Myogenic Nature of Increase in Intestinal Vascular Resistance with Venous Pressure Elevation

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These studies attempted to distinguish between the various intrinsic mechanisms which might be responsible for the increase in vascular resistance seen in the intestine with venous pressure elevation (the venous-arteriolar response). Intra-arterial infusion of procaine did not block the response, indicating that a local reflex is not involved. However, the resistance increase was greatly modified during infusion of a smooth muscle relaxant (papaverine). Cyanide and ischemia were more effective than papaverine, resistance remaining unchanged as venous pressure was elevated. The tone of the intestinal wall was not increased with venous pressure elevation. In preparations perfused with an oil-kerosene mixture, resistance decreased as venous pressure was elevated, although intravascular and extravascular fluid accumulation was quantitatively the same as in the blood-perfused control. It is concluded that the venous-arteriolar response is not dependent upon neural mechanisms, nor is it due to purely physical factors. It is suggested that elevation of venous pressure causes sufficient increase of arteriolar pressure to induce a myogenic response of the resistance vessels.

An increase in the resistance of the arterioles with elevation of venous pressure has been described previously in a number of organs, and has been ascribed variously to extrinsic nerve activity, local nerve reflexes, physical encroachment of the capacity vessels upon the resistance vessels, interstitial fluid accumulation, or a myogenic phenomenon. In an earlier investigation, it was shown that an elevation of portal venous pressure increases the vascular resistance of the intestine. Since these experiments showed that extrinsic denervation of the intestine and treatment with phenoxybenzamine (Dibenzyline) did not block the response, extrinsic nerve activity would not appear to be involved in this phenomenon. Also, accumulation of interstitial fluid was eliminated as a primary factor. Therefore, the purpose of these experiments was to determine whether an intrinsic nervous mechanism, a vascular myogenic mechanism, tonus of the intestinal wall, or engorgement of the capacity vessels are responsible for the decreased caliber of the resistance vessels of the intestinal bed with increased venous pressure.

Methods

These studies, composed of 5 series of 5 experiments each, were performed on dogs anesthetized with sodium pentobarbital (30 mg./Kg.), given intravenously and employed a combination of gravimetric and flow determinations in denervated segments of intestine. The details of the weight and flow techniques have been reported previously.

Venous pressure was elevated in 5 cm. increments from 5 to 30 cm. of water, holding the pressure constant for 5 min. at each level, with readings being taken each minute. Venous pressure was then reduced to 5 cm. in one step and readings were continued for 10 min.

Resistance was calculated as the P/F ratio, where P is the arterial-venous pressure in millimeters of mercury and F is the flow in milliliters per min. per 100 Gm. tissue. The resistance is expressed in peripheral resistance units (PRU).
The weight change recorded by this method is the sum of the increases in blood volume and interstitial fluid volume. Since the rate of weight change appeared constant after the first minute, it was assumed that the slow change represented interstitial fluid accumulation and the rapid initial change an alteration of blood volume.

It is possible to make an approximation of the capillary filtration coefficient by assuming that with a given increase of venous pressure, capillary pressure is altered by 80 per cent of that amount, a relation which has been reported in the hind limb of the dog and the cat. The filtration coefficient is calculated as the difference in rate of weight change at two venous pressure levels in Gm./100 Gm. tissue/min. divided by 80 per cent of the difference in the venous pressures in millimeters of mercury.

In the first group of 5 denervated preparations procaine was continuously infused intra-arterially in a concentration of 4 per cent, in 0.45 per cent saline to achieve isotonicity, at a rate of 0.8 ml./min., beginning 15 to 20 min. before the experiment. The pH of the solution was adjusted to 7.4 before infusion. The concentrations averaged 0.12 per cent procaine in arterial blood. Stimulation of the sympathetic nerve to the segment always caused a large reduction of flow before procaine. After 10 min. of infusion sympathetic stimulation was ineffective. In the second group of experiments, papaverine hydrochloride, an antispasmodic agent, was infused intra-arterially continuously in concentrations which averaged 25 mg./L of arterial blood. The concentration of papaverine was adjusted to achieve maximal dilatation without reduction of arterial pressure.

In a third group of experiments, sodium cyanide in saline was continuously infused intra-arterially in 3 preparations in concentrations of 0.25, 0.4, and 0.9 mM/L of arterial blood, beginning 15 min. before the experiment. Ischemia was produced in 2 other preparations by clamping the appropriate artery for 45 min. The experiment was begun after the reactive hyperemia had passed.

The change in tonus of the intestinal wall with elevation of venous pressure from 5 to 30 cm. H₂O in a single step for a period of 5 min. was studied in the fourth series of experiments. Tonus was measured by a balloon placed in the lumen of the segment and recorded by an optical manometer.

To observe the mechanical characteristics of the vascular system when vital processes were effectively absent, the intestine was perfused with a non-nutrient solution in the last series of experiments, beginning 45 min. after cessation of blood flow. Since preliminary studies with plasma showed rapid and profuse edema formation, an oil-kerosene mixture, such as previously used by Waugh in the kidney, was employed. The mixture had a specific gravity of 0.82 and a viscosity of 4.16 centipoises at 25 C. as determined by an Ostwald viscometer. After a control study the supply artery was cannulated. Flow was begun by forcing the mixture through the segment with a syringe to flush out the aqueous phase. Flow was continued by gravity feed from a reservoir at a pressure of 85 to 95 mm. Hg. The experimental procedure was similar to that in the viable preparation.

RESULTS

Effects of Procaine Infusion. To determine the possible influence of the abundant local nerve network of the intestine, the response to venous pressure elevation was studied before and after procaine treatment. Initial peripheral resistance was 5.52 PRU in the control preparations and 8.10 PRU in the procaine treated segments. The higher vascular resistance after procaine was due in part to the residual constrictor effect of the control pressure elevations and in part to an enhancement of tone of the muscular wall by procaine. Elevation of venous pressure to 30 cm. H₂O in control preparations caused an increase in peripheral vascular resistance, blood volume and interstitial fluid volume (fig. 1). With reduction of venous pressure, blood volume and interstitial fluid volume returned to normal in 10 to 20 min., but the resistances remained somewhat elevated. With procaine treatment the vascular response was not reduced; on the contrary, it appeared to be slightly increased. However, the difference was not statistically significant. In several instances the procaine solution was introduced into the lumen of the intestine for 20 min. This was also ineffective. The capillary filtration coefficient in the procaine preparations averaged 0.053 as compared to 0.090 Gm./100 Gm. tissue/mm. Hg/min. in the control preparations, which accounts for the lesser accumulation of fluid in the former group.

Inasmuch as procaine did not block the response of the resistance vessels to venous
Effects of Procaïn. The second series of experiments therefore studied the influence of a local muscle relaxant, papaverine hydrochloride (fig. 2). This agent caused a reduction of initial resistance, 3.43 cm Hg compared with 4.50 PRU in the control preparations. In contrast to the control group, there was little change in resistance as venous pressure was elevated. The difference between the 2 groups was statistically significant ($p < 0.05$ by $t$ test) at all elevated venous pressures except 15 cm. H$_2$O. Five minutes after return of venous pressure to 5 cm. H$_2$O, resistance in the papaverine treated segments was 4 per cent below the initial resistance, as compared with a value of 34 per cent above initial resistance in the control segments. Thus papaverine reduced not only the intensity, but also the duration of the response.

In 3 of 5 experiments blood volume and interstitial fluid volume were measured. The increase in interstitial fluid volume was significantly greater in the papaverine preparations than in the controls at all increased pressures. The capillary filtration coefficient appeared somewhat higher in the papaverine preparations, 0.040 as compared with 0.033 Gm./100 Gm./min. H$_2$O in the controls. However, the isogravimetric venous pressure was about 15 cm. H$_2$O lower in the papaverine treated group, indicating that capillary pressure is considerably higher in these preparations. It would be anticipated that increased interstitial volume would elevate tissue pressure and therefore partially offset pressure elevation, it is concluded that the phenomenon is apparently not due to a local nerve reflex. However, it is still possible that an active constriction of the resistance vessels is responsible.

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the increase in transmural pressure produced by venous pressure elevation. However, such elevation of tissue pressure as occurs is apparently not an important factor in these experiments, in spite of the large increase in intestinal fluid volume, unless the gradual increase in resistance beyond 10 cm. H₂O venous pressure is attributed to this. Alternatively, the residual increase in resistance may have been due to encroachment of the capacity vessels upon the resistance vessels, or to incomplete blockade of the constrictor phenomenon. In the latter case, if the constrictor mechanism were completely abolished, resistance should gradually decrease as venous pressure increases, due to passive expansion of the vessels. The succeeding experiments were devoted to testing these alternatives.

Effects of Cyanide Infusion and Ischemia. In an attempt to assure more complete blockade of the mechanism, in the third series cyanide was infused in 3 preparations and ischemia was produced in 2 others. The results (fig. 3) show a more effective blockade of the constrictor mechanism than by papaverine. All average resistance values were significantly different from control values (p < 0.05) at all but a venous pressure of 15 cm. H₂O. However, the progressive reduction in resistance expected on the basis of passive expansion of the resistance vessels was seen only to 10 cm. H₂O.

In spite of the seemingly drastic nature of the procedures employed to eliminate the constrictor response, it seemed entirely possible that the failure of the resistance vessels to dilate progressively might be due to residual activity engendered by the pressure elevation.

The intestinal vasculature is apparently not refractory to all stimuli after cyanide or ischemia. Changes in the concentration of sodium cyanide infused were observed to alter the resistance immediately, with larger doses causing dilatation. The latter effect is probably due to the action of dilator metabolites formed with the blockade of aerobic metabolism by cyanide. Likewise, it cannot be assumed that 45 min. of ischemia renders the vessels completely refractory, since some reactive hyperemia was seen.

The initial vascular resistance of the ischemic and the cyanide treated preparations covered a range of 1.12 PRU to 6.75 PRU, indicating that the basal tone of the vessels is not a primary determining factor in the response.

Influence of Tonus. Blood flow is known to be influenced by changes in the tone of the intestinal wall. It is possible that effects of papaverine, cyanide and ischemia are explainable on the basis of their influence on the intestinal musculature. Therefore, this factor was recorded during venous pressure elevation. Intestinal tone was not increased as venous pressure increased (fig. 4), although resistance did increase. However, a brief period of arterial occlusion would induce an increase in tonus. Atropine, 0.3 to 1.0 mg. given intravenously in 4 preparations, did not notably alter the vascular response to venous pressure elevation. Thus, changes of tonus are apparently not contributory to the resistance increase.

Effects of Venous Pressure Elevation During Oil-Kerosene Perfusion. To determine the effects of venous pressure on the vascular resistance of an inert preparation, the anoxic intestine was perfused with a non-nutrient oil-kerosene mixture (fig. 5). In this case the vascular resistance did progressively decrease with venous pressure elevation. At the higher venous pressures small amounts of oil were observed dripping from the lumen of the intestine. The fact that the total fluid volume changed to the same extent in the oil perfused preparation as in the control, while the resistance decreased, indicates that pressure exerted on the resistance vessels by tissue fluids and venous vessels may not be appreciable in the normal preparation. The difference between these results and those obtained with cyanide and ischemia suggests that in the latter case there is some residual constrictor ability. A less likely alternative is
that the anomalous viscosity of blood with the slightly lower flows counteracts the dilatation of resistance vessels.

The effect of the kerosene upon the elastic characteristics of the vasculature has not been determined. However, the vascular resistance tended to return to control values after reduction of venous pressure (average 10 per cent below control), indicating that the elasticity was not completely lost.

**Discussion**

In the first series, the vascular response to venous pressure elevation persisted in spite of the infusion of large amounts of procaine. Since this was sufficient to block the effect of direct stimulation of the vasoconstrictor fibers, it seems reasonable to suppose that local reflexes causing vasoconstriction would be blocked as well. This would appear to eliminate nerve influence as being an important feature of this response. For this reason we feel the term *venous-arteriolar reflex* employed by others cannot be properly applied here and have used the more general term *venous-arteriolar response*.

These results are not in harmony with those of others on this phenomenon. Rosenberg found the response abolished in 2 of 3 experiments on the hind limb of the rabbit after injection of 2 and 4 per cent procaine solutions as judged by critical closing pressure and suggested that a local reflex was involved. However, recent evidence indicates that critical closure may not be a reliable index of vascular tone. Others have found the autoregulation of the kidney to be abolished by infusion of procaine at somewhat lower concentrations in arterial blood than employed in our experiments. However, in the latter case it has been suggested that autoregulation is a myogenic phenomenon. Possibly unique regional characteristics may be responsible for some of the observed differences. The results of the present experiments are interpreted only on the basis of the local anesthetic properties of this drug.

Papaverine hydrochloride is a potent arteriolar relaxant. The ability of this drug to attenuate the venous-arteriolar response in the intestine, coupled with the ineffectiveness of nerve blockade, indicate that a myogenic response may indeed be responsible for the resistance increase. The tendency for the resistance to remain elevated in the controls well after the venous pressure is decreased also suggests that some phenomenon akin to a smooth muscle spasm, rather than a nerve reflex may be involved. The fact that tonus is not altered with venous pressure elevation indicates that the effect is not dependent upon contraction of the intestinal wall.

The effectiveness of cyanide and ischemia in reducing the response and the decrease
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in resistance with oil perfusion, rule against the resistance increase as being purely secondary to physical factors such as engorgement of the capacity vessels or interstitial fluid accumulation. However, when vessel distention and extravascular accumulation are large it is possible that these factors may influence resistance, although their influence would appear to be limited.

The foregoing experiments appear to eliminate all factors except the myogenic phenomenon as the primary mechanism of the venous-arteriolar response in the intestine. Patterson and Shepherd have previously suggested that the venous-arteriolar response may be myogenic. This was based on the observation that the response persists in the human forearm after chronic denervation. However, the sound establishment of the myogenic phenomenon as the operative factor in the venous-arteriolar response requires that 2 basic conditions be satisfied: (1) that the myogenic phenomenon be shown to exist in the organ studied and to be capable of increasing the resistance of the arterioles with elevation of arterial pressure, and (2) that elevation of venous pressure be shown to increase arteriolar distending pressure sufficiently to induce the resistance increase observed.

In a study of the pressure-flow characteristics of the intestinal arterial bed, Selkurt et al. reported that 9 preparations which were more responsive and therefore approached more nearly a normal physiologic state showed an increase in resistance with elevation of arterial pressure. Eighteen other, more inert preparations showed a dilatation of the resistance vessels with elevation of arterial pressure. The changes in the former type preparation may very well be interpreted as evidence of the existence of the myogenic phenomenon in the intestine. Folkow has reported that vascular responsiveness is reduced when an artificial perfusion system is used. Thus, the possibility of the myogenic phenomenon being present in our preparations is somewhat greater.

While no direct measurements have been made on the effect of venous pressure on the distending pressure of the arterioles, certain basic considerations are apparent. The resistance of the veins and capillaries is small as compared with that of the arterial system. Thus, most of the venous pressure elevation will be reflected through the capillaries to the arterioles. The 18 per cent resistance decrease with venous pressure elevation during oil-kerosene perfusion indicates the resistance vessels are distended appreciably. Also, Folkow and Lofving have found that venous pressure elevation causes a notable reduction in resistance in the hind limb preparation perfused with a dextran-Tyrode solution, which is interpreted as due to passive distention of the arterial, as well as the venous vessels. However, if the vasculature possessed myogenic ability, such distention should lead to a constriction of the resistance vessels. These facts support the suggestion that the intestinal resistance vessels possess the capacity for myogenic response and that venous pressure elevation may provide sufficient stimulus to induce such a response.

This study also provides information on the effect of various drugs on interstitial fluid formation in the intestine. Papaverine greatly increases it, apparently as a result of increased capillary pressure primarily, procaine appears to decrease it in most cases, apparently as a result of reduced capillary filtration coefficient.

The nature and magnitude of the change in blood volume is of interest. Alexander and associates report that the portal vascular bed will pool about 13 ml. of blood as portal pressure is raised from 5 to 30 cm. saline, due to elastic distention of the system. A secondary slow increase of greater magnitude in the venous capacity, termed "delayed compliance" which proved more difficult to quantitate, was also suggested. In our experiments the estimated increase in blood volume would average 15 to 20 ml. for a 500 Gm. small intestine. The rate of weight increase after the first minute of pressure eleva-
tion appeared relatively constant, as illustrated previously.\textsuperscript{8} Thus, slow changes in blood volume such as may have been due to delayed compliance either were completed by the end of the first minute, or were of such constancy as to be indistinguishable from interstitial fluid accumulation.

**Summary**

Previous studies have shown that the increase in vascular resistance of the intestine occurring with venous pressure elevation is not due to extrinsic innervation. The present studies demonstrate that the response is likewise not due to a local reflex, since procaine infusion did not attenuate the resistance increase. Therefore the more general term venous-arteriolar response, rather than venous-arteriolar reflex, is employed. The resistance increase is the result of an active vas constriction, since infusion of a smooth muscle relaxant (papaverine) lessened the response to increased venous pressure. Cya

nide and ischemia were more effective than papaverine, resistance remaining unchanged as venous pressure was increased. The tone of the intestinal wall was not influenced by venous pressure elevation. During perfusion of the intestine with an oil-kerosene mixture, resistance decreased with elevation of venous pressure. In the latter case, intravascular and extravascular fluid sequestration seemed about the same as in the blood-perfused intestinal segment. Since resistance decreased, such sequestration apparently does not contribute appreciably to the venous-arteriolar response.

The results indicate that the venous arteriolar response in the intestine is not dependent upon neural pathways, nor is it due to the above mentioned physical factors. The magnitude of the resistance decrease seen with venous pressure elevation during oil-kerosene perfusion indicates that this pressure increase does cause appreciable distention of the arterioles. By consideration of this fact and the elimination of other possible mechanisms, it appears that venous pressure elevation causes an increase in vascular resistance by inducing a myogenic response of the arterioles by transmission of the pressure increment through the capillaries to the arterioles.

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**Summary in Interlingua**

Previ studios ha monstrate que le augmento del resistencia vascular que occurre in le intestinos con le elevation del tension venose non es le effecto de un innervation extrinsec. Le presente studios demonstra quo le responsa es etiam non le effecto de un reflexo local, proque infusiones de procaina non attenuava le augmento del resistencia. A causa de iste observationes, le termino general "responsa veno-arteriolar" es usate in iste caso in loco de "reflexo veno-arteriolar." Le augmento del resistencia es le resultato de un vasoconstrictione active, proque le infusion de un agente de relaxation de musculo lisie (papaverina) reduceva le responsa al elevation del tension venose. Cyanuro e ischemia esseva plus efficace que papaverina; in lor presentia le resistencia remaneva inalterate quando le tension venose esseva augmentate. Le tono del pariete intestinal non esseva influentiate per elevationes del tension venose. Durante le perfusion del intestino con un mixtura de oleo e petroleo, le resistencia decreceseva con le elevation del tension venose. In iste ultimo caso, le sequestration de fluido intra- e extravascular esseva apparentemente plus o minus identic con illo observate quando le segmento intestinal esseva perfundite con sangnine. Viste que le resistencia decreceseva, le mentionate sequestration non pare contribuir appreciablemente al responsa veno-arteriolar.

Le resultatos indica que le responsa veno-arteriolar in le intestino non depende de circuitos neural e similmente que illo non depende del supra-mentionate factores physic. Le magnitude del reduction que occurre in le resistencia quando le tension venose es aug-
mentate sub le conditiones de perfusion a oleo e petroleo indica que iste augmento del tension causa un appreciable distension del arteriolas. Si nos considera iste facto, il pare-post le elimination de certe altere mechanismos possible—que le elevation del tension venose causa un augmento del resistencia vascular per inducir un response myogene del arteriolas como effecto del facto que illo transmitte le augmento de tension via le capillares a le arteriolas.

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