed is essential. Conversely, when acid is not being secreted, much of the capillary bed in the mucosa is not perfused, the blood being shunted directly from the artery to the vein in the gastric submucosa.

Such a concept of the relationship between cellular function and the provision of cellular substrate serves to explain a number of physiologic phenomena in other parts of the body not the least of which is the mechanism by which a relatively small amount of blood can supply adequately a huge capillary bed. Transport to a cell membrane, which is equally as important as transport across a cell membrane, will be subject to local and to systemic modification as this sluice-like mechanism is affected.

Our observations demonstrated also that the control of the sphincteric mechanism was in all probability humoral in view of the fact it was responsive to some of the catechol amines and to histamine. Other substances normally present in the circulation that could alter smooth muscle irritability could also obviously be concerned. There are many such substances. Since

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most of these are sensitive to the integrity of hepatic cellular function either in their formation or in their degradation, certain types of hepatic damage could conceivably affect control of this sluice-like mechanism adversely. Information is not too complete regarding the metabolic control of such substances, and it is already known that other tissues than the liver are involved in their synthesis and breakdown. Nevertheless, the liver in many instances plays an important role and in so doing acts as a factor controlling capillary perfusion.

Realizing the presence of such a shunting mechanism and the nature of its control it became of great interest to us when we noted that the oxygen saturation of the blood in some of the radicles of the portal vein was considerably increased in cirrhosis of the liver, much beyond that normally found. At times it may approach that encountered in the arteries of such patients. This is suggestive, therefore, that in cirrhosis of the liver, controls normally at play in regulating the shunt of blood from arterioles to venules are disturbed.

In recent years arteriovenous shunts have been demonstrated to occur normally in many diverse tissues. In most instances this distribution seems to be related to a phasic need for energy. In certain types of liver injury such as that seen in cirrhosis it would be reasonable to anticipate some interference in the function of certain tissues even more remote than the portal bed, and this interference should be apparent either as a decrease in a specific function or evidence of a compensatory mechanism should be present. Observations by ourselves and by others have shown that such may be true. These have previously been recorded [20] and will only be summarized here.

Most patients with a considerable degree of cirrhosis of the liver present evidence of a hyperkinetic circulation. Universally there is tachycardia. Often one may note capillary pulsation. Frequently there is present a precordial thrust. The extremities are warm and flushed, and the palms and soles are erythematous (liver palms). In patients with cirrhosis there is a lowered level of oxygen saturation in the arterial blood which along with an increase in venous oxygen saturation results in a lowered arteriovenous difference.[12]

This arterial desaturation can be marked enough to cause cyanosis and clubbing of the fingers particularly in so-called juvenile cirrhosis.[17] There is also a decrease in peripheral vascular resistance. There is an increase in the cardiac output, in one of our patients reaching the enormous level of 17 liters a minute. There is an increase in blood volume, the plasma being in greater abundance than the red cell mass and although the latter is also present in greater amount than normal, one gets a superficial picture of anemia. It is an "anemia," however, that is usually refractory to iron therapy. The red cells are not stable, the half-life often being considerably lessened. On two occasions we have been able to hear a bruit over the spleen and in the majority of instances with careful examination a thrill may be detected in the splenic vein at laparotomy. Small areas of telangiectasia involving vessels of the skin (spiders) are a common observation.

Among the viscera that appear to be concerned particularly with this altered vascular mechanism in cirrhosis is the spleen and this has been the subject of a recent report by us.[20] Certain of the observations that we were able to make are pertinent to the present consideration. We could, for instance, find little evidence to support the concept that the splenomegaly of cirrhosis was associated with obstruction of the blood flow through the splenic vein. Experimentally while obstruction of the splenic vein resulted in initial splenomegaly and elevation of splenic venous pressure, this was followed shortly by a decrease in splenic size to well below that of normal, the decrease in size being greater

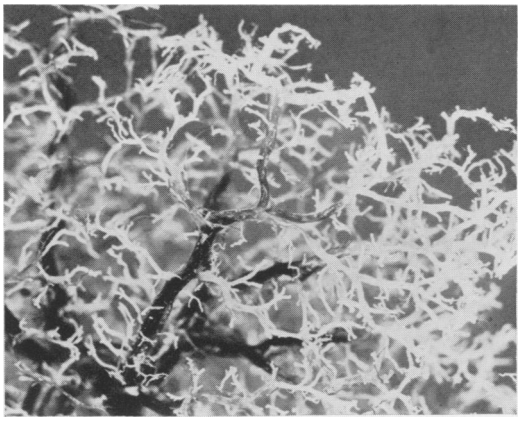

FIG. 1. Liver from patient with cirrhosis. Hepatic artery perfused with red vinyl acetate and portal vein with white after which liver was digested by concentrated acid. Here one sees dark plastic filling larger portal radicles suggesting A-V connections more centrally located than those in the perilobular area.

the fewer collaterals that were established. There was also noted to be a decrease in splenic arterial flow and splenic venous pressure in proportion to the degree of obstruction. We have never encountered the so-called extrahepatic obstruction of the splenic vein in cirrhosis but rather have been impressed with the evidence of increase in flow.

That this increase in splenic venous flow in cirrhosis was due to a direct connection between the arterial and venous radicles of significant size within the spleen was demonstrated by the lessened arteriovenous oxygen saturation differences, the passage of glass spheres as large as 600 micra in diameter from splenic artery to splenic vein and the actual demonstration of abnormal arteriovenous connections within the spleen by the injection of colored vinyl plastic into the vascular bed with subsequent digestion of the splenic substance with acid. All these observations support the thesis that splenic venous flow is increased in cirrhosis due to direct connections between the arterial and venous radicles within the spleen. Whether similar abberations could be present in the liver and stomach next needed clarification.

Morphologic Observations

While morphologic changes in both the human liver and the stomach have been investigated, the former will be more fully described at a later time. It is apparent that in cirrhosis the arterial blood supply to the liver is considerably increased. Connections between the hepatic arterial circulation and the portal venous are rich and may take place peripheral to the perilobular areas. The pattern of the mixing of colored vinyl plastic when injected into the hepatic artery and portal vein of patients dying with cirrhosis is suggestive of this and can be noted in Figure 1. These injections were made after a modification of the method used by Narat *et al.*[13] and by Liebow *et al.*[9] The viscosity of the material prevents it from penetrating into the perilobular areas. White plastic was injected into the portal vein and dark (red) into the hepatic artery. The central or hepatic radicles of the portal vein are filled with the white plastic while the larger or more peripheral branches are intermixed with the arterial injections.

Because of technical reasons injection of the gastric and esophageal vascular bed in patients dying with cirrhosis has not been satisfactory as yet. Morphologic observations are therefore limited to gross and microscopic study. Those observations made at the operating table with direct vision of the vascular lesions in the opened stomach and esophagus have been most valuable. Most of the vascular changes noted have been submucosal.

In the esophagus one sees large serpigenous vessels usually three or four in number appearing at the cardiac portion of the esophagus and extending upward for a distance of about 10 cm. In those patients coming to surgery we have been impressed with how seldom we have encountered bleeding from these vessels. Occasionally there has been erosion noted but in most instances the massive bleeding has come

from the upper stomach. Microscopic studies have been made of these esophageal vessels using formalin fixation with hematoxylin-eosin, the Verhoeff-Van Giessen stain for elastic tissue, Masson's hematoxylin, acid fuchsin, ponceau de xylidine and aniline blue for smooth muscle and connective tissue elements, and Gridley's ammoniacal silver and gold chloride stain for reticulin. On occasions when the esophagus has been sectioned longitudinally, an unusual vascular arrangement has been noted.

This is illustrated in Figure 2 which represents almost the entire vessel visible in the block of tissue. On one side it presents a thick muscular wall in which elastic fibers can be identified. This muscular wall rapidly gives place to what is chiefly collagen in which one may identify sparse smooth muscle cells and only occasional short elastic tissue fibers. Such a structure is highly suggestive of an arteriovenous junction and the collection of muscle in the center could represent a hypertrophic and distorted sphincter mechanism. The structure resembles that described by Morris [11] as being found accidentally in a set of class sections of the lower end of the esophagus. In his report he referred to a previous description of Conti and Passarelli of both direct and coiled arteriovenous anastomoses in the human esophagus.[7] Such a morphologic observation as we have noted above becomes significant when it is realized that the oxygen saturation of the venous blood from this area of the esophagus in such a patient with cirrhosis is close to that of arterial.

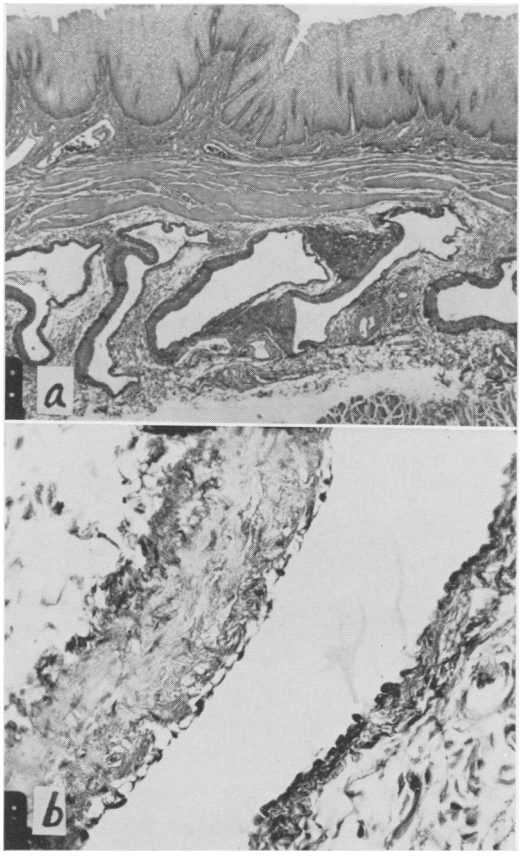

FIG. 2. a. Section through terminal esophagus of patient with cirrhosis. Note the thick muscular wall of the vessel on one side and the thin wall on the other. b. Higher magnification of area shown in 2a. On the left there is a thick muscular wall with elastic fibers apparently representing distorted internal and external elastic lamina. The wall on the right has only a few bundles of collagen beneath the endothelium.

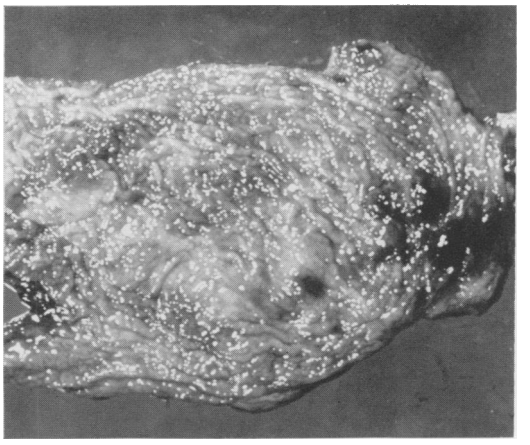

FIG. 3. Angioma-like vascular formations in the fundus of the stomach from a patient with cirrhosis. Blood was noted to be spurting from one of these areas.

In the stomach the vessels have appeared grossly to be of two main configurations. Both are common. In one (Fig. 3), they appear as angiomas varying from a millimeter in size to several centimeters. These are usually multiple and are as a

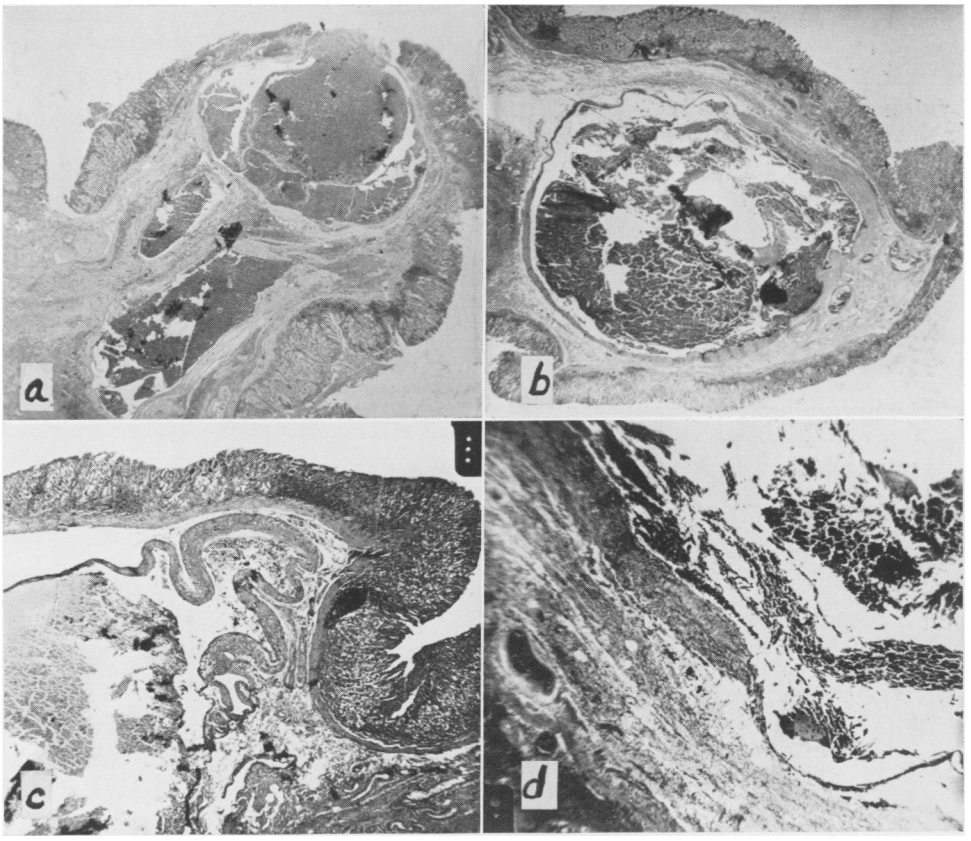

FIG. 4. Four different areas from specimen shown in Figure 3. a. Erosion of mucosa with bleeding (from × 10). b. Large submucosal vessel showing thick muscular wall on one side, continuous with wall showing only endothelium with thin layer of collagen beneath (from × 10). c. Vessel showing thick muscular wall with adjacent thin wall containing neither muscle nor elastic tissue. Note the atrophic mucosa overlying (from × 40). d. Higher magnification showing transition of the vascular wall at what is probably the site of an A-V anastomosis (from × 40).

rule located in the fundus, not far from the esophageal aperture and often partially surrounding this opening. We have not encountered this vascular formation distal to the body of the stomach. Bleeding is usually from an erosion of the mucosa over one of these angioma-like formations. We have operated upon five patients while active bleeding was in progress from such a vascular formation and in each instance the bleeding was controlled by ligation of the artery supplying blood to this area. Usually it has been an upper branch of the left gastric artery. In the specimen illustrated in Figure 3 the blood spurted and was observed to be arterial.

The microscopic picture shown in Figure 4 is taken from sections cut through several of such angiomatous formations. Here again one notes a muscular wall on one side and a dense fibrous wall on the other suggesting the site of arteriovenous anastomosis. In other areas both the arteries and the veins are larger than those found under normal circumstances and the vascular walls are thicker.

The other type of vascular formation frequently encountered in the stomach is illustrated in Figure 5a. While these areas, to a certain extent, resemble those described above, they are much smaller as can be noted from the magnification in the

gross photograph. The microscopic section noted in Figure 5b is taken through one of these sites and the thickness of the vascular walls is impressive. These also probably represent the sites of distorted arteriovenous anastomoses. These areas are difficult to identify at autopsy but can be seen easily at surgery if the mucosa overlying them has not been traumatized. They stand out as small pink areas not elevated and with clear-cut margins. While they are frequently the location of mucosal erosion and bleeding, they also may be associated with the more classical type of peptic ulceration which in our experience has generally been located at the junction of the esophageal and gastric mucosa, on the lesser curvature side.

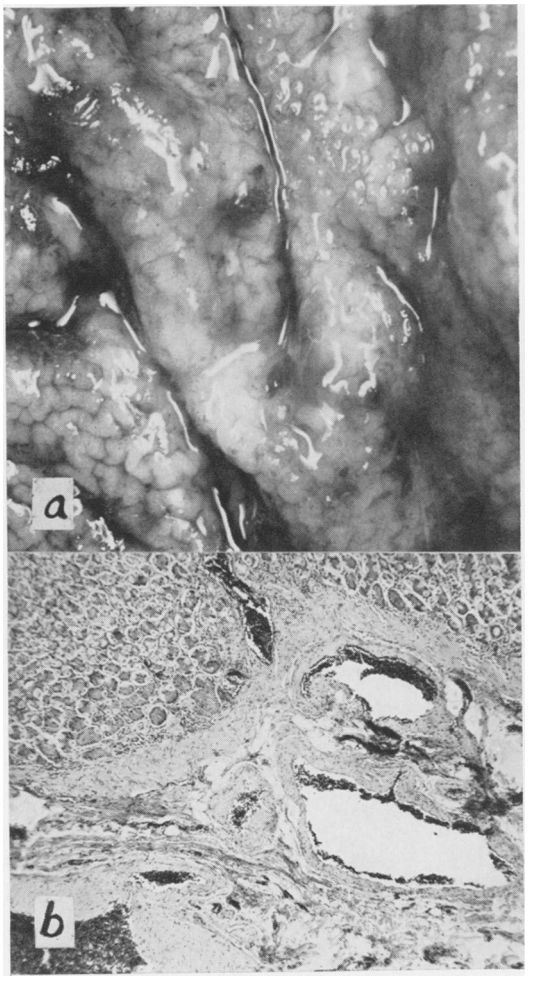

FIG. 5. a. Small angiomatous areas commonly seen in the mucosa of the stomach of patients with decompensated cirrhosis. b. Section taken through one of the vascular areas illustrating an unusually thick arterial wall in the lower left with vessels with walls not so thick on the right. This probably represents an A-V fistulous area with hypertrophic musculature showing early decompensation.

TABLE 1. *Difference in Saturation Between Control and Cirrhotic Patients*

	Mean Oxygen Sat. %	Standard Deviation	Standard Deviation Mean	P value Diff. Mean
Arterial				
Control	96	2.66	<1	<.01
Cirrhotic	93	3.9	<1	
Splenic vein				
Control	88	4.9	2.2	.04
Cirrhotic	71	15.9	6.0	
Gastroepiploic vein				
Control	90	4.7	1.4	.02
Cirrhotic	81	10.6	3.3	
Hemiazygos vein				
Control	72	12.05	2.2	.04
Cirrhotic	88	2.17	1.25	
Hemiazygos vein				
Before resection	88	2.17	1.25	.05
After resection	79	7.07	5.00	

Physiologic Observations

As a correlate to the anatomical data given above, physiologic and biochemical studies have been made on a number of patients entering the hospital because of variceal bleeding as well as on other patients with a normal hepatic mechanism who were used as controls. Experimental animals have also been used.

During laparotomies for duodenal ulcer, cholecystitis, thrombocytopenia, and thoracotomy for pulmonary resection, samples of arterial blood and blood from the splenic vein, a gastroepiploic vein or the hemiazygous vein were drawn. The oxygen saturation was determined with the Water's double oximeter or by modification of the Holling method with a spectrophotometer. Twenty-three such patients were studied. In 22 patients operated upon with

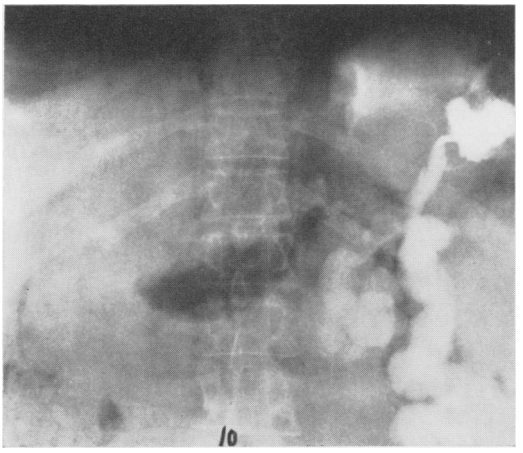

FIG. 6. Splenoportogram illustrating huge tortuous branches of the splenic vein. Note also the absence of a clear-cut hepatogram. There was no obstruction to the splenic vein at operation, an unusually rapid flow being present with a palpable thrill in the vein. Thus, even apparent obstruction was not confirmed.

cirrhosis and varices of the stomach and esophagus similar samples were drawn. The results are shown in Table 1.

The arterial saturation is significantly lower in all of the cirrhotic patients with p value of less than .01 (Table 1). This is consistent with the findings of arteriovenous shunting between the portal venous and pulmonary venous systems as has been so nicely shown by Fritts *et al.*[8] The saturation of the venous blood in the splenic and gastroepiploic veins was lower in the cirrhotic than the control group. Part, but not all, of this difference between the control and cirrhotic groups can be explained by the low arterial oxygen saturation in the latter. Cooling and manipulation of the stomach is reported by Barclay[1] and Zweifach[21] to cause the arteriovenous shunts to open and this may explain the high control venous saturations. In addition, the standard deviation of samples from the cirrhotic group was high due to some low figures in patients in poor condition. The blood from the hemiazygous vein which directly drains the vascular bed of the lower esophagus was significantly more saturated in the cirrhotic patients. There was no significant change in the venous oxygen saturation before and after the operative procedures described below except that the mean hemiazygous oxygen saturation fell 11 per cent (p value = .05).

Resting cardiac output determinations were performed by the dye dilution technic in three of the patients with cirrhosis and in all of these the cardiac output was elevated, in one more than twice that predicted.

In a group of dogs to be reported in detail elsewhere it was demonstrated that obstruction to the portal venous system led to a fall in cardiac output that was more profound than if the vena cava below the inlet of the renals was obstructed. This was associated with no change in the blood histamine levels, but as we have shown previously, led to a fall in portal venous oxygen saturation.

Pressures in the portal venous bed in the cirrhotic patient were in the normal range in half, and in the other half elevated. The highest value was 41.5 cm. of water. In those with satisfactory preoperative and postoperative measurements all but one returned toward the normal range. Pressures

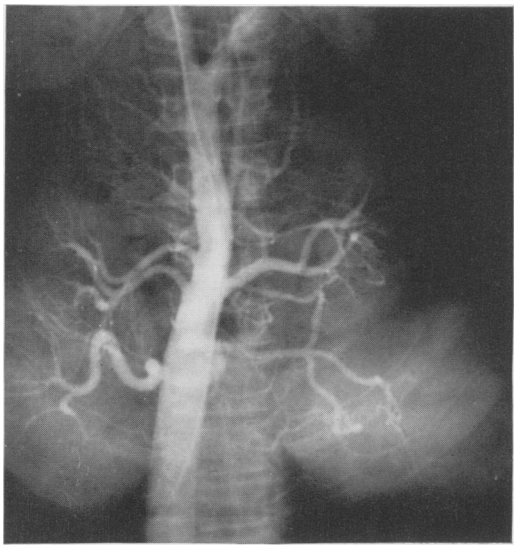

FIG. 7. Arteriogram showing enlarged splenic and hepatic arteries.

TABLE 2

Operations	No. of Patients*	Hospital Deaths	Recurrent Hemorrhage	Apparent Recovery	Remaining Liver Disability	Late Death Other Cause
I						
Resection of lower esophagus, upper stomach and spleen	2 (1)			1	1	
II						
Sleeve resection of upper stomach and splenectomy with and without plication of esophageal varices	8 (2)	3	3	1	1	
III						
Plication of varices only	7 (6)	5	2			
IV						
Splenectomy	3	1	1		1	
V						
Splenectomy, resection of greater curvature of stomach with ligation of blood supply**	16 (5)	5	1	8		2

* Numbers in parenthesis represent number of patients of total operated upon during acute bleeding.
** Follow up patients who survived ranges from 1 month to 2 years with mean 8 months.

were recorded by means of a Statham strain gauge manometer as has been previously described.

A total of 32 patients have been operated upon for some complication from cirrhosis at the North Carolina Memorial Hospital. All but four had had episodes of bleeding and 15 of the operative procedures were done for uncontrollable hemorrhage. In all the patients who were operated on for acute bleeding the source was ulceration or erosion in the cardia of the stomach. The blood was bright red and spurting under apparent arterial pressure. A number of patients had spleno-portograms and all of these showed the major portion of the dye going to and through the liver, Figure 6. Arteriograms were done on four patients and in these the left gastric and splenic vessels were larger than in normals, Figure 7.

All patients in the series had elevated cephalin flocculation levels. The alkaline phosphatase level was elevated in 13 patients, the plasma bilirubin was up in 12 patients, and the BSP retention in 10 patients. In some patients all of these determinations are not available. The plasma albumin level in the patients who were not bleeding acutely was greater than 3.0 mg. per 100 ml. in 14, between 2.5 and 3.0 in four, and below 2.5 in four. In the acute bleeders, most of whom had had multiple transfusions, it was greater than 3.0 mg. per 100 ml. in two, between 2.5 and 3.0 in two, and less than 2.5 in three. The only laboratory test which is well correlated with surgical mortality is the total serum bilirubin, this representing as would be anticipated a hazardous state of liver function. A low serum albumin level was associated with some greater degree of liver failure than were the other tests but was not as critical as we had thought. Insofar as laboratory appraisal of liver function was concerned, many of these patients presented severe operative risks. Wherever it was thought the patient's condition could be improved by diet, etc., surgery was delayed. In many instances this could not be

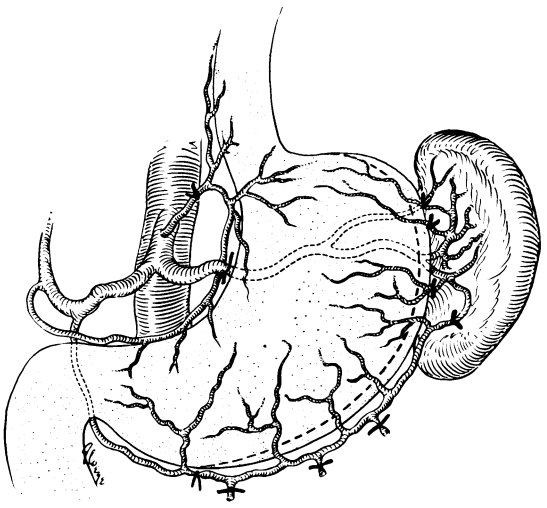

FIG. 8. A drawing of the type operative procedure carried out. Ties show points of ligation of splenic artery, left gastric artery, right and left gastroepiploic and shunt gastrics. Dotted line represents portion of stomach resected.

done, surgery being carried out on bad risk patients as a calculated hazard.

Treatment

If the variceal hemorrhage is a result of abnormal vascular formation resulting from distortion at the site of arteriovenous fistulas, a direct approach toward the involved area would be logical. While, as we have demonstrated, many viscera are often involved and, therefore, this lesion does not lend itself too well in its entirety to surgical correction; the problem of bleeding, nevertheless, can be met by a local approach. We have tried a number of operative procedures in the course of developing a satisfactory surgical solution to this problem (Table 2). The Phemister operation of esophagogastric resection with modifications was the earliest definitive procedure used. Two of these were done, one for acute bleeding and both patients were alive 24 months later, when last examined. This has the advantage of excising the bleeding area but previous experience with this procedure at another institution has shown the risk of gastric regurgitation and esophagitis. Sleeve resection of the stomach with splenectomy and plication of varices in the esophagus proved to be unsatisfactory with high mortality and high recurrence rate. Likewise, simple ligation of esophageal varices has been abandoned since this is rarely the site of bleeding. In essence the only good result in our experience is in one patient who had a ligation of a vessel leading to a varix high in the stomach as a secondary operation following a splenectomy and tubular gastric resection. This patient is well six years later. Previous experience with splenectomy alone has at times given excellent temporary improvement. Recurrences have been frequent, particularly when the blood supply to the stomach was undisturbed.

Group V incorporates the patients in whom we feel a definitive operative procedure has been used. Ideally this procedure drastically destroys the blood supply to the lower esophagus and upper stomach (Fig. 8). It includes splenectomy with proximal ligation of the splenic artery, shortly after it has crossed the aorta, ligation of the left gastric artery, and the right and left gastro-epiploic arteries and resection of the margin of the greater curvature of the stomach. This latter procedure insures occlusion of all of the vessels entering and leaving the stomach through the greater curvature. Where feasible, the bleeding area has also been excised. The marked variation in the arterial supply to the stomach as so beautifully demonstrated by Michels [10] requires that the procedure be carefully individualized to assure adequate ligation of this blood supply. This surgery has proved less shocking and technically easier when done transthoracically.

There are 16 patients in this group with all of the acute deaths occurring in patients operated on for uncontrolled hemorrhage. There was one late death due to coronary occlusion and two to liver failure. One patient bled again and as noted above in the discussion of plication; this patient is alive

six years after the second operation which in essence was the completion of the procedure that we now propose. The follow up is short on the eight others. This is in large part due to the difficulty in obtaining return visits from this type of patient. This follow up represents not time since operation but the time between the last operation and the last examination. Follow up by letter, we have considered too inaccurate. In appraising the value of the procedure, we have used as criteria of success the disappearance of esophageal and gastric varices by esophagoscopic and gastroscopic examination and by x-ray studies. While recurrence of bleeding of clinical severity has usually been associated with the reappearance of varices, varices may also be present without bleeding and hemorrhage, therefore, is not an accurate criterion. Disappearance of the varices is usually immediate, as shown by esophagoscopy during the operative procedure. All patients were examined esophagoscopically and by x-rays before discharge from the hospital.

Discussion

We have presented morphologic and biochemical evidence that the vascular distortions seen in the lower esophagus and upper stomach are not the result of centrally located venous obstruction but rather result from decompensation of the arteriovenous sphincteric control. The location of these vascular abnormalities is not diffuse over the entire portal system as would be expected if the etiology was an increase in portal pressure. This location is eccentric, fairly constant and obviously not haphazard. In explaining vascular dilatations in the region of arteriovenous fistulas some insight may be obtained by applying the physical formula of Laplace to this phenomenon.

Burton and his colleagues have called attention to this rule recently in several important papers in which the tension of the vascular wall has been studied in relationship to pressure and radius.[4-6, 14] These three variables relate to each other, the radius of the cylindrical vessel, the total tension in the wall and the difference in pressures across the wall (blood pressure minus tissue pressure). At equilibrium this can be expressed by the equation $T = p \times r$, where T equals tension in dynes per centimeter, "p" the transmural pressure in dynes per square centimeter and "r" the radius of the cylinder in centimeters. As will be noticed, the smaller the radius of the tube, the greater the pressure that can be accommodated without appreciably altering the tension in the wall. When this principle is applied to the vascular bed, it can be seen that the walls of the larger vessels become much more vulnerable to increases in intra-luminal pressures than do those of vessels of smaller diameters. If in cirrhosis the vascular problem was simply one of portal hypertension, the vein most vulnerable would be the portal vein itself. This, of course, is rarely the site of varicosity and never the site of spontaneous hemorrhage.

In discussing the formula as it applies to blood vessels, Burton calls attention to the presence of both passive and active tension in the walls, particularly of the smaller vessels. By passive tension is meant that chiefly derived from elastic tissue and collagen, tissues that possess a more or less inert elastic quality. Active tension is supplied chiefly by smooth muscle. Dilatation of the blood vessel by muscular relaxation must always be a hazardous procedure. A considerable dilatation of the vascular lumen at this site could easily lead to a loss of integrity unless there was concommitant a considerable increase in smooth muscle. Vascular sphincters would provide just such an unstable site. A considerable dilatation of the vascular lumen here could easily lead to a loss of competence of the vessel wall, there being little or no elastic tissue present. There are then two regions

of vascular instability as a result of an increase in the diameter of the vascular lumen, the largest veins and the precapillary sphincter area in the submucosa. As has been noted, the larger veins, such as the portal and the superior mesenteric, are not frequently greatly distorted in the presence of portal hypertension. Most consistently the varicosities that bleed appear in the submucosa of the lower esophagus and upper stomach. It is unlikely, therefore, that one is concerned here with a problem of simple portal hypertension. It suggests more the possibility of a defect in the system of active tension in the vascular walls, particularly in the sphincteric mechanism.

Localizing the lesion at the arteriovenous anastomotic site also has virtue in explaining the failure of adequate perfusion of the capillary bed of the stomach in cirrhosis. This failure of adequate mucosal perfusion could be responsible for the mucosal atrophy often encountered in cirrhosis as well as the areas of erosion previously described. Gastric acid secretion in the patient with cirrhosis is usually decreased. In three of the five patients upon whom acid studies were made by us before operation there was no free hydrochloric acid. In the two others the 12-hour fasting secretion was half that of normal. All of these patients exhibited a tendency to histamine refractoriness. This subject has recently been more completely studied by Ostrow *et al.*[15] They have noted that in patients with decompensated cirrhosis all of the gastric secretory parameters studied showed lessened activity. These included uropepsin excretion, blood pepsin level and gastric output of both acid and pepsin under basal circumstances and after histamine.

We have been unable to elicit a great amount of evidence of the existence of mechanical obstruction of the portal vein in so-called portal hypertension. Intrahepatic blood flow is most complex and the dynamic state of the intrahepatic circulation will apparently vary in different portions of the liver and at different times. More information is needed concerning this. We have operated upon only one patient with partial obstruction of the hepatic vein from a thrombus. In this instance while collateral venous circulation was apparent, the pressure in the portal vein was 18 cm. of water and the spleen was not enlarged. We have called attention to the fact that obstruction of the portal vein lowers markedly the cardiac output while in cirrhosis the cardiac output is increased. Portal obstruction also is associated with a lowered oxygen saturation of the portal venous blood while, as we have noted, this is elevated in cirrhosis. Frequently, a splenoportogram will present a shadow highly suggestive of obstruction of the splenic vein. Figure 6 illustrates such an instance. At operation no such obstruction was present, flow actually being increased. In all probability it is this increase in flow producing dilution of the contrast medium that produces the radiologic mimicry of obstruction. We have previously stressed the point that venous obstruction must be associated with decrease in arterial flow or circulatory catastrophy rapidly intervenes.

We would, therefore, explain the increase in portal venous pressure often seen in cirrhosis as due to the development of lack of control of the arteriovenous communications.

Before there was apparent a physiologic interpretation of the lesion producing the bleeding other than portal venous obstruction, we were concerned about the hazard of ligating what seemed to be a collateral circulation. But there were no adverse sequella noted following such ligation. Not only was chronic passive congestion of the portal bed not evident but upon secondary operation there was no reformation of the enlarged veins.

In the surgical procedure that we have employed, the vascular supply to the stomach and lower esophagus has to a large extent been destroyed. The right gastric

artery and branches of the right gastroepiploic artery supplying the antrum are left intact. This has not compromised too greatly the nutrition of the stomach although on one occasion a gastric ulcer has been noted postoperatively. The addition of splenectomy, we believe, is not only important because of the removal of the large arteriovenous fistula existing in the spleen but ligating the splenic artery just as it has crossed over the aorta further serves to lower the blood supply to the stomach. This is not an ideal operation for diffuse arteriovenous fistulae and it may be that with the passage of time and the persistence or exacerbation of liver decompensation recurrences will be noted. As yet only one patient has had a recurrence of his varices and this occurred following an acute episode of hepatic decompensation. The varix was small and was regressing when last seen as the liver recovered. Such regression of varices has been described on other occasions.

The surgical approach that we have recommended does result in a disappearance of esophageal varices that were present before operation as noted upon esophagoscopy, and it does this without seriously interfering with the nutritional state of the patient. It can be made a safe operation.

Summary

1. Evidence has been presented to support the thesis that the vascular abnormalities as well as the portal hypertension in patients with cirrhosis are due to changes in the region of the arteriovenous connections and are not caused by portal hypertension.

2. The alterations in these arteriovenous connections occur in many areas of the body.

3. An operative procedure is presented that is designed to control the arteriovenous fistulae in the stomach, spleen and lower esophagus.

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Discussion

Dr. David V. Habif: I think this is a very nice piece of work but I would like to express a word of caution about the recommended procedure. The operation seems to be an extension of that proposed many years ago by the late Howard Gray.

Our experience with attempting to control bleeding in those patients who cannot have a portacaval shunt embraces some 25 patients. A few years ago, we published our thought that an almost total gastrectomy was not effective in that five of seven patients had rebleeding. We recommended a partial esophago-gastrectomy, removing the lower third of the esophagus and upper 5.0 cm. of the stomach and interposing a segment of jejunum to prevent the regrowth of a rich collateral between the stomach and esophagus. Because we had seen varices in the middle third of the esophagus disappear following this operation, we believed it was adequate.

In a discussion of that paper, Drs. Cooley and Zimmerman expressed their opinion that where a shunt was not possible, middle as well as lower third esophagectomy should be done for cure. I think that time has proven them right.

We now have four patients who returned with bleeding from the middle third of the esophagus who had a lower third resection. One of these occurred seven years postoperatively and was so massive, it was impossible to save his life.

The maximum follow up of six years in Dr. Peter's paper is in our experience too short to draw strong conclusions.

We are of the opinion that an adequate, effective portacaval shunt is the best method of preventing bleeding from esophageal varices. For those who cannot have one of the standard shunts, the operation described independently by Marion, in France, and Clatworthy, Wall and Watman, in Columbus, should receive more attention. This consists of an anastomosis between the proximal end of the divided inferior vena cava and the side of the superior mesenteric vein. At the Presbyterian Hospital in New York Drs. Voorhees and Blakemore now have more than 25 patients where this shunt was performed with excellent results.

For those who cannot have any type of shunt, we are now resecting the esophagus at a point above the azygos vein in the light of our experience cited above.

Dr. Robert R. Linton: I hesitate to get up and discuss a paper before I give my presentation which is next; but having been interested in this subject for a number of years, and having certain opinions with regard to it, I thought it might be worthwhile expressing some of them, since they will not come out in my presentation.

I have enjoyed the presentation of Drs. Peters and Womack and think that they certainly may have explained some of the fundamental problems that are troubling us in the handling of these particular patients. I, together with Dr. Habif, however, question how long this type of procedure they describe will last. There is a certain homeostasis factor which takes place in the vascular system, as in the biochemical system of the body, and although you reduce blood flow to a part by ligating and dividing some of the arteries leading to it, I would suspect that after a matter of weeks or a few months, the previous hemodynamics will be restored.

The method I wish to recommend to you if you have a patient whom you cannot do a shunt procedure on, is to perform a transthoracoesophageal suture of the esophageal varices. It is the best procedure I know to control massive bleeding esophageal varices after they have been temporarily controlled by intragastric balloon tamponage. It is important to point out that not only can you suture these varices once, but it can be repeated again if necessary. I have done this on one patient and it is worth pointing out to you that it was much easier the second time than the first, since the esophagus takes on a certain fibrous texture that makes closure of it much more readily accomplished.

I would also like to recommend this procedure in preference to an esophagogastrectomy for