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THE SYNDROME OF PORTAL HYPERTENSION

SENIOR THESIS

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THE SYNDROME OF PORTAL HYPERTENSION

In the year 1818, Professor Puchett (1), of Heidelberg, stated that inflammation of the vena portae was so rare that he knew of no example of its occurrence. In a period of thirty-two years following this, much information had been collected; and among lesions of this system, several instances of lesions of the vena portae have been observed and recorded. Craigie, in 1841, recorded the pathological findings in a case of marked ascites associated with gastric hemorrhage. At the autopsy table he found that the portal vein was almost completely occluded by a firm elastic mass. This excited very little attention to the pathologies of the portal system except in the minds of a few men. The first demonstration of the fatal effects of acute obstruction of the portal vein has been attributed to Ore (2), who, in 1856, discovered that ligation of the portal vein in rabbits was consistently followed by death in a short time. Various theories including the production of toxins, loss of blood in the portal system and excessive stimulation of sympathetic nerve fibers in the neighborhood of the ligature had been offered as possible explanations of the fatal outcome. It has only been a matter of a few years since this question was definitely and conclusively explained by Elman and Cole (2).

The occurrence of fatal Gastro-intestinal hemorrhage due to cirrhosis of the liver has been known for many years,

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but the cases have never been collected, and the subject of some importance, is merely mentioned in most text books. Since Faurel, in 1858, reported the first case of ruptured esophageal varices in cirrhosis of the liver, this subject has been treated in several French theses but the conclusions reached were on two few cases.

The real study of portal congestion was carried on by Gilbert and his pupil Villaret in 1899. From this time on, due to their untiring efforts, the subject disclosed many factors which aided in the development and understanding of the complete picture of portal obstruction syndrome. In Gilbert's summary are the essential facts as recognized by the modern text book. His conclusions are summarized, as follows (3):- "Whenever there exists an intrahepatic vascular obstruction, there occurs a series of symptoms which have been grouped under the name of the Syndrome of portal hypertension. The fundamental features of the syndrome are as follows: 'Opsiuria' or delay in the elimination of urine, ascites, splenomegaly; hemorrhoids, gastro-intestinal hemorrhages, and the development of the collateral circulation as seen in the anterior abdominal wall. Of all the symptoms which constitute this syndrome, the earliest is opsiuria. The others vary in their order of appearance according to the type of case."

This short summary is the bases of my paper, and after having given these few high points I shall attempt to go back and develop the picture of the syndrome and carry it thru to its completeness as recognized today. My conception is that

the picture is by far not complete and holds the unfolded key to many problems of metabolism, growth and probably degenerative processes of the viscera.

(4) (5) The portal system of veins arise by means of a series of transformations which take place in the vitelline and umbilical veins of the embryo. The proximal ends of the vitelline veins, where they lie between the umbilicals, are early developed in, and invaded by, the growing liver. The columns of liver cells while not penetrating the endothelium, subject the vitelline veins to a process of fenestration by which the original channels are subdivided into innumerable smaller vessels or sinusoids, The sinusoids arising from the two vitelline veins inter-communicate to form a continuous network within the liver in which the vessels are larger in the afferent (portal) and efferent (hepatic) areas than in the intermediate zone.

The formation of the portal vein is effected by the disappearance of the portion of the right vitelline vein on the distal side of the dorsal connection and of the portion of the left vitelline upon the proximal side. The portal vein is joined by the superior mesenteric vein upon the left side of the duodenum and by the splenic vein behind it; the portion of the common vitelline vein beyond the junction of the superior mesenteric with the left vitelline subsequently disappears.

(6) In the adult, the portal system is composed of all the veins which have their origin in the walls of the digestive

tract below the diaphragm and includes also the veins which return the blood from the pancreas, spleen and gall bladder. It presents a marked peculiarity in that the system begins and ends in capillaries, the blood which it contains having entered its constituent veins from the capillaries of the intestine, stomach, and the other organs mentioned, and passing hence to the liver where it transverse another set of capillaries, by which it reaches the hepatic veins and so the heart. Coming as it does principally from the intestine, the portal blood is laden with nutritive material which has been digested and absorbed through the intestinal walls, but is not as yet in a condition, so far as its constituents are concerned, suitable for assimilation by the tissues. To undergo the changes necessary for its conversion into assimilable material it is carried by the portal vein into the liver, and as it passes thru the capillaries of that organ it undergoes the necessary modifications. In other words, the portal vein stands in a similar relation to the liver that the pulmonary vein does to the lungs. Its purpose is not to convey material to the organ for its nutrition, but to carry to the liver crude material upon which the organs may act, elaborating it and returning it to the circulation in an assimilable form.

Only a superficial consideration of these factors, makes one realize the extreme delicacy and vital importance of the system. It also makes one wonder that with only the slightest modification of the normal mechanism just how the normal physio-

logical processes of the body with all its delicate dependencies could carry on when possibly such a disturbance may eliminate and deprive the cellular structures of some linking component. It becomes still more important when we consider that the beginning of the portal system in a capillary bed into which absorption must take place and upon which cellular normality of the body is dependent. It is not hard to conceive of some process, either mechanical or inflammatory taking place in the thin walls of such capillaries producing sufficient changes in semi-permeable membrane to set up a selective absorbing process. If such should occur, and the changes were such that certain vital components, vitamin and elements were excluded from the digestive tract, then it would follow that metabolism and growth would be disturbed. One may also wonder why, if some necessary substance is required by the cells of the body for normal physiology, mitosis and growth; exclusion of such substance might upset the balanced mitosis and growth so that malignant hyperplasia take place.

Grossly, the portal vein is formed behind the head of the pancreas by the union of the superior mesenteric and splenic veins, the latter receiving the inferior mesenteric vein shortly before its union with the superior mesenteric, the two veins unite almost at right angles, and from their point of union the portal vein passes obliquely upward and to the right, along the free edge of the lesser omentum, towards the porta of the liver. There it divides into two trunks, of which the right is

the larger and shorter and quickly bifurcates into an anterior and posterior branch. It is distributed to the whole of the right lobe and to the greater part of the caudate and quadrate lobes. The remainder of these lobes and the left lobe receiving branches from the left trunk. The trunks of the vein enter the substance of the liver and divide in a more or less distinctly dichotomous manner to form interlobular veins, which, as their names indicate, occupy a position between the lobules of the organ, and give off capillaries which traverse the lobule and empty into the intralobular veins which are the origin of the hepatic veins.

The walls of the portal vein contain a considerable quantity of muscle tissue and it is destitute of valves.

In order to understand the extra portal faction which is at times responsible for obstruction, a review of the relationships to other structures is desirable. At its origin, the portal vein lies behind the head of the pancreas and to the left of the inferior vena cava. As it ascends it becomes to lie behind the first portion of the duodenum and then between the two layers of lesser omentum. In this latter portion of its course it is associated with the hepatic artery and the common bile duct, both of which lie anterior to it, the artery to the left and the duct to the right. It enters the porta toward its right extremity.

The principal tributaries of the portal vein are:

1 - Superior mesenteric; 2 - Lienal splenic; 3 - Inferior mesenteric; 4 - Coronary; 5 - Pyloric; 6 - Cystic veins. In addition

to these principal tributaries, the portal vein, or its branches within the liver, also receive a number of small veins which have their origin in the falciform ligament of the liver and in the lesser omentum. At the porta some parumbilical veins are received and ascend the anterior abdominal wall along with the round ligament. The subject matter of this paper does not demand a detailed description of all the branches of the portal vein. It appears sufficient to know the portal in rather completeness, remembering that there are seven tributaries and that the occlusion of any one does enter into the picture of portal hypertension but not included in the scope of hypertension syndrome.

Before discussing the possible routes of collateral circulation in the portal system, a consideration of anomalies in this region is important. Altho quite rare in view of the scant literature on the subject, it becomes a possibility to have **venous** anomalies which may distort the clinical picture when confronted with a case of portal hypertension. (7) Rothe, a German surgeon, reported many years ago a case in which one of the large hepatic trunks from the right lobe of the liver, terminated not in the inferior vena cava or right auricle but in the base of the right ventricle. (7) Morgagni reports a case of the hepatic vein joining the inferior vena cava after it had pierced the diaphragm. The umbilical vein has occasionally been found patent for a variable distance below the liver. It may communicate with the epigastric and thus establish a collateral circulation. (8) J. A. Russel reports two cases of persistent communication between the umbilical and portal veins.

(8) F. Champreys describes a communication between the external iliac and portal veins thru the epigastric and umbilical veins.

(7) W. Krouse mentions a number of cases of communication between the portal vein and the iliac veins by means of a patent umbilical, connected directly by a branch, or thru the epigastric vein. These communications are all due to persistent fetal conditions and are much more apparent when there is any obstruction to the portal circulation.

Coincident with the obliteration of the portohepatic venous pathway, collateral channels are gradually opened between the portal and caval systems. Such channels are, of course, developed at points at which contact between the two systems already exist potentially. The anastomatic channels fall logically into three groups according to the situation.

1. (10) At the two situations in the gastro-intestinal tract where absorbing epithelium comes in contact with protective epithelium, that is, the cardia of stomach and the anus. The former represents the site of anastomoses of the coronary vein of the stomach with the intercostals, azygos minor and diaphragmatic veins of the caval circulation, here producing esophageal varices. At the latter, the superior hemorrhoidal vein of the portal circulation anastomoses with the middle and inferior hemorrhoidal veins of the caval circulation. Occasionally hemorrhoids develop but are usually not clinically important.

2. At the site of the obliterated embryonic circulation, that is, the falciform ligament containing the parumbilical veins.

3. At all situations within the abdomen where the gastrointestinal tract, its appendages or the glands developed from it, become retroperitoneal developmentally or adherent to the abdominal walls pathologically. This includes the duodenum, small intestine, colon, omentum, spleen and pancreas, containing the veins of Retzuis and the liver with its accessory vein of Sappy both establishing an anastomoses between the portal and caval veins. These channels may be increased artificially by operative procedures, as for instance, by producing adhesions between the liver and diaphragm or by attaching the omentum to the anterior abdominal wall as in the Talma-Morrison operation. At autopsy, many cases are observed in which there is not enlargement of the collateral circulation and in others, which have not been associated with ascites, this circulation is increased. The retroperitoneal veins may be enlarged while the parumbilical veins remain normal in size. It may be just the reverse and the marked caput medusae is produced. Occasionally, a single vein may attain the size of the smallfinger and run directly between the liver hilum and the epigastric vein at the umbilicus. (9) Veins about the gall bladder, lesser omentum and lesser curvature sometime connect with the para-umbilical veins and thereby with the caval system of the anterior abdominal wall.

With this background of development, anatomy of the portal system with its possible routes of collateral circulation, the relationship of the vena portal to the surrounding structures and function of this venous pathway; the way is clear to consider the causes of portal obstruction and the mechanism in which it is produced.

The portal system may be obstructed by (a) tumors or swellings involving the liver; (b) Enlargement of gall bladder from new growths or concretions; (c) tumors of contiguous structures as disease of lymph glands in the portal fissure or between layers of the lesser omentum or carcinoma of the head of the pancreas; (d) Disease of liver tissue - especially cirrhosis; (e) Valvular disease of the heart.

The consequences of obstruction to the portal system are various but may be understood by referring each symptom to its anatomical basis in obstruction of one or the other of the venous tributaries. In the clear cut case of obstruction just what happens? The chief results are: (1) Enlargement of liver itself, at first congestive, later from hyperplasia. Diminution in the quantity of bile or alterations in its character may cause constipation and indigestion; or escape of its coloring matter and its absorption by the hepatic veins may give rise to jaundice. (2) From congestion of the gastric and intestinal mucosa, there may develop indigestion, flatulence, eructations and vomiting often bloody; serous exudation into the bowel - giving intestinal indigestion and diarrhea, sometimes with black stools from decomposed blood - or into the peritoneal cavity - ascites; enlargement and tenderness of the spleen; hemorrhoids; varicosities in the lower extremities from direct interference by an enlarged liver with the current in the inferior vena cava.

(11) Sir W. Jenner, in a lecture on ascites from obstruction of the portal vein, gives a very clear cut picture of the syndrome as is recognized clinically - quote:

"Today, Gentlemen, I propose to speak of a case of Robert A., a man who was under my care recently and whose abdomen burst; you may remember that it gave way at the umbilicus with a loud report and a quantity of fluid came out. He had ascites and also a considerable amount of gas in his peritoneum. You know quite well, when an abdomen is resonant to percussion, and the resonance, i.e., the air within the abdomen - move as you move the patient, that, as rule, the gas is not in the peritoneum but in the intestines. In this case the gas was in the peritoneal cavity, that is why there was a report when the abdomen gave away. The real cause of the man's trouble was an impediment to the flow of blood thru the portal vein and an impediment to the escape of bile from the hepatic duct. Whatever permanently impedes the flow of blood thru the portal vein must have, as its necessary result, one or more mechanical consequences of congestion of the part the blood from which is poured into the portal vein. The blood cannot easily escape from the portal vein, and of course, all the tributaries of the portal vein are swollen. The consequence is that where there is impediment to the passage of blood thru the portal vein, there is enlargement of the organs. The same impediment to the onward flow of the blood may also produce rupture of the vessels. Common seats of hemorrhage are the stomach and the rectum - bleeding piles and hematemesis. These patients suffering from obstruction to the flow of blood thru the portal vein have effusion of

serosity from the congested vessels. There is effusion of serum into the cavity of the peritoneum - ascites. The mucus membrane of the bowels often suffers from this when there is mechanical congestion. The patients have watery diarrhea, the coats of the stomach become thick, again inflammation is very apt to supervene in the congested parts resulting in a catarrhal inflammation of the stomach. When there is a sudden impediment to the flow thru the portal vein, hemorrhage occurs. When the impediment is more slowly developed, serous effusion occurs. When hematemesis takes place, it generally relieves greatly and at once the congested organs. You find a man's spleen big today, he has hematemesis and tomorrow his spleen is greatly diminished in size. When the portal vein is stopped, the remains of the umbilical vein become dilated and this little channel grows into a big vein, communicates with the mammary veins and so returns the blood on the surface and you see greatly distended veins.

"Whatever impedes the flow of bile thru the hepatic duct or thru the ductus choledochus must impede the escape of bile from the liver; and whatever prevents the escape of bile from the liver will have jaundice following it, and all the consequences of jaundice."

Sir Jenner's discussion includes most of the important points in the syndrome but does not go into the mechanism of production in detail. With a consideration of the various types of obstruction, these points will automatically come to

a head. The progression in this entity is such that to follow it from beginning to end will show itself manifested as a definite clinical syndrome in which the mechanism, cause and effect are evident. Before discussing the possible obstructions with their definite cause, pathology and effect on the human mechanism, it appears logical to give a case history of the clinical manifestation, and then attempt to correlate the subjective and objective findings.

Case 1. (12) Charles Macfaden, age 32, admitted into the Royal Infirmary, April 1841, with symptoms of ascites. Two years previous he had noticed a swelling in the left side of abdomen about the size of a fist but causing no uneasiness. Not long after this he was attacked by profuse discharge of blood from the stomach and this was followed by distention and enlargement of the abdomen. Countenance appeared pale and colorless, features were round and bore no marks of emaciation. The abdomen was greatly distended and communicated a distant sense of fluctuation. A hard mass was palpable in left side of upper abdomen. Distention appeared to be at first stationary, but eventually the swelling impeded respiration so much that a paracentesis was done. Following this the patient was much better. The swelling, tho returning, was doing so slowly. During this time he took solutions of hydriodate of potass; bitartrate of potass; while counter irritating remedies and friction over abdomen by means of iodine ointment, were employed. Swelling gradually reappeared; respiration

became oppressed; and the patient was less able to move about. The various means of alternating his suffering were continued. After a second paracentesis patient died.

Autopsy: 64 oz. of fluid, of straw color, were withdrawn from peritoneal cavity. The liver was greatly diminished in size and the whole organ was altered in shape, being more rounded and contracted. Left lobe had almost completely disappeared, the surface of the liver was irregularly nodular and of a slight yellow color. The portal vein was found to be completely filled and distended by firm, yet compressible, elastic matter, this adhered to the coats of the vessel.

Case 2. (13) On December 28th, 1866, about 2:00 P.M., shortly after partaking of luncheon, H.F.W., Esq., age 57, formerly an Officer in the Army, was seized with faintness which was soon followed by vomiting of about one pint of blood. Medical aid was promptly obtained and the usual treatment resorted to, consisting of the free use of ice and ice water, accompanied by frequent doses of Gallic acid and subsequently of lead acetate with opium. Under this treatment there was no return of hemorrhage until the following morning at nine o'clock, when the patient, while on the right chair in an adjoining room, was again seized with faintness and vomited about 1-1/2 pints of dark liquid blood.

Dr. Smith, who was in attendance, again advised a continuance of the styptics and other treatment. During this day, patient rejected nearly everything swallowed and brought up at intervals small amounts of blood. Again in

the P.M., the hemorrhage began and amounted to about one pint. Dr. Moorehead was called in the absence of Dr. Smith.

On his arrival at 10:00 P.M., he, ^{found} the patient, nearly pulseless and perfectly blanched. Within a few moments, he vomited another pint of blood. It was then obvious that the case was hopeless, unless hemostatics would help. With this in mind, half a drachm of tincture of perchloride of iron with morphia in ice water was administered, but only ten minutes passed until the stomach ejected its contents. Patient died December 30th, 3:00 P.M.

Autopsy: On opening the abdomen, the omentum was seen to be loaded with fat, the stomach was removed from the body and after being opened, was found to be empty and greatly congested toward its cardiac extremity. The spleen was enlarged to nearly three times its normal size but was otherwise healthy. On dissecting the parts contained in the gastro-hepatic omentum, the attention was arrested by an osseous or calcareous spiculum about an inch in length, situated in the wall of the portal vein about one inch from its entrance into the transverse fissure of the liver. It was found that the calcareous plate was produced by degeneration of the middle coat of the vein and extended along its anterior aspect for about one inch, the canal of the vein, at the part where the degenerations presented themselves, was found completely occluded by a dark red fibrous concretion or thrombus. The vein was considerably dilated at the site of the thrombus, while on the hepatic side of the obstruction it was contracted

and assumed the appearance of a fibrous cord.

Case 3. (14) A. N., aged 24, admitted to Royal Infirmary, April 15th, with ascites; the swelling was rendered all the more apparent as he was a slight built man; the abdominal walls were tense and shining; and the superficial veins were distinctly outlined. The patient stated that on the morning of April 9th, he noticed that his belly was very prominent, the only discomfort was a sensation of weight and tightness in the abdomen. There was no history of abdominal pain or tenderness or of edema of any part of the body. Twelve months ago he had been under treatment for a chancre, not followed by any secondary symptoms. On April 23rd, the abdominal swelling had increased half an inch and it seemed impossible to relieve the distention except by paracentesis, however, before resorting to this I determined to try elaterium in eight grain doses, twice daily. After the second dose there was some irritation of his stomach and the bowels had acted very freely. I omitted the elaterium.

On measuring his abdomen three days later, it was found to be one inch less in girth and from this day to May 3rd, ten days later, the swelling completely subsided. On May 17th, the patient was discharged as cured.

With the conclusion of Gilbert (3) and Jenner (11), these three cases seem to fit in fairly well. In my review of the literature, I could find no case in which opsiruria was made to stand out as an important and alarming sign. Neither could I find a case in which jaundice was the outstanding and

important clinical finding. In many of the cases, some of which I shall cite later, these symptoms were present but it seems that hemorrhage and ascites took so much of the picture that little attention was attached to opsiuria or jaundice. Piersol (6) states that development of hemorrhoids, at times, occur but are not clinically important. It may be concluded that gastrointestinal hemorrhage and ascites are the outstanding clinical features, and for this reason I selected the three given cases to give a general picture of what one may expect to observe in the terminal states. They also show the significance of the progression, altho the cases of ascites with slight gastric hemorrhage and of marked terminal hematemesis are very close together, they serve to show just what relationship, between the hypertension and abdominal ascites must occur before there is fatal hematemesis. The third case illustrates the early type, in which the vascular hypertension is just sufficient to cause effusion of fluid into the peritoneal cavity, but not sufficient force to cause; first, a dilatation of the cardiac veins to form varices, and then gradual increased hypertension in the portal system to cause rupture of these vessels. It also shows that clinically, the obstructing thrombosis may become organized or break loose so as to pass to one of the branches leading into the liver, thereby releasing the portal hypertension and relieving the patient of symptoms. In this case, just which happened was never determined but in so young an individual it is easy to believe that the thrombosis became organized allowing more blood to

pass to the liver. Again it could have broken loose so as to block just one branch into the liver - in this case, in due time, it could be expected that either the right or left lobes became atrophic.

To explain why one case develops hematemesis without ascites, and the other develops tremendously embarrassing ascites with slight hematemesis in somewhat imperical. As stated by Gilbert (3): Of all the symptoms which constitute this syndrome, the earliest is opsiuria. It is still maintained by the French Writers that the most delicate index of portal congestion lies in the delay in the elimination of ingested water by the kidneys. The following investigation was carried out in order to ascertain if any experimental evidence could be obtained in favor of this suggestion. (3) In order to ascertain the part played by portal congestion on the absorption and elimination of water, it was decided to produce simple mechanical congestion by partial occlusion of the portal vein with a ligature. Rats were selected for these experiments, as the curve of diuresis following the administration of water to these animals has been found to resemble that occurring in the human subject (15).

The aseptic operations were carried out on rats under ether anesthesia. The abdomen was opened and a ligature passed around the portal vein about the entrance of the coronary vein of the stomach. The knot was tightened until congestion and slight cyanosis of the intestine became evident. Portal congestion having been produced in this manner, the abdomen was

closed. The rats were given bread and milk for three or four days, and no food for several hours before the beginning of the experiments on water absorption and diuresis. The animals were weighed and given five percent body weight of warm water by a small stomach tube. To determine the rate of water absorption, the animals were killed by chloroform 35 minutes after giving water. The alimentary canal, from the cardio-esophageal junction to the rectum was dissected out and weighed together with its contents. The average percentage weight of the gut plus the contents depends upon the amount of water absorbed, and the average alimentary absorption rates of the various groups of animals may thus be compared. Rats which had a previous diet of bread and milk were given five percent body weight of warm water by stomach tube and the animals were kept in small wire cages during the experiment. Since it is found that rats will void their urine upon shaking the cage, it was possible to collect the urine on cotton wool swabs which can be weighed, and the amount of urine determined by subtracting the weights.

It has been shown that anesthesia, and anesthesia and laparotomy, in themselves cause for some hours a delay in the absorption of water from the alimentary tract. In order to control the observations upon animals with portal obstruction, a set of animals from the same batch were subject to anesthesia and laparotomy only. The figures obtained in this series of rats indicate that the rate of water absorption does not differ materially from that in normal controls.

From this experiment it was concluded that portal congestion causes a delay in the absorption of water from the

alimentary tract. This delay is still present at the end of a week, although slightly relieved by the development of a collateral circulation. The day after the operation of portal obstruction, there is definite delay in diuresis. It is probable that the slow absorption of water from the alimentary canal is a contributing factor in this delayed excretion of water. Provided enough water is absorbed to start a diuresis, then the diminished rate of absorption would be sufficient to maintain a normal or nearly normal rate of water excretion. In the course of a week, venous anastomoses open up and the delayed absorption rate improves but does not return to normal. The diuresis curve may at this time have returned to normal despite the moderate delay in water absorption.

This experiment points the way to an explanation why hematemesis may occur without ascites and why the marked ascites sometimes develops without hematemesis. After obstruction has taken place it is seen that delayed absorption and delayed diuresis takes place. When this obstruction occurs acutely, the venous channels do not have time to compensate, and during this time diuresis is diminished along with the absorption. The intestinal mucosa becomes water logged and effusion takes place into the peritoneal cavity. Unless collateral circulation is set up, marked ascites develops. If during this process, the collateral circulation is more readily and quickly set up, the rate of absorption improves and diuresis is more marked. The intestinal mucosa does not become so water logged and ascites occurs in the same proportion as is permitted by

the degree of collateral flow. It may be assumed then that in cases of acute hematemesis without clinical evidence of ascites, that at some time a varying degree of ascites has occurred altho probably not detected. It must be assumed that the collateral circulation has opened up more quickly, and that the portal obstruction present has caused a direct strain up these collateral pathways. The gradual and continual dilation has lessened the hypertension in the tributaries of the portal vein resulting in a degree of increased absorption and diuresis enough to prevent any marked effusion of serum thru the intestinal walls into the peritoneal cavity. Assuming that this is true, it can be seen that all the strain of an existing portal hypertension would be upon the established routes of collateral circulation. The process continues, and the nervous walls at the junction of the portal and caval systems gradually become thinned out, tortuous varices. These, as the sequence continues, rupture to give rise to fatal hematemesis as seen in the case described.

The portal vein obstructions have already been listed. Clinically it appears that cirrhosis of the liver is most frequently met with in the general run of case, and should be considered first.

(16) The name cirrhosis, meaning yellow or tawny, was first used by Laennec to describe that form of liver disease which is now known by his name, and in spite of the fact that many cirrhotic livers are not yellow but green, the name has persisted and is now synonymous with sclerosis or fibrosia.

One of the greatest difficulties which one encounters in studying the subject of cirrhosis is the nomenclature. There is atrophic cirrhosis and hypertrophic cirrhosis, portal cirrhosis and biliary cirrhosis, Laennec's cirrhosis and Hanot's cirrhosis, multilobular cirrhosis and monolobular cirrhosis. Most of these terms mean very little and are of little help to the Physician studying a case at the bed side.

(17) Mallory recognizes the following five varieties of cirrhosis. Alcoholic, syphilitic, pigment (Hemochromatosis) infectious, and toxic.

I shall describe the etiology and pathology of each type and then consider the mechanism and consequences of portal hypertension as produced by the cirrhosis.

Alcoholic Cirrhosis

This form of cirrhosis is known as alcoholic cirrhosis, gin-drinkers cirrhosis, and other names indicating the part which alcohol plays in the etiology. In recent years, scientific research has shown that the bases from this idea is by no means firm. It has been impossible to produce absolutely characteristic lesion by feeding animals alcohol. At the autopsy table, it has been found that a large number of confirmed drinkers show no evidence of cirrhosis. Also it has come to light that in Scotland, where the consumption of alcoholic liquors is high, the incidence of cirrhosis is low. It is probable that the lesion is produced by some impurity in the alcohol. (18) Prolonged excess in any variety of alcohol produces a chronic form of inflammation, which, as is usual in such inflammations, selects for special attack the supporting structure rather than

the parenchyma of the organ. Alcohol absorbed from the stomach is brought directly unchanged to the liver, where it acts as an irritant. Clinically, alcoholic cirrhosis is accompanied by enlargement of the liver, the edge being felt hard, firm, tough and blunt, somewhere between the ribs and the umbilicus. (18) A study of cases by author seems to show that in all cases of alcoholic cirrhosis of liver, the liver is generally enlarged in all stages of the disease and whether enlarged or contracted, the clinical symptoms and cause of the disease are much the same.

It has been suggested by Boyd that bacteria may play a part in relation to alcohol in the production of cirrhosis. (19) Opie found that the administration of chloroform, followed by the intravenous injection either of streptococci or of bacilli coli, led to the development of cirrhosis. The chloroform apparently damaged the liver and enabled the microorganisms to maintain a footing. It may be that alcohol plays a similar part in the production of cirrhosis.

Microscopically there is a combination of atrophy of liver cells and increase in the fibrous tissue. It would appear that the process is a continuous one, extending over months or years, liver cells being continually destroyed and continually replaced by regeneration of the remaining cells. The degenerated cells may be found in any part of the liver lobule, but usually the process is most pronounced in the portal region. The architecture of the lobules is lost owing to the continued process of destruction and regeneration. The fibrous tissue shares in the process equally with the parenchyma. In the

early stages it is infiltrated with inflammatory cells, there is a marked fibroblastic proliferation and by this means the fibrous tissue is increased. As time passes this tissue becomes dense and undergoes marked contraction. It is in this stage, that clinically there may be manifestations of pain over the liver. The peritoneal reflections over the liver have been also contracted so that there is a continual tug - especially marked with the excursions of the diaphragm. (20) Dr. Chester Jones states: "I think that it is important to remember that in the terminal stages of cirrhosis, very severe abdominal pain is not uncommon. It very frequently resembles a surgical emergency yet at autopsy nothing is found but cirrhosis.

Syphilitic Cirrhosis

(21) The lesions of congenital and acquired syphilis in the liver are practically identical, the only modification arising being due to the element of time and the difference in the reactive powers of the liver at different periods of life.

(22) In the congenital form the liver may be large, smooth, and diffusely scarred or it may present gummata which later become scarred. In the acquired form gummata heal with large scars, producing deep grooves and lobulations. This form of cirrhosis may be dismissed since rarely is there any marked portal obstruction to produce a hypertension.

Pigment (Hemochromatosis) Cirrhosis

Pigment cirrhosis is the name applied by Mallory to those cases of cirrhosis which appear to be due to the deposition of pigment in the liver. It is usually known by the name of hemochromatosis. (16) Von Recklinhausen demonstrated many years

ago that there are two pigments deposited in hemochromatosis, one containing iron, Hemosiderin, and the other containing no iron, Hemofuscin. It was then pointed out that the Hemofuscin was deposited first. When this material appears in the liver cells it is a sure indication of the presence of hemochromatosis. The deposition of pigment leads to extensive necrosis of the parenchymal cells. As is the case in alcoholic cirrhosis, the process is a continual one, with constant destruction and regeneration of liver cells. It is the slowest type of cirrhosis to develop and probably requires ten to fifteen years before the appearance of symptoms. There is a great development of fibrous tissue probably due to the irritation set up in the intestinal tissues. The cirrhosis is of a portal type and resembles that seen in the alcoholic form.

(23) In 1882, Hanot and Chauffard described a case under the title of bronzed diabetes with pigmentary hypertrophic cirrhosis. It has since been recognized as a clinical entity. In regard to etiology, it was first noted that the disease is confined almost entirely to the male. The only undoubted case of hemochromatosis in a woman is that reported by (23) Maude and Abbott. A definite cause has never been found.

Infective Cirrhosis

(24) Adami calls this type sporadic cirrhosis and states that it is the same as Hanot's cirrhosis. It is claimed to be due to focal necroses and is secondary to the inflammatory foci which act as centers from which there radiates fibroid changes. This is the second type of cirrhosis in which there

is no portal obstruction and therefore can too be dismissed lightly. I have mentioned these two forms only for the sake of completeness.

Toxic Cirrhosis

(16) Boyd calls this form by the name of healed yellow atrophy, the process is not continued, as in the case of alcohol cirrhosi, but intermittent. In many cases the patient gives a history of repeated attacks of jaundice which may be associated with pain, fever, and vomiting. The damage done to the liver is acute or subacute, followed by active regeneration. The extent of the damage varies greatly, and may be added to in subsequent attacks.

The liver presents a real atrophic cirrhosis, but in some instances it may be enlarged. Its most remarkable and distinguishing feature is the presence of a very pronounced modular hyperplasia. The nodules vary more in size than in the alcoholic form and the result is a distortion of the liver.

The following case report is said by author to be the first and only on record.

(25) Many chlorinated hydrocarbons have a toxic action on the liver. Of these, chloroform is the best known. Carbon-tetrachloride is closely related chemically to chloroform and likewise is a hepatic poison. Because of its non-inflammable character and because it is an excellent solvent for fats and greases, it is extensively used in industry.

Many cases have been recorded after the inhalation of fumes of carbon tetrachloride. This may produce only a slight

nauses or dizziness, but in severe cases death ensues. In many of these fatal cases death is due to acute necrosis of the liver with the clinical picture of acute yellow atrophy. In addition to this acute injury, Ballman (26) has shown that hepatic cirrhosis can be produced in dogs by the repeated administration of small doses of carbon tetrachloride.

(25) Case: A man, 46, an Italian, presented himself at the hospital, May 10th, 1933, because of "swelling of stomach" of three months' duration. For the past 11 years his occupation had been that of a cleaner of clothes. The cleaning fluid used since 1908 was a mixture of 55% carbon-tetrachloride and 45% naphtha and benzene. During this period many men worked with him but none could continue because they soon became ill with loss of appetite, diarrhea and vomiting. Many times, patient had attacks of nausea and vomiting with vertigo. Patient insisted that he did not drink hard liquors. He has had difficulty for past three years with gas on stomach. He has had dull aching pains localized in right upper quadrant, this usually appeared after a heavy meal or after greasy foods. Three months before admission this pain had been more severe and he noticed that there was considerable tenderness over the area of pain. Coincident with onset of tenderness, the abdomen gradually began to increase in size.

Physical findings: Sallow complexion, sclerae were slightly icteric. The face and arms were thin as compared to the abdomen. Liver palpable four fingers below costal margin. Spleen also palpable. There was some venous enlargement in the

superficial veins of abdomen.

During stay in Hospital it became impossible to control the ascites. An explanatory laporatomy peformed and a large amount of fluid was found, liver was markedly cirrhotic. A splenectomy was done and the omentum implanted into the anterior abdominal wall. Patient died three days after operation.

Pathological diagnosis at autopsy: Chronic toxic cirrhosis.

I cite this case because I think it is interesting and because it is a recent development. It becomes an important factor since there is so much carbon tetrachloride used in modern industry. It is probable that many more cases of like etiology will be brought to the front.

Although the starting point and the pathogenesis of the various forms of cirrhosis are very different, the end result is on the whole similar. It may be thought that symptoms of hepatic insufficiency would be prominent owing to the extensive destruction of liver tissue. This is not the case. Tests for liver function usually give disappointingly normal results. The reason for this is the large margin of safety which the liver possesses. It is in the acute necroses that real insufficiency of the organ is apparent. In cirrhosis the symptoms appear to be rather obstructive in type. The condition begins insidiously and may continue to an extreme condition without producing any symptoms which call attention to the existence of the process. Very frequently the earliest symptoms are associated with the alimentary tract; next in order are evidences of portal obstruction. For this reason

it may be well to divide the symptoms into:

(1) The disturbances occurring in connection with the alimentary tract.

(2) Symptoms of vascular obstruction.

Symptoms of gastric and intestinal disturbances: Of these the most noticeable are: At the very earliest stage, slight dyspepsia, morning vomiting or nausea, and furred tongue; added to these there may be eructations and irregularity of the bowels. There is often an alternation of constipation and diarrhea. During the former, the stools often present remarkable modifications. Some days they are normal, then they become very dry and are covered with a thick layer of mucus; at other times they are colorless. To these disturbances of the digestive tract may be largely attributed the emaciation of the later stages of the disease.

Symptoms of disturbance of circulation:

As long as there is a good collateral circulation, symptoms will be negligible. It is only when this collateral circulation becomes inadequate to carry the portal blood to the heart that ascites and other obstructive disturbances supervene. It is for this reason that at times cases of extensive cirrhosis are met with, without a sign of ascites. Gastric hemorrhage occurs, as stated before, in the plexus of submucosal veins at the lower end of esophagus. These veins play a vital part in the collateral circulation and are practically unsupported toward the free surface of the esophagus. When the hypertension becomes too great they become

varicosed and relatively enormous. The patient may appear in very fair health and the liver performing its functions satisfactorily but with a thirty-second of an inch, or less, intervening between life and death, for it is these varicosed sub-esophageal veins which are especially liable to rupture and produce so extreme a hemorrhage that death follows in the course of a few hours.

Preble (32) in a report of sixty fatal cases of gastrointestinal hemorrhage, considers it an infrequent but not a rare complication of cirrhosis. In 80% of his cases varices were present and in half of these macroscopic ruptures were found. He adds that many more varices and perforations would be found if fluids were injected to detect them. Blumenau, according to McIndoe (31), reported that of 126 patients affected with obvious portal cirrhosis at the time of death, 19% died from vascular lesions and 19% from cirrhosis itself. Besides this group there appears to be an increasing number of patients who have steered past the fate of hemorrhage and ascites, succumb to hepatic insufficiency. McIndoe, believes these too, are probably of vascular origin.

The ascites of portal cirrhosis develops gradually and in this way it may be distinguished from that produced by portal thrombosis. While this symptom is the most prominent of the condition, it is by no means always present. It is because of this factor that many errors in diagnosis are made. It is generally believed that ascites must be present and

therefore many cases are missed. The older Writers state that ascites is present in about 80% of cases; more recent observations seem to point that it does not occur in more than 30% of cases (24).

As to the cause of the ascites, opinion is being more strongly expressed that portal narrowing alone is not the cause. When the liver becomes greatly contracted the portal territory within the liver is diminished, and with the smaller field there must be some obstruction. A fact which has never been explained is the continued vascularity of the organ.

Edema of the feet is not infrequently secondary to ascites, and is, in the main, due to a pressure of the distended abdominal contents upon the veins coming from the lower extremities. Edema of the feet may precede the development of the ascites. In this case it is due to the malnutrition of the patient and the impoverished condition of the blood. Adami (24) cites a case of angioneurotic edema associated with cirrhosis of the liver in a male patient of forty years.

Enlargement of the spleen is by far more frequent than ascites, and may be considered as the most frequent symptom associated with portal cirrhosis. The enlargement is, in general, not so marked as is biliary types; the organ averages between one and one-half times to twice the normal weight. Adami (24) believes that this enlargement of the spleen is not entirely due to portal obstruction because it appears at so

early a stage before other signs of obstruction are evident.

Hemorrhoids - While hemorrhoids are frequent in cases of portal cirrhosis, the majority of Writers seem to be of the opinion that they are far from being as common as generally believed.

Pain over the region of the liver is often the most noticeable symptom in the early stages, and is often accompanied by a sense of epigastric fullness and tension, which may be present throughout the duration of the disease. The liver is innervated from the seventh to the tenth dorsal, and, as a consequence, the pain affecting the organ may be referred to the cutaneous branches of these nerves by overflow of impulses in the cord. When this occurs, pain may be felt in the region of the angle of the right scapula. Another pain sometimes experienced is at the tip of the right shoulder, more rarely both shoulders. Where this is the case there is an indication of involvement of the upper surface of the organ, extending to the diaphragm, for such a pain is brought about by the overflow of impulse at the point of entry of the phrenic nerve into the spinal cord, and so there is reference to pain along the branches of the lower cervical nerves.

Wasting is always a striking feature in abdominal cases; the contrast between the distended abdomen and the emaciated thorax, face and extremities may be most marked.

In the earlier stages there may be little or no changes in the urine but as the condition progresses, the quantity of urine diminishes in amount, the color becomes

dark. The urea is often found diminished, as also are the chlorides when ascites is present; the urates are then usually markedly increased. Albumin is, at times, present with casts.

There is very little that is characteristic about the condition of the blood in portal cirrhosis. There is no marked increase in leucocytes, no extensive diminution, either of hemoglobin or in the number of red blood cells.

Altho there are other causes of obstruction to the portal circulation, the problem of cirrhosis just about covers the essential points of the syndrome. Its study gives a better concept of just what can happen since the process is so insidious and you can follow through from the very earliest of gastrointestinal upset, spleen enlargement, urinary oliguria, ascites, hemorrhage - to the later phenomena of edema, pain, cachexia and death.

In the other obstructions of the porta, the process is more rapid, such that the ascites may come on very rapidly and may be the first noticed. Depending upon the collateral circulation, it may not develop, and in this case, about the first thing found is a severe gastric hemorrhage which may or may not be fatal. From this point of view, these mechanical obstructions of the portal vein may be considered an intermediate emergency in which the system is suddenly embarrassed and does not have time to compensate. There is not the chronicity and the full drawn-out course of events as found in the subject just discussed. For this reason it will not be necessary to

recapitulate, and the obstructions to be considered may be thought of as intermediate emergencies, the important thing of which is the immediate pathology.

The Mechanical Obstructions of Portal Vein

A. Valvular diseases of heart:

Ascites in chronic valvular diseases of the heart has long been recognized. In long standing cases of stenosis of the aortic valve, a mechanical process takes place, the final picture of which may incorporate a typical portal obstruction syndrome. When the valve becomes insufficient, blood regurgitates into the left ventricle. The ventricle, in order to care for the excess blood, must compensate in some way and it does this by a hypertrophy of its wall. If the load does not become too great, this is quite sufficient but as a greater burden comes into play, a strain is placed upon the mitral valve, and this too gives away. There is now a new load for the left auricle and in order to handle it there must be a hypertrophy of its wall. Again, as the burden becomes too great for the left auricle, the pulmonary veins dilate in an effort to carry on the work. When nature can no longer carry on, the blood begins to back up into the lung so that the volume there is increased, resulting in a passive congestion. When this becomes too great the process continues so that the right ventricle and right auricle become enlarged because of the ever increasing volume of blood. Finally the liver is reached and a passive congestion results. As this is increased, there results an obstruction to the incoming portal blood; it is forced to back up in the portal system, re-

sulting in a splenomegalia, congestion of the walls of the stomach and intestine and establishment of collateral circulation to the caval system.

This, in general, is the process which takes place. It may also result from defects in the mitral valve and the mechanism is the same except for the starting place. Death may result from the congestion caused in the portal system; exitus by hemorrhage from varices, ascites or cachexia. In a recent case reported by (41) Feldman and Gross, this process actually took place and the patient dies of retroperitoneal hemoperitoneum, an extremely rare complication, even in true cases of portal cirrhosis. The mechanism of this so-called "cardiac cirrhosis" and its relation to the production of a picture of portal obstruction are discussed.

Case:

F. T., a female, 18 years, was admitted to the Montefiore Hospital on January 26th, 1933, with dyspnea and swelling of the abdomen as her chief complaints. At the age of seven years she had had chorea. One year later, following a sore throat and tonsillitis, she had migratory joint pains. Numerous sore throats subsequently occurred, at least once per month. A heart lesion was diagnosed at the age of fourteen years, dyspnea on slight exertion was experienced, and one year later signs of congestive heart failure with slight edema of ankles appeared. She entered a hospital where the congestive heart failure subsided. However, numerous bouts of congestive failure necessitated numerous admissions to various hospitals for the restoration of compensation. In

June 1932, swelling of the abdomen was observed for the first time with very slight edema of the ankles. The patient was admitted at that time to another institution where six paracenteses of the abdomen were performed for recurrent ascites. Her condition gradually became worse, and she was transferred to the Montefiore Hospital as a case of chronic congestive heart failure.

Examination revealed an extremely ill-looking young woman, orthopneic; markedly dyspneic with cyanosis of the lips, ears and tip of the nose. The skin and sclerae had an icteric hue. The nasal septum revealed a large perforation anteriorly. The superficial neck veins were markedly distended and showed a totally irregular ventricular type of venous pulse. The trachea was in the mid-line. Over the anterior chest wall and the abdomen the superficial veins were markedly dilated, prominent and tortuous.

The apical impulse of the heart was in the left anterior axillary line at the level of the sixth intercostal space. There was marked systolic retraction in the apical region; change of position did not affect the location of the apex. A diastolic thrill was felt at the apex where there was heard a rumbling diastolic murmur preceded by a rough systolic murmur. At the aortic regions there were blowing murmurs throughout systole and diastole. The second heart sound in the pulmonic area was markedly accentuated and reduplicated. The peripheral vessels revealed a Corrigan pulse and pistol shot sounds over the femoral arteries, but no

capillary pulsations were noted.

The abdomen was markedly distended, with bulging of the flanks and protrusion of the umbilicus. White striae were prominent over the abdomen. A definite fluid wave was present. The liver was very large, pulsatile, and extended down to the pelvic brim, 12 cm below the right costal margin in the mid clavicular line. The spleen was markedly enlarged.

In contrast to the marked ascites, the lower extremities revealed only slight pitting edema.

There was a moderate secondary anemia with hemoglobin count of 60 percent, the erythrocytes numbered 3,500,000 per cubic millimeter, the leucocyte and differential smear were normal.

X-ray and flourescopic examination of the chest revealed marked enlargement of the left ventricle and of the left auricle. There was marked enlargement of both the inflow and outflow tracts of the right ventricle. No definite enlargement of the right auricle could be demonstrated.

The electrocardiogram showed auricular fibrillation with right axis deviation.

In the middle of May 1933, she developed mild diffuse abdominal pain following the removal of nearly 11 liters of ascitic fluid at one time. Marked diffuse abdominal tenderness was elicited but there was no rigidity. The pains persisted, the umbilicus became red, and an explanatory punctures of the abdomen yielded a homogeneously bloody fluid. Because of the number of varices in the abdominal wall, it was suspected

that one of these veins had accidentally been punctured during the last paracentesis. Her abdominal symptoms gradually disappeared. Two weeks later the patient suddenly died while asleep.

Necropsy: The peritoneal cavity contained two liters of homogeneously bloody fluid. A large hematoma, 7 to 8 cm in diameter, was found in the lateral wall of the right side of the abdomen just beneath the parital peritoneum. It overlay a very markedly distended and partially thrombosed vein which had ruptured.

The heart was very large. The pericardial sac was completely obliterated, and the inferior vena cava was partially constricted and embarrassed by adhesions as it entered the pericardial sac. The myocardium revealed marked hypertrophy of the muscle fibres with numerous scars throughout. All the chambers of the heart were markedly distended. The aortic valve showed rolling and shortening of the anterior and mesial leaflets. The mitral valve was only slightly thickened along the line of the closure.

Microscopic sections revealed an extensive tuberculous pneumonic process in the consolidated region; typical tubercles and areas of caseation were seen. The left lung was markedly edematous but there was no evidence of tuberculosis.

The liver was massive and firm, weighing 2,200 grams. The hepatic venous radicles were markedly distended. The portal and splenic veins were patent and distended. The portal veins appeared to be kinked at the hilus of the liver. On section the liver was nutmeg in appearance, Microscopically the picture was that of extensive chronic passive congestion, com-

pressing the surrounding liver tissue, with some central fibrosis and atrophy and moderate fatty changes.

The spleen showed a marked chronic passive congestion.

The authors state that the case is extremely rare in view of the complication, and that it presents the unusual feature of clinical portal obstruction associated with fatal intraperitoneal hemorrhage from a ruptured abdominal varix. In selection of this case, it appeared to me that it gave the most vivid picture of just what could happen to the portal system in valvular defects of the heart.

B. Portal Obstruction caused by enlargement of Gall Bladder from new Growths or Concretions.

Because of the close relationship of the gall bladder to the portal vein it is quite easy to believe that obstruction of the vein could easily be caused by conditions in the gall bladder. The mechanism of production of a portal vein hypertension does not need discussion because it can readily be seen what would happen if pressure of the gall bladder were applied to the portal vein. The essential thing is to produce evidence that it can and has occurred altho considered as a very rare pathological process. I was fortunate to find just one such case.

(42). The following case which fell under my observation, is, as far as I have been able to ascertain, the only instance hitherto recorded in which death resulted from the mechanical pressure of a mass of gall stones on the vena cava leading to fatal obstruction of the portal circulation.

Case: Mr. C, aged 56, a Miller, of large and robust frame and temperate habits, had generally enjoyed good health, but three years ago he suffered a severe accident, requiring amputation of the thigh followed by perfect recovery. On April 4th 1868, he took a hearty supper and went to bed in his usual health. Soon after mid-night he awoke in great agony from intense pain in the abdomen accompanied with vomiting. Wife gave dose of Epsom salts which acted on bowels without relieving the vomiting. Enormous quantities of fluid were periodically ejected from stomach. At this period pain and vomiting still continued, with tenderness on pressure. Enemata and laxatives were given without relief. Nine leeches were applied over epigastrium but the patient grew worse. There was no increased dullness in the region of the liver; there were no febrile symptoms and the pulse was not rapid the very feeble. The urine was depressed, no doubt from deprivation of blood serum. The diagnosis at which we arrived was that some serious mechanical impediment to the portal circulation existed, most probably a tumor near the inferior surface of the liver.

Autopsy: Greater omentum deeply congested and clots of dark blood were scattered between its folds, the ascending mesocolon was gorged with blood - this congestion extended to the transverse mesocolon. The cecum was highly congested and the ascending colon was so distended that it presented a blackish color. Stomach contained a considerable amount of fluid. The liver was perfectly healthy and of natural size but on

removing it the Gall bladder presented a remarkable appearance. It contained three very large biliary calculi of about equal size. Posterior extremity rested in the portal fissure over the portal vein where it entered the liver, thus producing a mechanical compression of the portal vein to such a degree as to give rise to all the phenomena of congestion.

This case undoubtedly represents an acute portal obstruction resulting from pressure of the concretions in the Gall bladder, upon the portal vein. It serves to illustrate the ever existing possibility of obstruction of the venae porta by pathology in the contiguous structures. In the same way tumors and swellings of the liver; carcinoma of the head of the pancreas and diseased lymph glands in the portal fissure may produce similar disturbances.

(38) In an effort to explain the cause of death following ligation of the portal vein, Elman and Cole resorted to many different types of experiments. It seems clear to them that the rapid death which so regularly follows total obstruction of the portal vein is due to circulatory failure, because of extensive loss of blood from the general circulation into the trapped splanchnic area. No evidence of the production or absorption of a toxic substance was found, the evidence in favor of purely physical factors was uniformly consistent. The increase in weight of the splanchnic area following occlusion of the portal vein was great enough, on the basis of the amount of entrapped blood it contained, to have caused death from shock alone. The fall in blood pressure was similar to

that noted after an extensive hemorrhage except that the pressure was sustained at a low level until death. The behavior and appearance of the animal after ligation of the portal vein were similar to those seen after marked loss of blood from hemorrhage. Attempts to raise the blood pressure and prolong life by transfusion of blood were successful and it was possible to postpone death for more than six hours. It was also possible to prolong life and present the characteristic fall of blood pressure following occlusion of the portal vein by ligating the aorta above the celiac axis, which effectively stopped blood from entering the splanchnic area. These animals lived only a few hours but they lived as long as animals with ligation of the aorta alone. The results of the experiment were the same if, in addition, several hundred cc of blood were injected into the mesenteric arteries to produce the cyanosis and congestion ordinarily seen after ligation of the portal vein. Death following ligation of the portal vein is probably hastened by the fact that ligation, at the same time, prevents the gastrointestinal tract from aiding in the loss of fluid. When a low blood pressure is maintained too long it can never be corrected, no matter how much blood is transfused. (42) It is likely that too low a pressure slows the metabolic exchange and causes irreparable alterations in the central nervous system. On the other hand, the defect caused by a low pressure may be due to increased capillary permeability.

The Author admits that it is difficult to correlate these findings with clinical experience when one considers that patients with cirrhosis of the liver may develop a complete

obliteration of the portal vein and still live. Physicians likewise see patients with progressive infective thrombosis of the portal vein, such as the pylephlebitis, complicating peritonitis of appendiceal origin, who live at least a few days after the portal vein has become thrombosed. However, he points out that the obliteration occurring in both of these cases is gradual. In animals, a gradual obliteration of the portal vein is not fatal.

As would be expected, by means of blood transfusion, they were able to definitely prolong the life of the animal whose portal vein had been ligated. If the administration of blood was begun 15 to 20 minutes after the ligation of the portal vein and maintained constantly at a rate of 35 cc per kilo per hour, the life of the animal could be prolonged at least five to six hours. However, if the blood was administered at irregular intervals, allowing the blood pressure to drop below a level of 50 to 55 mm. for several minutes, death would usually follow in a short time in spite of the injection of large quantities of blood. This observation and explanation appears to be substantiated clinically by the well known fact that patients with shock of several hours duration respond quite poorly to restorative procedures and may die in spite of transfusion.

SUMMARY

In view of Gilbert's (3) early statement of the essentials in portal obstruction, I have attempted to carry the mechanical features foremost. Regardless of the pathology which may be responsible for this syndrome, the clinical

features are more or less the same but vary according to the starting point, the degree of obstruction and rapidity in which it takes place.

A study of this subject reveals a tremendous compensatory power of the body. It affords an excellent example of just what the human body is able to tolerate in the presence of pathology. It indicates the extremes of a compensation in which normal structures have been distorted in an effort to continue life. Opsiuria and delayed water absorption from the intestine appear to be the obstacles of the compensation. It is seen that if the obstruction takes place insidiously, nature is able to handle these defects for a considerable length of time, but if the process continues, the opsiuria and delayed absorption are more profound, resulting in a situation which places more of a compensatory demand upon the body. It is under these conditions that the patient begins to suffer.

Portal Cirrhosis is the condition most frequently met with in cases of obstruction. Regardless of the type of cirrhosis, the clinical picture is the same. It can be distinguished from the other pathologies causing obstruction by its insidious nature. Because of this fact, the complete picture can be visualized and studied from beginning to end. The progress and remissions of compensation can be identified and the stages recognized. In the pathologies causing more acute obstruction of the portal vein, the mechanism of production of symptoms is an intermediate part of the whole picture. It takes place abruptly so that nature is not able to handle the situation. The symptoms which

appear are comparable to those found in the late stages of portal cirrhosis.

The cause of death in portal obstruction has not been conclusively worked out. Elman and Cole are convinced that the rapid death which so regularly follows total occlusion of the portal vein is due to circulatory failure because of extensive loss of blood from the general circulation into the trapped splanchnic area. No evidence of the production or absorption of toxic substances, causing death, has been found.

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