PORTAL TENSION AND ITS DEPENDENCE ON EXTERNAL PRESSURE*

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ABUNDANT RECENT LITERATURE has indicated the wide interest in surgery of the portal system. Some of this has been controversial, some questionable, but all has been stimulating and provocative. Of prime interest has been the role played by an increased pressure in the portal system. It is with this phase of the problem that the present discussion deals.

Much controversy concerns the intrinsic effect of a scarred liver or an occluded vein on the portal pressure. These undoubtedly are factors in the disease of portal hypertension. They are considered by most to be the only important factors. It is the purpose of this discussion to point out a little considered and often ignored phase of portal pressure, namely the effect of external pressures upon the portal vein. It is thought that this has direct bearing and may be a most important feature in varices and hemorrhage.

Such a discussion must include a definition of what constitutes normal and what abnormal portal pressure. In the past these terms have been quite loosely used. For nearly a century[18] there have been suggestions that cirrhosis of the liver causes portal obstruction, resulting in an increased portal pressure. These speculations waited for the Presbyterian Group[22] to measure the portal venous pressure in such patients and to coin the irresistible term "portal hypertension." At the time of this initial study the normal portal pressure was considered to range between 10.5 to 12 cm. of water (saline). These figures were altered somewhat by others[2, 3, 12, 13] who considered pressures above 20 cm. as being distinctly abnormal and in the hypertensive range. Portal pressures associated with Banti's Disease varied from 20 to 50 cm. of water.

Pressures were taken with the usual glass standpipe manometer. For the most part they were recorded without reference to any base line. A few were centered at the "level of the right auricle." Apparently Bellis[1] was the first to use the portal vein as a base line. It is obvious that a common measuring point must be chosen if we are to discuss the natural history and therapy of portal hypertension.

In order to form a more accurate concept as to what might constitute normal and what abnormal pressures it was decided to determine pressures on a number of normal operative patients. The pressure base line was arbitrarily chosen as the anterior surface of the upper lumbar vertebral bodies.[21] This had the disadvantage of lying 1 to 2 cm. posterior to the supposed position of the portal vein during a laparotomy. It had the distinct advantage of being a measuring point which could be blindly and rapidly located at operation. As a normal group, we chose patients who were undergoing operation for cholecystectomy or gastric resection. There was nothing in their history or subsequent course to suspect that their portal pressures were abnormal.

Pressures have now been taken on 55 "normals." These pressures ranged from 14 to

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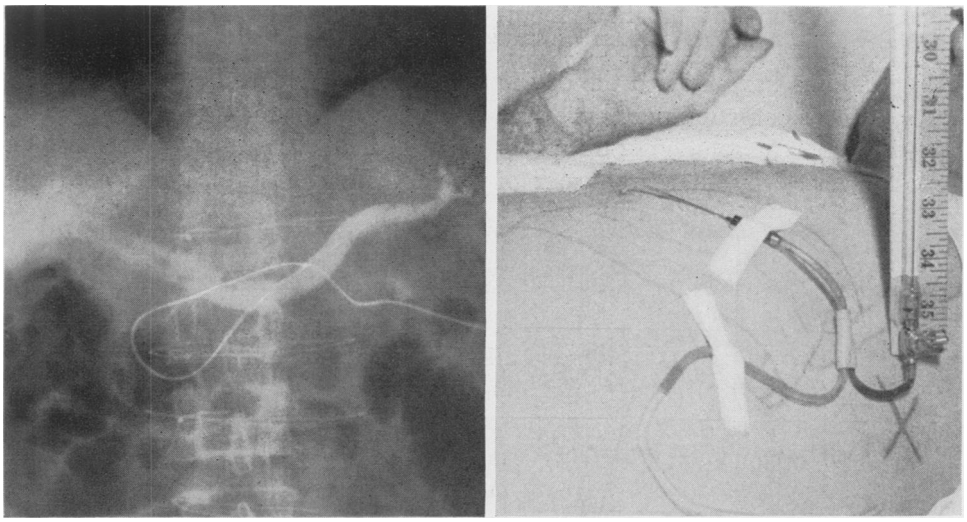

FIG. 1. Right. Plastic tube connected with manometer passes through the abdominal wall (upper left) and into the portal vein. Lower limb of Y tube allows a dilute heparin solution to perfuse the apparatus at all times. Left. Injection of Diodrast into plastic tube to demonstrate position of its tip and to outline the portal radicals.

26 cm. of water, averaged 21 cm., and approached the normal pressures reported by Bellis.[1] It should again be noted that these pressures were taken from the anterior surface of lumbar vertebra as a measuring point. Since the portal vein lies in front of this, a more accurate average reading might be 19 or 20 cm. This suggests that the measuring of portal pressure is rather crude, and is subject to variables. It is at best accurate to only 1 or 2 cm. Therefore, it is strongly urged that the millimeter reading of these pressures be discontinued, as it suggests an accuracy that is not warranted by the present conditions of taking portal pressures.

Similar pressures taken on patients who were considered to have portal hypertension ranged from 40 to 55 cm. of water. This leaves a group whose pressures were between 30 and 40 cm., and which defied accurate classification. Others have considered these pressures as definitely hypertensive and have included them in their various operative results. There are also reports of patients with portal pressures less than 30 cm. who have received one or another type of shunt for the relief of esophageal or gastric hemorrhage. Since these pressures are within physiological range there can be no justification for such practice.

Occasional patients were studied who had cirrhosis or ascites and whose portal pressures were definitely in the normal range. Others without the classical portal cirrhosis had interesting pressure readings which are worth recording. These included diffuse metastatic carcinoma to the liver with a pressure of 36 cm.; cholangiolytic hepatitis, 24 cm.; early biliary cirrhosis, 31 cm. A woman with a pan-hematocytopenia had a portal pressure of 23 cm. before splenectomy and 18 cm. after. This latter pressure continued for three days of observation. The dilated subcutaneous veins of a patient with Cruveilhier-Baumgarten Syndrome proved to have a pressure of 26 cm. A man with a severe constrictive pericarditis gave a pressure which varied from 30 to 40 cm.

ALTERATIONS IN PORTAL PRESSURE

With this background it seemed worthwhile to attempt temporary alterations of the portal pressure in man. It is important to know the normal variations in such pres-

sures and to learn how they might be modified by drugs or by external forces. To our knowledge no one has followed the day in and day out record of man's portal pressure in a group study. The solution of this problem was suggested by the early work of Hoffbauer, Bollman and Grindlay.[10] The placing of fine plastic tubes in the portal veins of patients to obtain continued pressure readings has been done by others.[8, 20]

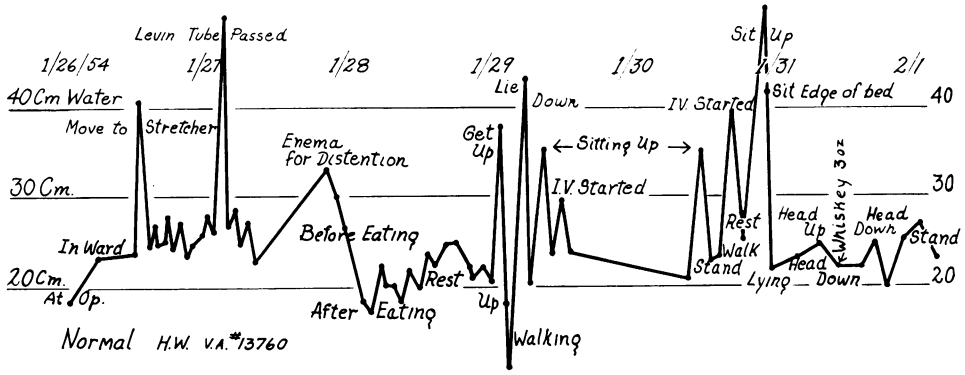

FIG. 2. Representative graph of portal pressures in a "normal" patient carried over a period of six days. Features which were used to induce a variation in pressure are indicated. It will be noted the only common factor which invariably caused a portal hypertension was a severe muscular effort. Seizures such as choking or cough changed normal into hypertension.

The great value of this method is that portal pressure readings can be continued for days after operation. Pressures can be recorded at any desired time or continuous pressures may be plotted. They may be taken without regard to anesthesia and with the patient in his normal environment. Above all, it allows a study of forces which change or alter the portal pressure.

The method used was considerably simpler than that of the Lilly capacitance manometer.[8, 20] It consists of a simple graduated glass manometer connected with a plastic tube which in turn was inserted into a radical of the portal vein (Fig. 1). At the time of operation the gastroepiploic vein or one of its larger branches was isolated a short distance to the left of the pylorus. It was opened between silk sutures and a boilable vinyl compound tube (1.65 mm. o.d.) was inserted. This was threaded around to the right and beneath the pylorus for a distance of six inches. The tip of the plastic tube had been rounded with scissors and a seration was made near the end to allow free flow of fluid should the tip become clogged or forced against the vein wall. Sutures about the vein and tube were then tied snugly to prevent leaks, and it was wrapped in omentum for a distance of an inch or so. The size of this tubing is such that it can be led out from the abdominal cavity through a 13-gauge needle. A constant flow of dilute heparin solution was allowed to perfuse the manometer system. This consisted of 1000 ml. of 5 per cent glucose solution containing 20 mg. of heparin. The heparin solution was allowed to drip at a rate of two to three drops a minute. At this rate it did not falsify the manometer readings and was readily metabolized by the patient without altering the blood clotting time. As will be seen in Figure 1, the manometer is connected to one wing of the Y connection and the source of heparin solution to the other.

While the abdomen was open the level and site of the portal vein was noted. A point at this same level was then marked on the lateral chest wall of the patient (X in Fig. 1). When making a reading it is important to level the base end of the manometer with the point on the outside of the chest which indicates the portal vein level.

This pressure apparatus could, presumably, stay in place indefinitely. Ours were in place and recording over periods of from two to ten days. Twenty-one such studies have been done; six on cirrhotics, ten on "normals," and five on interesting cases where it was hoped a pressure deviation might be present.

As a rule the tip of the plastic tube was identified as to position by means of the injection of 35 per cent Diodrast. This was done primarily because of a simultaneous study (portal vein blood culture). It was found that the tip of the tube occasionally took a course out through the splenic vein, and it was sometimes necessary to withdraw it a short distance to keep it near the portal vein origin.

Portal pressures were taken at the time of operation with the abdomen open and continued during the first few days of convalescence. Attempts were then made to produce changes in the recorded pressure. The patient was given various drugs, fed, exercised, and put through a wide range of procedures we hoped might alter the portal pressure. Representative pressures and tracings are shown in Figures 2 and 3.

Some of the interesting results are shown in the graphs. First of all, in the normal tracing (Fig. 2) it is noted that portal pressure is little changed by drugs, food, position or mental state. On the other hand, there were some maneuvers which resulted in a real portal hypertension. These had in common the muscular contraction of the diaphragm and abdominal wall. They caused a normal portal pressure to become definitely hypertensive (50 to 55 cm.). These increases recurred constantly with repetition of the particular muscular act. They occurred in the portal hypertensive patients as well as in the "normals." Nothing else approached the degree of this change. Minor elevations and indifferent depressions of pressure only, were produced with drugs. The drugs used included adrenalin, ephedrine, mephedrine, histamine, pituitrin, hexamethonium bromide and Arfonad. In animals and in man it was necessary to produce near shock levels of blood pressure before the portal pressure dropped to any significant degree.

For some time we have been interested in the relation of body position upon portal pressure. It seemed logical to assume that the portal pressure of a patient standing on his head or leaning down to tie his shoe would be enormously increased. In fact, the increase should be directly proportional to the increased hydrostatic head produced by the inverted position. Conversely, the pressure in the portal vein should be greatly lessened by placing the patient in the upright position. Strangely enough, we were unable to demonstrate this. Both "hypertensives" and "normals" were stood up, inverted to a 45 degree angle and again placed in a supine position without any significant portal pressure change. At times an elevation would appear but would disappear upon repetition of the test, using care to eliminate muscular straining.

How this pressure is kept more or less constant is not clear. The only logical answer would seem that as position is changed, collateral veins are brought into play, opening new by-passes to the vena cava from the portal system. In some ten patients in whom pressures were recorded for varying body positions there was one exception to the above tendency of constant pressure. This occurred in a rather small, slight man who had had a cholecystectomy. Four days following operation his pressure was in the range of 14 to 19 cm. for supine and head-down positions. When this man stood up the pressure promptly and repeatedly dropped to zero. Unfortunately this occurred in the early days of our tests and was not checked subsequently.

There seemed to be little difference in alteration of portal pressure in the normal or hypertensive cirrhotic. A cough or splinting of the abdominal muscles sent both upward to 50 or 60 cm. of water. The increase

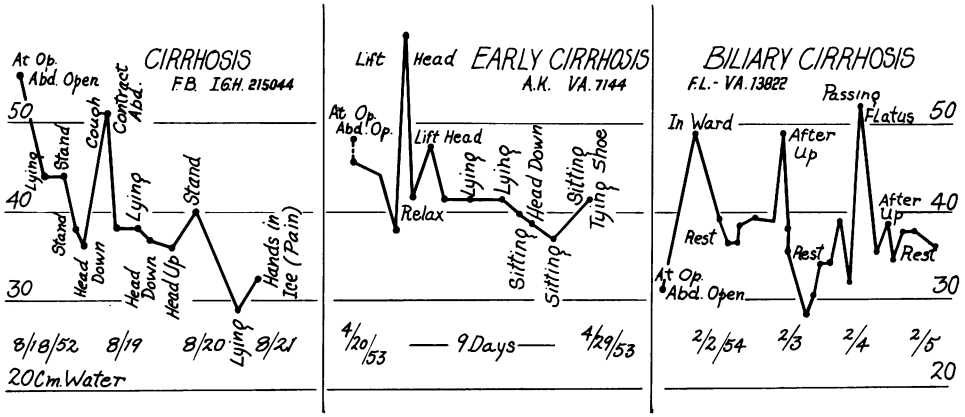

FIG. 3. Graphs showing portal pressures in three cirrhotic patients. The changes produced by drugs, foods or external influences were identical with those induced in the "normals." Note the variations in the hypertensive levels and the tendency of that in the first patient (F. B.) to return to normal from quite an elevated pressure.

brought about by this mechanical means seemed to be somewhat less in the cirrhotics than in the normals. Thus both types tended to reach an "effort" plateau of around 55 cm. regardless of the previous resting level. Our only explanation for this was that with any increase in portal pressure, nature must bring into play collateral vessels which tend to limit the increase to the neighborhood of 55 cm.*

No significant portal pressure differences were noted with the use of drugs on cirrhotics versus normals. They both responded similarly and for the most part insignificantly. One interesting pressure change was noted in the cirrhotic group. The portal pressures of all individuals decreased somewhat postoperatively. There were five such cases which had an average drop in pressure of 12 cm. in the immediate postoperative days. The most remarkable change was noted in patient F. B. in the first graph, Figure 3. This patient was explored to obtain a liver biopsy. He had had ascites but no gastro-intestinal hemorrhage. We were therefore surprised to find that the portal pressure was 55 cm. As indicated, this dropped to a normal range (30 cm.) during the immediate postoperative days. The explanation of this is a mystery, but it is thought that it fits into our concept that, regardless of their level, portal pressures are quite labile and by no means fixed. It seems quite probable that at least some pressures are hypertensive at one time and normal a week later.

The foregoing discussion suggests another unsolved riddle of the portal system, namely, the frequency of varicosities and bleeding in the upper end of the gastro-intestinal tract as compared to the lower. When hemorrhage occurs in "portal hypertension," it is the lower esophagus and adjacent stomach to which the accusing finger points. It seems strange, indeed, that the lower bowel does not have these violent hemorrhages, since it is drained by the same system and thus must have the same venous pressure. In fact, with the patient in the upright position, pressures in the rectal and sigmoid tributaries must be 20 to 30 cm. greater by virtue of the added hydrostatic head.

Hemorrhoids have long been thought to be associated with cirrhosis, and suggest an increased portal pressure. Of course, any patient may have hemorrhoidal varicosities and an occasional patient with portal hypertension is seen who also has hemorrhoids. We have yet to see a portal hypertensive patient present himself with a chief complaint

* Since the preparation of this discussion we have studied a cirrhotic patient who could repeatedly elevate his portal pressure to 96–98 cm. by severe coughing.

of hemorrhoids. Other writers[16] have made the same observation.

We tried to explore this question further. In patients operated upon for hemorrhoids, after full sphincter dilation, we were able to cannulate several of the thin-walled varicosities. Venous pressures were taken in these and the patient was then tipped head up and pressures were again taken.

This is an exasperating procedure, for the needle or cannula invariably seems to penetrate both walls of the short vein segment or become dislodged on moving the patient to a new position. Out of some two dozen such attempts there were four trials that seemed valid. These demonstrated a supine pressure which approximated the expected portal pressure (25 cm. of water). Sitting the patient upright increased the pressure roughly 20 cm.

This is in keeping with changes in venous pressure noted by others.[4, 17] They are directly proportional to the hydrostatic column of blood acting at the particular site. To this must be added the factor of muscular contraction of the abdominal wall. Our observations merely indicate that veins of the sigmoid and rectum contain an added hydrostatic pressure over that of the portal vein. They do not explain the freedom of this area from varicosities and exsanguinating hemorrhage. The conclusion seems inevitable that increased portal pressure alone has little, or maybe nothing, to do with gastro-intestinal hemorrhage.

PORTAL PRESSURE AND RESPIRATORY MOVEMENTS

The pumping action of the diaphragm on blood returning to the chest is known by all. It is also well known that the pressures caused by this pumping action may become quite considerable with forced respiration or violent contractions of abdominal musculature and diaphragm. During quiet respiration the pressure below the diaphragm reaches 8 cm. of water, while that just above the diaphragm reaches a negative 8 cm. The resultant effective force would then be 16 cm. This tends to pump blood upward through the diaphragm. It is not only effective in the inferior vena cava but also in the veins of the cardiac end of the stomach which communicate with those of the esophagus. The figures given are admittedly small and should be of little significance. However, under forced situations these pressures may become extremely large. The resulting pumping action might be great enough to augment any tendency to bleed or form varicosities in this critical area. It seemed desirable then to determine the extremes which might be reached by a positive pressure in the upper abdomen and the negative pressure in the thorax, separated only by the diaphragm.

Other authors[9, 14] have indicated that a positive pressure of 135 cm. of water may be registered in the thorax during a Valsalva procedure. During a reverse Valsalva a negative pressure of 108 cm. might be attained. It must be remembered that these pressures are directly transmitted upon the esophagus and thence to its veins. All negative thoracic pressures must be considered in the light of what they may do to the esophageal veins. Further, it must be emphasized that a negative pressure of 50 cm. outside a vein has exactly the same effect as 50 cm. of pressure inside the vein.

It seemed important to measure directly the pressures to which the stomach and esophagus might be subjected. Therefore, tests were carried out on "volunteers" (patients and residents) who swallowed a small rubber balloon filled with mercury. This was connected by a small hard rubber tube to a manometer. All manometric readings were made in the fluoroscopy room where the position of the mercury bag could be constantly checked.

The results of these determinations are shown graphically in Figure 4. Those for normal respirations show minimal pressure changes and are indicated by the dotted line near the center. The other lines are maximal

averages. They therefore represent values which are somewhat higher than might be expected with the usual cough, choking or hiccough. It would be well to remember that these conditions could, in their worst forms, duplicate the figures shown even though they might be effective for only short periods of time.

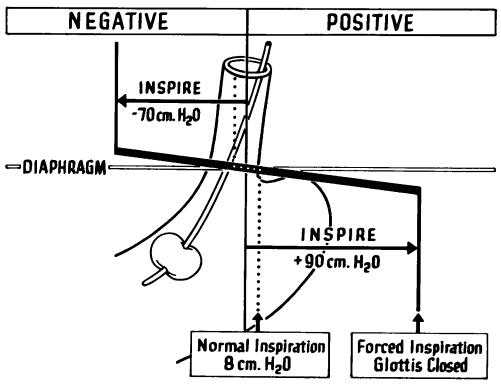

FIG. 4. Pressures induced in esophagus and stomach upon a mercury bag are diagrammatically indicated. Pressure below the diaphragm is always positive, while that with a reverse Valsalva is negative in the esophagus. As indicated, the combined effective venous pressure of forced reverse Valsalva is 70 plus 90 cm. of water. This completely dwarfs the usual recorded portal hypertensive pressure and affects the exact site where hemorrhage and varices occur.

An average negative pressure of 70 cm. was obtained in the esophagus with a reverse Valsalva (inspiration with glottis closed). As the mercury bag passed down the esophagus under continued influence of the reverse Valsalva and under fluoroscopic vision, the negative pressure remained unchanged. Suddenly and dramatically on passing through the diaphragm the pressure changed from a negative 70 cm. to a positive 90 cm. With the bag below the diaphragm the pressure remains positive at all times regardless of what respiratory or muscular efforts are brought into play. Likewise, there is no difference in this positive pressure high in the cardia beneath the diaphragm or near the pylorus.

When the maximum positive pressure below the diaphragm is added to the maximum negative pressure above the diaphragm, a tremendous aggregate pressure results. According to our determinations this averaged 160 cm. of water. These are maximal readings but even half their value is much greater than the highest pressure ever recorded in portal hypertension. Therefore, this effect on portal pressure cannot be considered lightly.

These figures indicate effective pressures which completely overshadow increases seen in portal hypertension. The slight increase of the abnormal over the normal pressure becomes insignificant. The hoped for 10 cm. drop in pressure following a portal shunt seems useless. Portal hypertension itself as a cause for varices and hemorrhage thus disappears before a dominant differential pressure resulting from normal physical factors. Furthermore, it is more than coincidence that this tremendous pressure change occurs just at the site where hemorrhage and varices are most frequently encountered.

DISCUSSION

These studies have brought us no closer to an explanation of the cause of portal hypertension or the diseases which are attributed to it. Certainly its origin cannot be explained on the simple basis of external or internal hepatic obstruction to the portal vein. This early concept must be questioned or completely disregarded as a result of the experimental work of Child and others.[6, 7, 15] Also the many ligations of the portal vein which have been done without incident seem completely to confirm these experimental investigations. We must, therefore, conclude that if the portal vein of man is occluded either in the liver or outside, there will be an abrupt rise in portal pressure to 40 or 50 cm. of water. It can likewise be assumed that four or five days after this rise, the pressure will again start to fall and will reach normal about the tenth or fourteenth day. There seems to be no reason to suspect that a gradual occlusion would act differently. The return to normal pressure two weeks after a portal occlusion undoubtedly must be the result of the formation of new

collaterals. A gradual occlusion, as occurs in cirrhosis, should form that many more collaterals. The return to normal should be just as certain.

It is this particular phase which is mystifying. Why does one patient carry double the portal pressure of another? Why do most, but not all, portal hypertensives have cirrhosis and conversely, why do many, but not all, cirrhotics have elevated portal pressures?

The present study indicates several facts which have so far been little appreciated. First of all, there is a very wide range in the normal portal venous pressure. What must now be considered normal pressures differ little from what formerly had been considered abnormal. Thus, arbitrarily, 30 cm. of water might be chosen for the upper limits of normal, and a hypertensive level indicated by 40 cm. or above. This leaves a debated pressure level of 10 cm. dividing normal from abnormal.

Ten centimeters of water pressure represents a column of water only four inches high. It is an amount which is completely overshadowed by portal pressure increases effected by a cough in a normal patient. When the patient coughs, chokes, or otherwise exerts his abdominal musculature, his portal pressure may be expected to quickly span this insignificant range and extend high into hypertensive levels.

A second factor which adds doubt to the importance of portal hypertension is the dynamic force which may be exerted in the region of the cardia and esophagus. As has been pointed out, here is a pressure gradient which can reach 160 cm. of water. This figure completely dwarfs the increase of 10 or 20 cm. separating normal from abnormal pressure.

Portal pressures which have formerly been considered dangerously elevated fade into insignificance when compared with those produced by normal physiological forces. These dominating pressures are seen in "normals" as well as in "portal hypertensives." With the exception of those cases reported by Jahnke,[11] the possible decrease in portal pressure which can be gained by vascular shunt is minute. Unless large decreases can be anticipated, such shunts are of questionable value.

SUMMARY

An attempt has been made to study the effects of external influences upon portal pressure. This was done by means of a small plastic tube placed in the portal vein. Pressures were taken over a period of several days in order to study the natural variations in portal pressure.

No startling natural variations were found. There were slight but unimportant changes resulting from food and drugs. Pressures were far from constant in any given patient.

A most important elevation in portal tension occurred as a result of forced thoracic or abdominal muscular activity. Thus, choking or a cough would send a normal portal pressure well into the hypertensive range. This elevation takes place in both the normal and hypertensive patient. It causes both to approach a pressure of 55 cm. of water.

Forced inspiration with the glottis closed (reverse Valsalva) produces a tremendous drive which may force blood from the veins of the cardia into the veins of the esophagus. The effective pressure thus produced might reach 160 cm. of water. This completely dwarfs any recorded "portal hypertension." Since this induced tension may be produced in both normal as well as hypertensive patients, some doubt is cast upon whether portal hypertension, as described, has much effect on the varices or hemorrhages attributed to it.

The cause of persistent increased pressure in portal hypertension is unknown. It is not likely the result of a simple intra- or extrahepatic obstruction.

BIBLIOGRAPHY

[1] Bellis, J. C.: Portal Venous Pressure in Man. Proc. Soc. Exper. Biol. and Med., **50:** 258, 1942.

[2] Blakemore, A. H., and J. W. Lord, Jr.: The Technique of Using Vitalium Tubes in Establishing Portacaval Shunts for Portal Hypertension. Ann. Surg., **122**: 476, 1945.

[3] Blakemore, A. H.: The Portacaval Shunt in the Surgical Treatment of Portal Hypertension. Ann. Surg., **128**: 825, 1948.

[4] Burch, G. E.: A Primer of Venous Pressure. Lea and Febiger, Philadelphia, 1950.

[5] Child, C. G., III, R. F. Milnes, G. R. Holswade and A. L. Gore: Sudden and Complete Occlusion of the Portal Vein in the Macaca Mulatta Monkey. Ann. Surg., **132**: 475, 1950.

[6] Child, C. G., III, G. R. Holswade, R. D. McClure, Jr., A. L. Gore and E. A. O'Neill: Pancreaticoduodenectomy with Resection of the Portal Vein in the Macaca Mulatta Monkey and in Man. Surg., Gynec. and Obst., **94**: 31, 1952.

[7] Douglass, J. C., W. H. Mehn, B. F. Lounsbury, L. L. Swigert and C. A. Tanturi: Attempts at the Experimental Production of Portal Hypertension. Arch. Surg., **62**: 785, 1951.

[8] Fitzpatrick, H. F., A. H. Blakemore, C. McC. Smythe and S. G. Weissman: A Study of Portal Vein Pressures and Blood Flow Pre- and Post-operatively in Patients with Portal Hypertension Treated by Portacaval Anastomosis. Surg. Forum. Am. College of Surgeons, p. 161, 1951.

[9] Heaton, T. G.: Artificial Pneumothorax in Pulmonary Tuberculosis. MacMillan Co., Toronto, 1947.

[10] Hoffbauer, F. W., J. L. Bollman and J. H. Grindlay: Factors Influencing Pressure in the Portal Vein as Measured in the Intact Animal. J. Lab. and Clin. Med., **32**: 1401, 1947.

[11] Jahnke, E. J., E. D. Palmer, V. M. Sborov, C. W. Hughes and S. F. Seeley: An Evaluation of the Shunt Operation for Portal Decompression. Surg., Gynec. and Obst., **97**: 471, 1953.

[12] Linton, R. R., C. M. Jones and W. Volwiler: Portal Hypertension. S. Clin. North America, **27**: 1162, 1947.

[13] Linton, R. R., A. B. Hardy, Jr., and W. Volwiler: Portacaval Shunts in the Treatment of Portal Hypertension. Surg., Gynec. and Obst., **87**: 129, 1948.

[14] MacLeod, J. J. R.: Physiology in Modern Medicine. C. V. Mosby, St. Louis, 1941.

[15] Morris, A. M., and H. H. Miller: Chronic Portal Vein Occlusion and Portal Hypertension in the Dog. Surg., **30**: 768, 1951.

[16] Nesselrod, J. P.: Proctology in General Practice, p. 69, Saunders, Philadelphia, 1950.

[17] Pierce, V. K., C. P. Boyan and J. G. Masterson: Studies on Venous Blood Pressure in Patients Undergoing Major Surgical Procedures. Surg., Gynec. and Obst., **96**: 310, 1953.

[18] Sappey: Recherches sur un point d'anatomie pathologique relatif 'a l'histoire de la cirrhose. Bulletin de l'Académie de Medicine. Paris, 1859, Tom XXIV.

[19] Schnabel, J. G., Jr., H. F. Fitzpatrick, L. H. Peterson, W. J. Rashkind, D. Talley, III, and R. L. Raphael: A Technique of Vascular Catheterization with Small Plastic Catheters. Circulation, **5**: 257, 1952.

[20] Smythe, C. McC., H. F. Fitzpatrick and A. H. Blakemore: Studies of Portal Venous Oxygen Contents in the Unanesthetized Man. J. Clin. Invest., **30**: 674, 1951.

[21] Taylor, F. W., and H. L. Egbert: Portal Tension. Surg., Gynec. and Obst., **92**: 64, 1951.

[22] Thompson, W. P., J. L. Caughey, A. O. Whipple and L. M. Rousselot: Splenic Vein Pressure in Congestive Splenomegaly (Bantis Syndrome). J. Clin. Invest., **16**: 571, 1937.

[23] Whipple, A. O.: The Problem of Portal Hypertension in Relation to the Hepatosplenopathies. Ann. Surg., **122**: 449, 1945.