Studies in the Hemodynamics Following Supradiaphragmatic Constriction of the Inferior Vena Cava *

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Introduction

IN AN EFFORT to perform more physiologic surgery for the effects of portal hypertension such as ascites, a study of the hemodynamics of supradiaphragmatic constriction of the inferior vena cava was undertaken.

Following supradiaphragmatic constriction of the inferior vena cava in the dog, an extensive collateral circulation develops which bypasses the constriction.[10] The general failure of attempts to produce permanent portal hypertension by this method has been attributed to the development of these porto-systemic and inferior vena caval-systemic collaterals. Despite the emphasis placed upon the importance of the porto-systemic collaterals, one is impressed by the absence of visible portal collateralization at laparotomy in the dog. The present study was therefore devised to investigate the relative importance of porto-systemic and inferior vena caval-systemic collaterals in this experimental animal. Since it is difficult to measure flow directly through collateral channels, this study was designed to evaluate this in an indirect manner.

Method

Twenty-two adult mongrel dogs weighing between 14 and 20 kg. were anesthetized with intravenous thiopental (30 mg/kg). Two series of experiments were performed in both control dogs and dogs in which supradiaphragmatic constriction of the inferior vena cava had been performed one to four months previously.

In the first series, pressures were recorded in the portal vein, hepatic artery and inferior vena cava (IVC) through a laparotomy incision. Five "experimental" and six normal control dogs were used in this series of experiments. All pressure determinations were obtained using polyethylene tubing connected to Statham strain gauges and recorded on a Sanborn 550M multi-channel recorder. The zero level for the strain gauges was a point two inches cephalad to the sternoclavicular junction on the right external jugular vein. The abdomen was entered through a midline incision (Fig. 1) after introducing catheters into both the femoral artery and vein. The porta hepatis was exposed and a ligature placed loosely about the portal vein just above the entrance of the gastroduodenal vein. The silk ligatures placed about all specified vessels were used as a means of completely occluding these vessels during the study. A ligature was applied about the hepatic artery approximately two centimeters distal to the celiac axis. Another ligature was placed about the IVC cephalad to the origin of the renal veins. The femoral vein catheter was then advanced to a point just caudal to the ligature about the IVC. A catheter was inserted into one of the splenic veins and advanced into the portal

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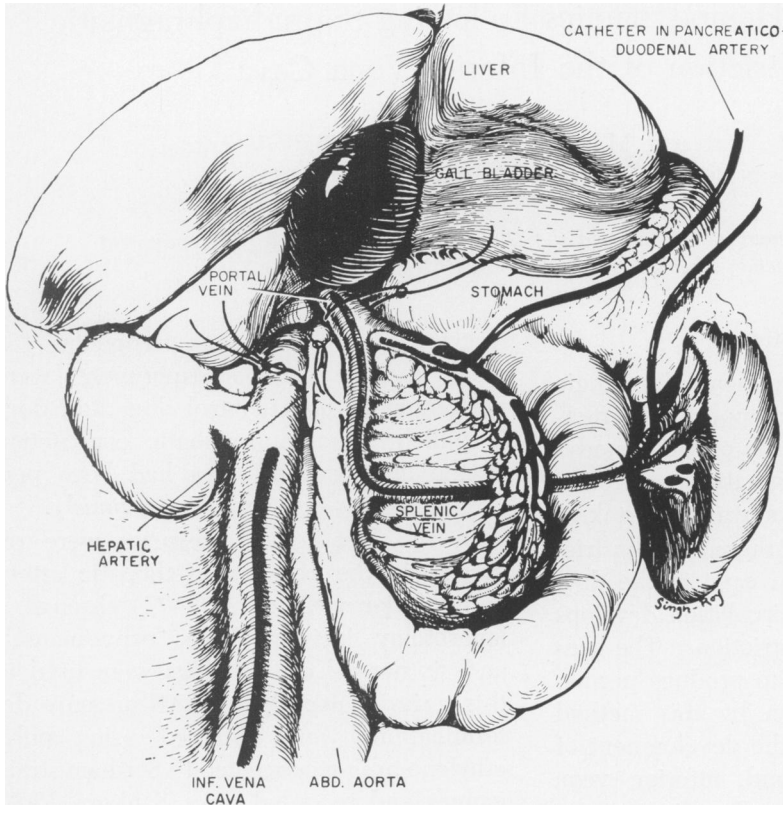

FIG. 1. Laparotomy series. The placement of catheters within the inferior vena cava, portal vein and pancreatoduodenal artery as well as the ligatures for vascular occlusion is demonstrated. These polyethylene catheters are connected to individual Statham strain gauges and maintained patent by a slow infusion of heparinized saline.

vein to a point just above the ligature on this vessel. A segment of superior pancreatoduodenal artery was isolated within the substance of the pancreas and a catheter was threaded through this vessel to the porta hepatis.

After recording baseline pressures within the femoral artery, IVC, portal vein and hepatic artery, the portal vein was occluded. Pressures were recorded immediately and repeated after three minutes of continued portal occlusion. In those cases in which shock rapidly developed, occlusion was maintained for only one and one-half minutes. After re-establishment of the baseline state, a bull-dog clamp was placed upon the hepatic artery at its identifying ligature. Pressures were immediately recorded and repeated after three minutes of continued hepatic arterial occlusion. Following removal of the clamp, the pressures were allowed to return to baseline level. The hepatic artery and portal vein were then occluded simultaneously. Pressures were recorded immediately and after one and one-half or three minutes of continued occlusion. Upon release of the occlusion and the return to the baseline level, the IVC was occluded and pressures recorded immediately and after three minutes of continued occlusion.

In the second series, pressures were recorded within the IVC and azygos vein through a thoracotomy incision (Fig. 2). Six dogs with vena cava constricted and five normal control dogs were used in this experiment.

Catheters were introduced into the femoral artery and vein. The femoral vein catheter was advanced into the abdominal portion of the IVC. The thoracic cavity was then entered through the fourth right interspace (Fig. 2) and a heavy silk ligature was placed loosely about a segment

of IVC just caudad to the right atrium. A catheter with an appropriately curved multiperforated tip was introduced through the external jugular vein into the superior vena cava. The tip of this catheter was then guided under direct vision into the azygos vein and advanced to a point just beyond the superior intercostal vein. A ligature was then placed loosely around the azygos vein proximal to the intercostal vein.

Baseline pressures were recorded within the femoral artery, IVC and azygos vein. The azygos vein was occluded and simultaneous pressure recordings were made in all vessels. After three minutes of continuous azygos occlusion, pressures were again recorded. The ligature on the azygos vein was released and pressures allowed to return to baseline level. A similar procedure was performed with occlusion of the IVC. Pressures were obtained after three minutes of IVC occlusion except in those cases where shock intervened. When pressures had returned to baseline level, the azygos vein and IVC were simultaneously occluded and all pressures were recorded.

Results

Laparotomy Series

Effects Upon Systemic Pressure (*Table 1*). Mean systemic pressure was lower in dogs with vena cava constriction (118 mm. Hg) than in normals (143 mm. Hg).

With portal vein occlusion in five normal dogs, two had a mean fall in femoral arterial pressure of 53 mm. Hg within one and one-half minutes, because of which the occlusion was discontinued. Three normal dogs tolerated portal vein occlusion for 3 minutes with a mean femoral arterial pressure fall of 38 mm. Hg. All dogs with vena cava constriction tolerated portal vein occlusion for 3 minutes with a mean fall in arterial pressure of 12 mm. Hg.

Suprarenal occlusion of the IVC in normal dogs caused a mean fall in femoral arterial pressure of 24 mm. Hg and no change in arterial pressure in the constricted group.

Effects Upon Portal Vein Pressure (*Table 2*). Mean portal vein pressure in normal dogs was 9.5 mm. Hg. In dogs with vena

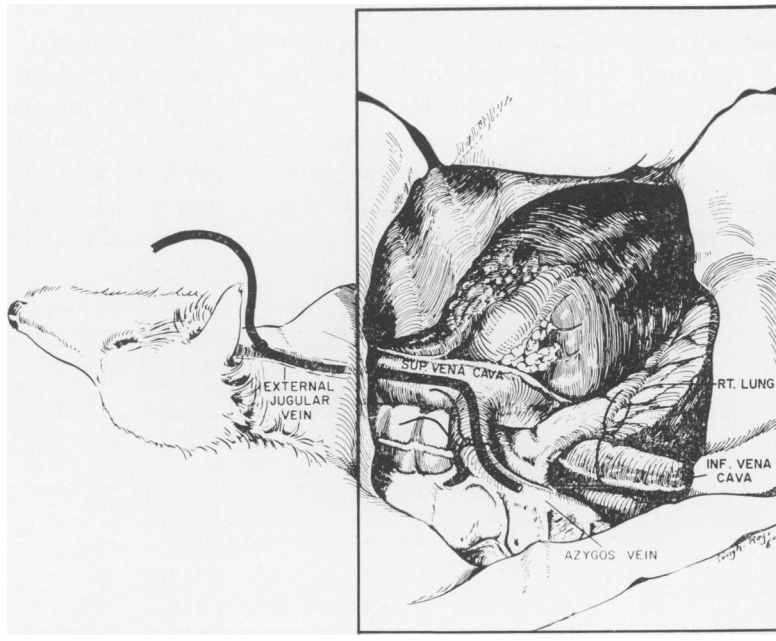

FIG. 2. Thoracotomy series. The position of the azygos vein catheter as well as the ligature for constriction of this vessel is shown. The ligature for inferior vena cava constriction is also shown but the catheter for monitoring pressure within this vessel is positioned caudal to the field of view in this diagram.

TABLE 1. *Laparotomy Series: Changes in Systemic Pressure*

	Baseline		Portal Vein Occluded 1½ min.		Portal Vein Occluded 3 min.		IVC Occluded	
	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mean mm. Hg
Normal	110 to 175	143	−45 to −60	−53	−18 to −55	−38	−15 to −35	−24
Constricted	70 to 145	118			0 to −35	−12	0	0

cava constriction, mean portal pressure was 13 mm. Hg.

Portal vein pressure was not uniformly effected by occlusion of either the hepatic artery or the IVC in either group of dogs.

Occlusion of the portal vein produced varied falls in pressure within the hepatic segment of the occluded portal vein. In the three normal dogs that tolerated occlusion of the portal vein for 3 minutes there was a mean fall in pressure of 1.6 mm. Hg. In dogs with vena caval constriction mean pressure fell 5.6 mm. Hg during the three-minute period. In two normal dogs, portal occlusion was discontinued after one and one-half minutes due to the development of profound hypotension. In this group, mean pressures fell 3.8 mm. Hg.

The addition of hepatic artery occlusion did not produce a significant further effect upon the pressure within the hepatic segment of the portal vein of dogs with caval constriction. Combined occlusion of the hepatic artery and portal vein for three minutes in three normal dogs produced a mean fall in hepatic segment–portal pressure of 3.0 mm. Hg in contrast to 1.6 mm. Hg with occlusion of the portal vein alone. In the two normal dogs that manifested shock after one and one half minutes, combined occlusion did not further alter pressure.

Effects Upon the Inferior Vena Cava Pressure (*Table 3*). In normal dogs, mean caval pressure was 5.6 mm. Hg. In dogs with caval constriction, mean pressure was 12.0 mm. Hg.

Occlusion of the hepatic artery in caval constricted dogs produced a consistent slight fall in mean caval pressure of 1.7 mm. Hg. Portal vein occlusion in these dogs produced a further fall in mean caval pressure of 2.4 mm. Hg. Simultaneous hepatic artery and portal vein occlusion resulted in

TABLE 2. *Laparotomy Series: Changes in Portal Vein Pressure*

	Baseline		IVC Occluded		Hepatic A. Occluded		Portal Vein Occluded 1½ min.		Portal Vein Occluded 3 min.		Hepatic A. & Portal V. Occluded 1½ min.		Hepatic A. & Portal V. Occluded 3 min.	
	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mean mm. Hg
Normal	5.0 to 14	9.5	−1.5 to +1.0	0.1	−4.0 to +1.5	−1.2	−6.0 to −1.5	−3.8	−2.3 to −0.5	−1.6	−4.5 to −4.0	−4.3	−5.0 to −1.6	−3.0
Constricted	8.0 to 13	13.1	−3.0 to −0.5	−1.9	−4.0 to −0.5	−2.0			−12 to −2.0	−5.6			−6.0 to −3.8	−4.8

TABLE 3. *Laparotomy Series: Changes in Inferior Vena Cava Pressure*

	Baseline		Hepatic A. Occluded 3 min.		Portal Vein Occluded 1½ min.		Portal Vein Occluded 3 min.		Hepatic A. & Portal V. Occluded 1½ min.		Hepatic A. & Portal V. Occluded 3 min.		IVC Occluded	
	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mran mm. Hg	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mean mm. Hg
Normal	2.7 to 10	5.6	−0.7 to +1.5	+0.2	0 to +1.0	+0.5	−1.0 to +4.0	+1.4	0 to −0.5	−0.3	−0.7 to +3.0	+1.1	+22 to +53	+40.
Constricted	7.0 to 18	12	−4.0 to −0.5	−1.7			−5.0 to +1.0	−2.4			−4.0 to −2.0	−3.0	0 to +11	+4.6

a mean caval pressure fall of 3.0 mm. Hg. No consistent effect upon caval pressure was produced in normal dogs.

Occlusion of the IVC above the caval catheter produced uniform increases in pressure within the inferior caval segment in almost all animals. In normal dogs the mean increment was 40.0 mm. Hg as compared with 4.6 mm. Hg in the group with caval constriction.

Thoracotomy Series

Effects Upon Systemic Pressure (*Table 4*). Occlusion of the IVC in normal dogs produced an abrupt fall in systemic pressure to shock levels. The mean fall for the normal group was 71.0 mm. Hg within 15 seconds. In vena cava constricted dogs, however, three minutes of caval occlusion produced no change in pressure in three of the six animals tested. In the remaining three vena cava constricted dogs, pressure fell an average of 26.0 mm. Hg.

Azygos vein occlusion for three minutes produced no significant change in arterial pressure in either group.

When both the IVC and azygos vein were occluded, systemic pressure decrements were slightly greater than those with caval occlusion alone. In those three dogs that had not previously developed hypotension with caval occlusion, combined caval and azygos occlusion produced a mean arterial pressure fall of 8.3 mm. Hg. In the remaining three "experimental" dogs, combined occlusion produced the same effect upon arterial pressure as did IVC occlusion alone.

Effects Upon Azygos Vein Pressure (*Table 5*). In normal dogs, mean azygos vein pressure was 6.4 mm. Hg. Mean azygos vein pressure in dogs with vena caval constriction was 6.0 mm. Hg.

When the IVC was occluded in normal dogs, there was a slight fall in mean azygos vein pressure of 1.0 mm. Hg. Mean azygos

TABLE 4. *Thoractomy Series: Changes in Systemic Pressure*

	Baseline		IVC Occluded		Azygos V. Occluded		Azygos V. & IVC Occl.	
	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Range mm. Hg	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mean mm. Hg
Normal	90 to 120	120	−115 to −35	−71	−5.0 to 0	−2.0	−110 to −60	−85
Constricted	50 to 160	103	−30 to 0	−12	−20 to 0	−6.0	−35 to −5.0	−17

TABLE 5. *Thoracotomy Series: Changes in Azygos Vein Pressure*

	Baseline		IVC Occluded		Azygos V. Occluded		Azygos V. & IVC Occl.	
	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mean mm. Hg
Normal	4.0 to 8.0	6.4	−2.0 to +0.5	−1.0	+1.0 to +5.0	+3.8	+1.0 to +4.5	+2.6
Constricted	3.0 to 8.0	6.0	0 to +4.0	+1.8	+2.0 to +12	+5.6	+3.0 to +6.5	+5.1

vein pressure rose 1.8 mm. Hg in the constricted group.

When the azygos vein was occluded, mean pressure within the occluded segment of the vein rose 3.8 mm. Hg in normal dogs and 5.6 mm. Hg in vena cava constricted dogs.

The superimposition of IVC occlusion on azygos occlusion did not significantly further alter the pressure within the occluded segment of azygos vein in either group.

Effects Upon Inferior Vena Cava Pressure (*Table 6*). Mean IVC pressure in normal dogs was 6.6 mm. Hg and in vena cava constricted dogs was 14.0 mm. Hg. Occlusion of the IVC in normal dogs produced a mean rise in pressure within the caudal segment of IVC of 14.0 mm. Hg within 15 seconds. In vena cava constricted dogs, three minutes of caval occlusion produced a mean rise of 2.0 mm. Hg within the caudal caval segment.

No significant change in caval pressure occurred in either group upon occlusion of the azygos vein. Simultaneous azygos and IVC occlusion in vena cava constricted dogs produced a mean rise in pressure of 3.7 mm. Hg within the caudal segment of the occluded IVC. This was a 1.7 mm. Hg greater increase than with occlusion of the IVC alone. In normal dogs, mean caudal segment pressure rose 14.0 mm. Hg. This did not represent a change from the effect of IVC occlusion alone.

Discussion

Ample evidence for the presence of inferior vena caval-systemic collaterals in dogs with vena caval constriction can be derived from the unique response of this group to IVC occlusion. The immediate (15-second) development of profound hypotension in normal dogs upon acute occlusion of the thoracic IVC is the result of

TABLE 6. *Thoracotomy Series: Changes in Inferior Vena Cava Pressure*

	Baseline		IVC Occluded		Azygos V. Occluded		Azygos V. & IVC Occl.	
	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mean mm. Hg	Range mm. Hg	Mean mm. Hg
Normal	5.0 to 7.5	6.6	+10 to +19	+14	−0.5 to +0.5	−0.1	+9.5 to +19	+14
Constricted	12 to 17	15	0 to +3.5	+2.0	−1.0 to +2.0	+0.7	+2.0 to +7.0	+3.7

a marked reduction in venous return to the heart. Since thoracic IVC occlusion for three minutes in vena cava constricted dogs produces at most only mild hypotension, it must be assumed that adequate venous return to the heart was maintained despite the occlusion. With suprarenal occlusion of the IVC, there is no noticeable reduction in systemic arterial pressure in the vena cava constricted group. The normal dogs, however, still manifest significant hypotension.

Further evidence for the importance of caval-systemic collaterals can be obtained from the difference in pressures between normal and experimental dogs that develop within the occluded caudal segment of IVC. Suprarenal occlusion of the IVC in the normal dog uniformly results in a marked elevation in caval pressure. In contrast to this, caval pressure is but slightly increased in the experimental dog. The absence of a significant increment in caval pressure indicates the presence of extracaval channels capable of returning venous blood to the heart, particularly since arterial pressure is unchanged. The extent of these collaterals is demonstrated by their ability to carry this large volume of blood with negligible changes in venous pressure.

In experimental dogs, it was noted that decrements in caval pressure resulted from procedures which reduced hepatic blood flow. Occlusion of the hepatic artery, the portal vein, and finally combined occlusion of these vessels produced increasing falls in caval pressure. These observations indicate a major utilization of the abdominal IVC and its collateral outflow for the transport of hepatic venous blood.

The collateral circulation that develops following IVC obstruction has been thoroughly described.[6] An excellent presentation of the subject and thorough review of the early literature is offered by Robinson.[8] The importance of the vertebral system of veins in bypassing caval obstruction is consistently emphasized. Batson,[1] interested in the mechanism of vertebral dissemination of prostatic and pelvic tumors, re-introduced a description of the anatomy of this system in 1940. Direct connections between the IVC and the vertebral system of veins exist in normal animals in the region of the iliac veins, juxtarenal IVC and the left renal vein. However, there is tremendous potential for increasing communication upon the development of caval obstruction. In the presence of caval obstruction, the expanded load of the vertebral veins is returned to the heart primarily by the azygos and accessory hemiazygos veins. The former joins the superior vena cava just cephalad to the right atrium. The accessory hemiazygos vein delivers its blood to the superior vena cava via the innominate vein. Due to its great capacity, plexiform structure, and the bony protection of the spinal column, the vertebral system of veins maintains a low pressure state even in the presence of augmented flow. This is correlated with the finding of similar azygos vein pressures in normal and experimental dogs. Upon complete thoracic IVC occlusion in vena cava constricted dogs, an increased azygos vein flow was reflected by a small but constant rise in azygos pressure. The varied response in azygos vein pressure to thoracic caval occlusion in normal dogs cannot be interpreted because of the rapid development of a hypotensive state. A comparison of the changes in pressure within the azygos vein that occurred following its occlusion in normal and vena cava constricted dogs was used as a rough index of comparative blood flow in these vessels. Insofar as this form of comparison is valid, a greater azygos blood flow was indicated in caval constricted dogs. The development of hypotension following combined IVC and azygos vein occlusion in three experimental dogs that had not manifested such change with IVC occlusion alone again indicates the importance of the azygos vein for the transport of caval

blood. Further testimony for the importance of the azygos vein in experimental dogs was offered by the effect of combined IVC and azygos occlusion upon caval pressure. Greater increments in caval pressure developed in dogs when these vessels were simultaneously occluded than with IVC occlusion alone. This indicated an additional degree of caval outflow obstruction. Although the azygos vein, and to a lesser degree, the hemiazygos vein are major terminals for the caval collaterals, adequate venous return still occurs in dogs with vena cava constrictions upon ligation of these vessels.[9] A consideration of the extensive potential for vertebral-caval communication in the head and neck readily explains this observation.

Although occlusion of the thoracic IVC results in increments in caval pressure in both groups, the changes are much less than those following suprarenal caval occlusion. Since far greater quantities of blood traverse the thoracic segment of the IVC than the abdominal segment, one would expect to find greater increments in caval pressure with occlusion at this point. The rapid development of profound hypotension in normal dogs upon thoracic caval occlusion may explain this paradox. If this were the entire explanation, however, one would expect to observe an initial abrupt rise in caval pressure followed by a fall upon the development of arterial hypotension. This was not borne out by observations of caval pressure changes immediately following thoracic caval obstruction. Further observation of this phenomenon was made in four dogs by observing caval pressures during the first week following a standard partial thoracic caval constriction. It was noted that caval pressure only increased by 1 to 1.5 mm. Hg immediately following this procedure. After 24 hours, however, IVC pressures had risen approximately 5.0 mm. Hg above preoperative levels, and at 48 hours from 10.0 to 16.0 mm. Hg. For most of these animals this represented a peak in IVC pressure.

It is apparent that the inclusion of the hepatic veins within the obstructed caval segment constitutes the major anatomic difference between thoracic and suprarenal IVC obstruction. The addition of the hepatic veins provides a considerable increase in effective caval volume to accept large quantities of blood. Under these circumstances elastic resistance of the vessels, which can be responsible for great increments in pressure, is not encountered. The slight increment in caval pressure and hence hepatic venous pressure results in increasing hepatic congestion. As the liver swells within the confines of Glisson's capsule, the hepatic veins are compressed. The capacity of the system is reduced and caval pressure rises. Caval pressure then remains elevated until the development of an adequate collateral circulation.

Since thoracic caval constriction ultimately increases caval pressure, one would expect a concomitant increase in portal pressure. In all animals observed, portal vein pressure remained equal to or greater than caval pressure. In normal dogs, there was a greater mean porto-caval pressure gradient (3.9 mm. Hg) than in constricted dogs (1.1 mm. Hg). The greater increase in caval pressure than portal pressure in constricted dogs, is evidence for the existance of an effective collateral system decompressing the portal bed. This system involves direct connections between the prehepatic portal bed and the vertebral system of veins. Such passage of blood is the natural consequence of the great pressure gradient between these two juxtaposed systems. The difference in response of arterial pressure to acute portal vein occlusion in normal and constricted dogs is striking. The rapid development of severe hypotension in normal dogs must assuredly be related to the pooling of blood in the splanchnic bed with a secondary reduction in cardiac output. Since dogs with vena

caval constriction were able to maintain arterial pressure much more successfully in the face of portal vein occlusion it is assumed that splanchnic pooling was not as severe in these animals. This pooling is minimized by the presence of capable porto-systemic collaterals in the constricted dog. Further evidence for this is derived from the finding that dye injected into the occluded portal circulation escapes more rapidly in constricted than normal dogs.[7] In addition, vena caval constricted dogs survive portal vein ligation [7] a procedure which is uniformly fatal in normal dogs.[5]

Partial constriction of the thoracic IVC impedes the return of both IVC and splanchnic blood to the heart through the constricted caval segment. The development of extensive vascular communications between the IVC and tributaries of the superior vena cava permits both hepatic outflow and caval blood to return to the heart. This cavo-caval collateral system appears to be the major vascular compensatory development in this experimental animal. The present work also indicates that pre-hepatic splanchnic blood is capable of returning to the heart in the presence of complete portal vein occlusion in the constricted dog. It has been shown that this ability is not shared with the normal dog. From studies [3, 4] which show that hepatic blood flow is not significantly reduced in the fasting constricted dog, it is suggested that these porto-systemic collateral channels carry only a small amount of blood under conditions of the experiment. Since many factors have been shown to alter visceral blood flow,[2] these collaterals may carry significant quantities of blood during periods of increased blood flow to the splanchnic bed.

Summary and Conclusions

1. Two series of experiments were performed in both control dogs and dogs in which supradiaphragmatic constriction of the inferior vena cava had been performed one to four months previously.

2. In the first series, pressures were recorded in the portal vein, hepatic artery, and inferior vena cava through a laparotomy incision. In the second series, pressures were recorded within the inferior vena cava and azygos vein through a thoracotomy incision. Femoral arterial pressure was monitored in both series.

3. Pressure relationships that developed following individual and simultaneous occlusion of these vessels were studied in an attempt to investigate the relative importance of the two major collateral systems that develop following inferior vena caval constriction.

4. Occlusion of the inferior vena cava, whether suprarenal or supradiaphragmatic, produced far less decrements in systemic arterial pressure and much less increments in caudal caval pressure in constricted than normal dogs. This is considered to indicate that extensive connection between the obstructed inferior vena cava and the vertebral azygos system of veins is the major compensatory development in this experimental animal.

5. A marked difference between normal and vena caval constricted dogs was noted in their response to acute portal vein occlusion. A much more marked hypotension developed in normal dogs with this procedure. These findings have contributed to the recognition of capable proto-systemic channels in the dog with vena caval constriction. These channels, however, are considered to carry only small amounts of blood under the conditions of the experiment.

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