The Distensibility Characteristics of the Portal Vascular Bed

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The distensibility of the splanchnic portion of the portal system has been evaluated by studying pressure and volume changes with or without any interruption of blood flow. The results of these determinations indicate two types of distensibility of the system: (1) a rapid elastic distension representing relatively small volumes, and (2) a secondary phase of slow distension, designated "delayed compliance," which represents a significantly greater volume change. It is concluded that delayed compliance is of major importance in the pooling of blood in peripheral blood vessels under conditions of maintained elevation of venous pressure.

If THE venous side of the circulatory system is very distensible, as is commonly assumed, then the venous pressure should play a significant role in determining the volume of blood "pooled" in peripheral venous channels. In the case of the portal system of the dog, acute elevation of portal pressure may lead to fatal shock, presumably associated with such a process of blood pooling. In the femoral venous system, however, it was found that acute elevations of venous pressure do not lead to the pooling of any significant volume of blood. It therefore appeared pertinent to investigate the possibilities of blood pooling in the splanchnic system associated with acute changes in portal venous pressure.

METHODS

All data were obtained from acute experiments upon heparinized dogs anesthetized with sodium barbital following sedation with morphine. In studies of the intact portal system the same surgical approach was employed as we have described elsewhere. In brief, the preparation involves splenectomy and then passage of a brass tube, 4 mm. in internal diameter, down the splenic vein into the portal vein. By means of a loose cord ligature held permanently in position, the entrance of the portal vein into the liver may be occluded at will so as to divert the portal blood out the cannula and thence to a flowmeter. It should be emphasized that normal portal-hepatic flow is maintained during the intervals between brief determinations of portal blood flow. An optical manometer connected to a side tube of the portal cannula records the portal pressure. By means of a second manometer recording the intra-abdominal pressure adjacent to the portal vein via a fluid-filled system, the portal pressure is corrected for the extravascular pressure to yield the "effective portal pressure."

Venous outflows were recorded with a strain gage flowmeter from an outflow tube whose hydraulic level could be suddenly shifted from one level to another. A compensating reservoir connected to the arterial system served to maintain arterial pressure constant in spite of the transient loss of blood associated with venous flow determinations.

Arterial inflows were also measured in one series of experiments by leading blood from a carotid artery to a bubble flowmeter and then through a "Y" connection into cannulas inserted into the celiac and superior mesenteric arteries. To avoid exposure and trauma to the abdominal viscera in cannulating the latter vessels, they were approached transthoracically after resection of the tenth and eleventh ribs on the left. The hepatic artery was ligated in the process of performing the cannulation, but there were numerous other routes of collateral arterial inflow as well as venous drainage which were not accounted for by this method. Nevertheless, it was found that in a series of 28 determinations on six dogs, inflow values were within 15 per cent of the outflow values in 68 per cent of the determinations, suggesting that, while not strictly quantitative, this technic should reveal any gross discrepancies in the measured flow relationships in the splanchnic bed.

In a final series of experiments in which it proved desirable to eliminate collateral circulation, a length of about 50 cm. of ileum was isolated by placing...
mass ligatures around both ends and tying off all other vessels except the main mesenteric artery and vein supplying the loop, taking care to damage as few nerves as possible. The vein was cannulated and the blood led out an open tube maintained at a constant hydrostatic level which delivered the blood to a collecting funnel leading into a jugular vein. A "T" connection in the venous cannula was used for recording pressure and a "T" connection in the outflow tubing was used for shunting the blood to a flowmeter or for performing controlled injections. After preparing this loop, it was carefully wrapped in gauze soaked with saline and kept at body temperature by the heat of a lamp placed over the preparation.

![Fig. 1. Example of original recording used to measure portal distensibility without interruption of blood flow as described in text. Flowmeter tracing ascending from left to right indicates cumulative outflow, pulse tracing indicates arterial pressure recorded from the iliac artery. At "A," portal pressure and flow had been equilibrated at a pressure of 20 cm.; at point "X" (signaled by disappearance of horizontal trace just above base line) portal pressure was lowered from 20 cm. to 10 cm. "D. V." indicates excess volume appearing in outflow at the lower pressure due to elastic distension of the system at the higher initial pressure. To simplify the illustration, recordings of the portal and extraportal pressures were omitted from this recording. Base line indicates time intervals of 1 second.](image)

In the isolated loop with collaterals excluded, it was possible to study distensibility of the vascular bed more directly by temporarily occluding inflow and outflow and then injecting or withdrawing known volumes of blood at controlled rates. This was accomplished with a syringe mounted in a rigid clamp and actuated by a screw mechanism driven by a crank. To assure smooth mechanical action, a mineral oil trap was inserted between the syringe and the injection cannula so that advancing the plunger of the oil-filled syringe served to displace blood from the trap into the preparation. Movement of the plunger and, therefore, the volume of blood injected or withdrawn were accurately recorded by coupling a rack and pinion to the plunger which actuated a potentiometer. The latter constituted one arm of a Wheatstone bridge whose degree of unbalance was recorded with a galvanometer.

**RESULTS**

The method initially employed for evaluating portal distensibility was adapted from that previously used in studying the femoral bed, as illustrated in figure 1. The flowmeter tracing on this record is represented by the heavy line ascending from left to right, the rate of ascent of this line indicating the rate of portal flow. After portal pressure had been allowed to equilibrate at a level of 20 cm. of blood, a flow measurement at "A" revealed a flow rate of 113 cc. per minute. At point "X," portal pressure was dropped to 10 cm. by suddenly lowering the outflow tube. Following this there is a sudden increment of blood flow which then re-equilibrates and appears to stabilize at "B" with a flow rate of 205 cc. per minute. The dashed line extrapolated from "X" and parallel to the flow rate at "B" indicates the steady flow rate minus the transient increment of outflow observed at the moment that the pressure was lowered. This sudden gush of excess blood with lowering of the pressure, as measured by "DV," is assumed to represent the volume of blood which was "pooled" in the portal system by distension of the vessels at the higher pressure, and which appears in the outflow when the pressure is lowered. The theoretic basis for and limitations of this method have been discussed previously.

By making successive determinations of this type at overlapping pressure intervals from 0 to 30 cm. of blood, it was possible to construct conventional distensibility plots of the splanchnic portion of the portal system as shown in figure 2. The values obtained in these three animals are representative of data which have been obtained upon 18 dogs employing this technic. The important point to note in figure 2 is that, for the entire splanchnic bed, the total volume change is less than 15 cc. over a pressure range in excess of the normal physiologic limits. In no animals were distensibility volumes of over 20 cc. measured by this method. In terms of blood pooling, this would mean that a rise in portal pressure to
lethal levels could account for pooling of less than 2 per cent of the total blood volume of a dog in the splanchnic circuit, an amount which could have relatively little hemodynamic consequence.

Suspicions that these data did not reveal the whole story were aroused by certain quantitative discrepancies in the data. Most dogs revealed a volume-pressure diagram that was linear or curvilinear with concavity toward the pressure axis, in contrast to the relationship of convexity to the pressure axis as should be anticipated. In addition, successive determinations revealed a failure to achieve the good quantitative reproducibility that had been observed when this method was applied to the femoral bed, and the "equilibrated" flows before and after the suddenly induced pressure change were not explainable on the basis of the small changes in arteriovenous pressure gradient.

The presence of some discrepancy is further illustrated in figure 3 in which the pressure was first raised and then returned to the control level while flows were recorded. The initial elevation of pressure is accompanied by a brief cessation of outflow (pooling), while the lowering of pressure is accompanied by a transient marked increase in blood flow (drainage of pooled blood). It is noteworthy, however, that following these transient changes the flows do not completely stabilize, a factor which is emphasized particularly at the right hand end of the figure where flow is still elevated over the control in spite of the fact that the original pressure had been restored.

To explore the possibility that this outflow method was not accurately revealing the pressure-volume relationships within the splanchnic bed, simultaneous inflow and outflow measurements were obtained, as illustrated by the experiment shown in figure 4. Initially the inflow and outflow were nearly identical, indicated by a flow ratio approximating unity. With the sudden lowering of portal pressure, a sudden gush of blood was observed in the outflow as has been described above, resulting in an outflow/inflow ratio far in excess of 1.0. For a considerable period thereafter, however, there continues to be a marked discrepancy between inflow and outflow, the outflow remaining well in excess of the inflow for a period of almost two minutes during which the observations were continued. The converse observation was made when the pressure was suddenly elevated, revealing an extreme initial deficit in outflow followed by a resumed outflow which remained significantly below the inflow for an extended period. Because of quantitative errors in the exact flow values and the asymptotic manner in which the outflow gradually approaches the inflow, the exact magnitude and duration of this effect could not be quantitated, but significant differences
between inflow and outflow have been observed for as long as five minutes after the pressure change.

These observations made upon the intact splanchnic vascular bed, without interruption of its blood supply and with controlled arterial pressure so as to confine most of the volume-pressure changes to the venous side, all point to the suggestion that there are two processes involved in the distension of the portal bed. Sudden pressure changes produce transient augmentations or diminutions of portal flow which represent pooling of relatively small volumes of blood, followed by a secondary phase of storage or drainage of a significantly larger volume of blood which equilibrates slowly with portal pressure.

Since all the foregoing evidence was obtained upon the intact splanchnic circulation in which collateral avenues for blood flow were not controlled, the experimental attack was shifted to the intestinal loop preparation in which the possibility of distortion of the data by such collateral flow was excluded. Experiments such as those reproduced in figures 2 and 3 were repeated on these intestinal loop preparations, yielding results that were so nearly identical to those found in the intact splanchnic system that they do not warrant description here. With the intestinal loop preparation, moreover, it became readily possible to reverse the form of the experiment and study changes in pressure with known changes in volume.

A preliminary experiment of this type was performed by suddenly occluding the arterial inflow and venous outflow and then rapidly injecting blood into the venous end of the preparation. The changes in pressure resulting from such a rapid injection of 3 cc. of blood are illustrated in figure 5. With such an injection there is initially a very steep rise in venous pressure to high levels. Simultaneous recording of the peripheral arterial pressure in such preparations indicated that this initial pressure peak was transmitted throughout the system in less than one second. For a considerable period after this initial peak, however, there is a slow but progressive decline in pressure persisting for several minutes after the injection was completed, in spite of the fact that arterial inflow channels and venous outflow channels remained occluded. Evidence that this was not dependent upon significant loss of fluid from the vascular bed was obtained by repeated injections and withdrawals of the same volume of blood. This could be repeated up to six times in sequence with only a slight deficit in pressure following the last withdrawal as compared with the pressure existing prior to the initial injection.

To accomplish injections and withdrawals of blood under more carefully controlled con-
ditions, an intestinal loop was prepared and, as should be emphasized, normal flow of blood through the loop was maintained except for the brief intervals of less than 30 seconds during which the injections were made. In addition, in view of the earlier results, the precaution was taken of allowing pressure-flow relationships to equilibrate for 10 minutes before occluding the circulation to perform an injection. Arterial inflow was then cut off and, after allowing three seconds for venous drainage and pressure equilibration from the arterial side of the bed, the venous channel was occluded. Blood was then injected and withdrawn from the venous end of the preparation at constant rates while recording pressures. The open circles in figure 6 illustrate such an experiment in which the injection was performed at the rate of 1 cc. per second. It is obvious that the injection pressure curve differs markedly from the withdrawal pressure curve; at the physiologic range of 15 cm. pressure the withdrawal curve exhibits a volume increment of approximately three times the volume increment necessary to achieve this pressure on the initial injection.

Further evidence of the nature of this change was obtained by immediately repeating the injection and withdrawal following the completion of the initial cycle. As illustrated by the solid circles labeled “2” in figure 6, the second injection immediately following the first shows some shift of the distensibility diagram over towards the withdrawal curve. In this preparation the two withdrawal curves happened to be superimposable. Such duplication of withdrawal curves was not uniformly observed, although the withdrawal curves did tend to be much closer together than were the injection curves. It should be noted that there is only a minor shift in the pressure recorded at “zero” volume, indicating that loss of fluid from the vascular bed during the brief exposures to high pressure was contributing a very minor part to the picture.

As could be predicted from figure 5, if the maximal volume was allowed to remain in the vascular bed for an interval before the withdrawal was started, a decay in pressure at this constant volume was observed and a much greater shift in the withdrawal curve was observed. Extended exposures of the vascular bed to high pressures, however, resulted in evidence of fluid loss from the system so that the quantitative validity of the results so obtained could not be trusted.

**DISCUSSION**

These data demonstrate that the splanchnic portion of the portal system exhibits two types of distensibility. A rapidly equilibrating component exhibits the properties which are to be expected of an elastic system, and presumably relates to the elasticity of the blood vessels. For reasons discussed earlier, it is logical to assume that in the first series of experiments the measured distensibilities were obtained dominantly from the venous channels. The relatively small volume represented by this elastic distension is in accord with in vitro studies of veins which have shown them to be only moderately distensible.

In addition, there is a slowly equilibrating distensibility volume which is quantitatively of far greater importance, and therefore appears to be the mechanism which could play a significant role in passive blood pooling. A variety of terms have been employed to
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identify this type of slow yielding of biologic systems to changes in an applied force, but many are either ambiguous or involve assumptions of specific physical mechanisms. To describe the slow change in volume of the vascular bed to a sudden change in pressure, we have selected the less specific term of "delayed compliance." In isolated blood vessels studied in vitro, delayed compliance has long been known, having originally been described over 100 years ago. The present report, together with the closely comparable study of the pulmonary vascular bed by Sarnoff and Berglund, appear to be the first clear demonstrations of delayed compliance in an entire vascular bed. The data in both of these studies suffer from some quantitative distortion due to leakage of fluid out of or into the capillary bed with changes in pressure. In both studies, however, such fluid leaks are capable of explaining only a small fraction of the delayed compliance effect, in view of the ability to recover the volume injected at high pressure without evidence of any marked displacement from the original pressure-volume reference level.

Attempts have frequently been made to explain the delayed compliance process in terms of some type of viscosity effect or in terms of a relaxation of vascular muscle tone. In reference to the peripheral vascular bed, however, at least three processes must be considered as capable of contributing to delayed compliance: (1) a visco-elastic effect in the walls of the blood vessels, (2) opening or closing of a fraction of the peripheral vessels with a change in pressure according to the "critical closure" hypothesis of Burton, or (3) a change in the active tension developed by the vascular musculature. An interrelationship between viscoelastic effects and the tension in the vascular musculature is suggested by the plastic behavior of smooth muscle as described by Bozler and others.

Recognition of the delayed compliance phenomenon raises difficulties in defining the volume-pressure diagram for a vascular bed. Physiologic variations are usually slowly superimposed on a vascular bed that is already under a moderate degree of pressure-volume distensibility and which, therefore, has had time to develop some increase in volume due to delayed compliance. This obviously excludes the pressure-volume diagram obtained by starting at zero pressure as being representative of normal physiologic relationships. On the other hand, increasing pressure from moderate levels to high levels yields still further increase in volume by delayed compliance. Therefore, to describe the normal relationship in terms of the descending curve, obtained by withdrawing fluid from a bed subjected initially to a high pressure, would also be inaccurate. To pick an average between the two extremes is a poor compromise. More accurately, one must visualize a whole spectrum of curves between the two extremes. At any moment, minimal pressure-volume changes will be expected to follow that curve in this spectrum of pressure-volume diagrams which corresponds with the stabilized pressure level existing initially. With larger changes, however, delayed compliance will occur to shift the pressure-volume diagram over to another member of this spectrum, provided there is enough time for the delayed compliance to develop. In short, the distensibility of the peripheral vascular bed must be defined by three variables. Changes in pressure will result in changes in intravascular volume as a function of (a) the previously existing pressure, (b) the magnitude of the pressure change, and (c) the length of time over which the new pressure is allowed to act.

In terms of blood pooling mechanisms, it is obvious that pooling may result passively from a rise in venous pressure through the delayed compliance process, provided the elevated venous pressure is maintained for a sufficient length of time. Thus Selkurt and co-workers demonstrated that elevation of portal pressure to 25 cm. saline results in a progressive blood pooling over a period of about 20 minutes, as evidenced by a progressive fall in arterial blood pressure to markedly hypotensive levels. On the other hand, we have frequently observed transient elevations in portal pressure far in excess of this value associated with various physiologic stimuli, which appear to have little significant effect on circulatory dynamics provided that the
period of elevated venous pressure is followed promptly by a return to normal venous pressure. In other words, by virtue of the relatively small elastic distensibility of the venous vasculature, the animal is protected against significant degrees of blood pooling because of transient elevations in venous pressure. However, maintained elevation of venous pressure, at least in the splanchnic bed, results in pooling of significant volumes of blood by virtue of the delayed compliance of the vascular bed.

**Summary**

1. In intact splanchnic beds and in isolated intestinal loops of anesthetized dogs, measurements of the distension of the vascular bed associated with increases in portal pressure have been made without interruption of the normal blood supply.

2. The results indicate that sudden changes in portal pressure produce an elastic distension of the blood vessels which represents a very small volume of blood. With maintained pressure, however, there is evidence of a much larger volume of blood “pooled” by a process that has been designated as “delayed compliance.”

3. Since the arterial pressures were held constant in these earlier experiments, it is assumed that peripheral venous channels are making the greatest contribution to the immediate elastic distension and the secondary delayed compliance.

4. Further evidence of delayed compliance in the peripheral vascular bed has been obtained by injecting and withdrawing blood in intestinal loops with the circulation temporarily occluded.

5. It is concluded that the peripheral vascular bed exhibits relatively little elastic distensibility and thereby avoids pooling of blood in the periphery with sudden transient pressure changes. With maintained elevations of venous pressure, however, the delayed compliance process in the vascular bed could result in significant pooling of blood.

**References**


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Circ Res. 1953;1:271-277
doi: 10.1161/01.RES.1.3.271

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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