Physical Effects of Increased Venous and Extrarenal Pressure on Renal Vascular Resistance

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The oil perfused kidney preparation was used to determine the passive effects of increased venous and extrarenal pressure on renal vascular resistance. The dimensional tube factor of renal resistance varied inversely with the arteriovenous pressure gradient because this pressure difference also determined the pressure difference across the walls of the intrarenal vessels.

An understanding of the purely hydraulic relationships which affect the relation of pressure to flow in the complex renal vasculature is a needed background for differentiating vasomotor changes with elevated venous renal pressure and effects of extrarenal pressure. It has not been determined, for example, whether increased venous pressure passively lowers or increases renal resistance. Venous distention within the relatively inextensible renal capsule might raise the total resistance of the kidney by increasing the interstitial pressure and effectively narrowing the high resistance intrarenal vessels. The reasons why the question can not be resolved by perfusion of the kidney with blood or saline have been discussed in the preceding paper.

The physical effect of increased extrarenal pressure on renal pressure-flow relationships is debatable. A difference of opinion has arisen whether intra-abdominal pressurization, like increased venous pressure, causes venous congestion of the kidney. An appropriate technic to determine the purely passive effects of increased venous and extrarenal pressure on renal vascular flow and resistance would appear to be oil perfusion of the isolated kidney.

Methods and Materials

Single kidneys were isolated from heparinized dogs, flushed initially with saline, and perfused with a mixture of kerosene and mineral oil at room temperature as previously described and pressures and flows were measured by the same methods. When the effects of a raised venous pressure was studied, venous pressure was regulated by means of a screw-clamp occluding a tube placed distal to a venous T-cannula or by adjustment of the height of the venous outflow tube into atmospheric air. In the experiments with increased extrarenal pressure, kidneys were placed in an air-tight chamber with effectively enclosed arterial and venous manometers. Segments of excised carotid artery and external jugular vein were joined to the arterial and venous T-cannulas, respectively, in order to have collapsible vessels on both vascular ends of the cannulas. Arterial and venous conduits passed via glass tubes to the outside of the chamber. By forcing air into the chamber, increments in extrarenal pressure were produced independently of changes in arterial and venous pressure.

Results and Discussion

Effects of Increased Venous Pressure when Arterial Pressure is Raised Equally. Upon raising equally the venous and arterial pressures of the oil perfused kidney (table 1A), flow and resistance between the main renal artery and vein remained constant. As the venous and arterial pressures were raised, kidney volume and capsular tenseness visibly increased. However, there was but slight change in the pressure drop between the intrarenal veins and the main extrarenal vein.

Increased interstitial pressure was made evident by enlargement of the kidney and tenseness of its capsule. The elevated interstitial pressure followed increase in venous pressure and resulted from engorgement of the highly distensible venous system. It did not result mainly from increased arterial pressure nor was it due to extravascular accumu-
INCREASED PRESSURE AND RENAL VASCULAR RESISTANCE

TABLE 1.—Effects of Increased Venous Pressure in Oil Perfused Kidneys

<table>
<thead>
<tr>
<th></th>
<th>A. With constant arterial-venous pressure gradient</th>
<th>B. With venous perfusion of the kidney</th>
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<tbody>
<tr>
<td>Total pressure gradient for renal flow.</td>
<td>90 90 90 90</td>
<td>12 40 80 100</td>
</tr>
<tr>
<td>Extrarenal effluent pressure†</td>
<td>0 6 30 50</td>
<td>0 0 0 0</td>
</tr>
<tr>
<td>Intrarenal effluent pressure‡</td>
<td>8 13 37 56.5</td>
<td>11.5 38 78 98</td>
</tr>
<tr>
<td>Vascular outflow ‡</td>
<td>170 171 172 170</td>
<td>0.35 0.60 0.75 0.80</td>
</tr>
<tr>
<td>Renal resistance</td>
<td>0.53 0.53 0.52 0.53</td>
<td>33.3 66.7 107 125</td>
</tr>
</tbody>
</table>

*Pressures expressed in mm. Hg, flows in ml./min., resistances as (mm. Hg X min.)/ml.
†Main renal venous pressure in A., main renal arterial pressure in B.
‡Arcuate venous pressure in A., deep interlobar arterial pressure in B.
§Exclusive of small collateral flow.

lation of oil. This last is evidenced by the fact that ureteral flow of oil did not occur and the capsular tenseness was immediately reversible by lowering the venous pressure.

Since renal resistance remained constant with equally raised arterial and venous pressures, one must conclude that a generalized distension of the intrarenal vasculature was prevented by a compensating rise in extravascular pressure equal to the increment in venous pressure. Because the arterial pressure was equally raised, the net intrarenal transmural or distending pressures of the high resistance vessels were not significantly changed.

It has been reported that resistance to a constant blood flow is not changed when the venous pressure is raised in the kidney denervated in situ by Haddy’s technic, in the isolated kidney treated with procaine or cyanide or when extrarenal pressure is raised equally. The local renal mechanism responsible for this unchanged resistance to a constant blood flow even though venous pressure is elevated (arterial pressure rises equally) is probably the same physical process as that described above in the oil perfused kidney, in which the net transmural pressures are maintained constant.

Effects of Increased Venous Pressure which Lowers the Arteriovenous Pressure Gradient. Elevated venous pressures, which reduced the renal arteriovenous pressure difference, distended the kidney and were apparently transmitted to the tissue spaces. However, increased venous pressure effectively narrowed rather than distended pre-venous vessels of the kidney since renal resistance increased at elevated venous pressures.

As can be seen from figure 1, renal vascular resistance bore, typically, the same inverse relation to arteriovenous pressure difference regardless of whether the difference was brought about by lowering the arterial pressure or elevating the venous pressure. An effective or net distension of the vascular bed was not produced by having a high mean intravascular pressure (e.g., AP 110, VP 90) rather than a low mean intravascular pressure (e.g., AP 30, VP 10).

Arteriovenous pressure differences produce flow. In the kidney, they also apparently represent the mean transmural pressure which physically distends the intrarenal vessels and passively determines the vascular resistance to flow. This relationship where vascular resistance is, paradoxically, not a simple function of the height of the intraluminal pressures seems peculiar to the renal vasculature. It may depend upon the fact that the subcapsular pressure is raised because the renal capsule is less distensible than the voluminous renal venous system.

As shown in the previous paper, a stricture of the renal artery or vein which produces local turbulence causes increased resistance as the flow is augmented by raising the arterial pressure. In the turbulent flow preparation, raising the venous pressure lowered rather than increased resistance. This was because a lowered rate of flow due to less pressure gradient lessened the amount of turbu-
In the oil perfused canine kidney, a pressure drop of 9-24 mm. Hg is normally found between the arcuate and extrarenal veins. Intrarenal pressure which produces effluent collapse as defined by Winton does not appear to be the cause of this sizable venous pressure gradient because, at arterial pressures of about 100 mm. Hg, a slight elevation in venous pressure always reduced renal flow of oil. Furthermore, arcuate venous pressure rose at once with little change in venous resistance upon raising the main venous pressure several millimeters of mercury. These findings contrast with the demonstration in models that, upon raising the pressure downstream from a vascular segment collapsed due to a high extramural pressure, flow is not perceptibly reduced and vascular pressure just upstream from the collapse is not elevated until the raised effluent pressure exceeds the high extramural pressure. Nevertheless, the effluent veins within the renal capsule were probably narrowed by extravascular pressure at normal arterial and venous pressures. The effluent veins were perhaps in the stage of venous depletion in which the venous transmural pressure at the collapsing site had not yet reached zero. It does not appear necessary to invoke structural effluent stenosis in order to account for the renal venous pressure gradient.

Intrarenal Pressure During Retrograde Perfusion of the Kidney. The extreme resistance of the kidney to retrograde perfusion with aqueous media has been attributed, since the time of Ludwig, to mutual compression or twisting of glomerular capillaries. With oil perfusion directed into the renal vein, the kidney is also much larger in volume than with arterial perfusion, the renal capsule is tense, the flow rate very small, and the resistance very high.

Intrarenal arterial pressures were measured by insertion of a small catheter deeply into interlobar arteries of 2 kidneys subjected to venous perfusion with oil. Arterial wedging of the catheter tip was avoided. In these 2 kidneys with venous-arterial pressure gradients from 12 to 100 mm. Hg, the large artery pressure drop and resistance accounted for 86 to 95 and 91 to 98 per cent, respectively, of the total renal pressure gradient and resistance (table 1B). Upon graded withdrawal of the catheter, an abrupt pressure fall nearly to atmospheric pressure occurred in the large arteries in the region of the renal sinus. It was also found that raising the effluent or external arterial pressure to levels less than the intrarenal arterial pressure during venous perfusion did not elevate the arterial pressure within the renal capsule. Therefore, retrograde perfusion of the kidney is limited by a very high intrarenal pressure which collapses the arteries downstream where the intraluminal pressure is the lowest of all the intrarenal vessels. The high intrarenal pressure with venous perfusion of oil seems to be the result of venous and, perhaps, peri-tubular capillary distension.

Effects of Elevated Extrarenal Pressure. When the renal arterial or venous pressure was measured by a manometer within the pressure chamber which enclosed the kidney and its vessels, the manometer registered...
increased transmural pressure. The data of a typical experiment are shown in table 2.

As the extrarenal pressure was increased, the intraluminal pressure in the renal vein remained constant until the ambient pressure exceeded the intraluminal venous pressure value downstream. At this time, the effluent vein narrowed or partially collapsed at its most downstream segment exposed to the high extrarenal pressure. This venous segment often closed and opened in a flutter-like fashion. A local acceleration of flow was produced. The frictional and inertial resistance to flow in this venous segment rose and progressively reduced flow from the chamber as the extrarenal pressure was increased further. This downstream venous resistance kept the renal veins in a moderately distended state by maintaining a back pressure, in the renal veins, which was slightly higher than the extrarenal pressure.

The transmural renal venous pressure would, if elevated, produce renal congestion. However, elevated extrarenal pressure had no tendency to increase the transmural venous pressure which fell to a low, essentially constant value (table 2). Observation of the pressurized oil perfused kidney demonstrated that, as the ambient pressure rose, the organ became smaller and its capsule more flaccid in contrast to the congestion, enlargement, and capsular tenseness which followed a rise in transmural venous pressure. Therefore, it is not justifiable to ascribe functional changes in the kidney which may be associated with elevated extrarenal pressure (e.g., with compression of the abdomen) to the sequence of simply increased venous pressure as implied by Smith and Bradley. Thus, it has been believed that extrarenal pressurization increases the transmural venous pressures and causes venous congestion of the kidney.

In the experiment illustrated in table 2, a sizable pressure drop occurred between the constant pressure reservoir and the renal artery due to the interposition of a filter. The intraluminal renal arterial pressure rose towards that in the perfusion reservoir when flow was reduced by extrarenal pressure. Nevertheless, the transmural renal arterial pressure diminished as the ambient pressure was increased.

The intravascular difference in pressure between renal artery and vein was the force which, working against resistance, determined the flow. This difference was the same whether we regarded intraluminal or transmural pressures. But, since in these experiments the distending effects of the intraluminal pressures were counterbalanced by the amount of the ambient pressure, it seems logical to regard transmural pressure as the important agent in the renal pressure-resistance relationship. The transmural pressure sets the degree of swelling and congestion on the venous side and the magnitude of the renal resistance on the arterial side.

The effect of increasing intra-abdominal pressure, if we are to argue from these experiments, is to decrease transmural arterial
pressure and to leave the transmural venous pressure at a low normal level. A narrowing is formed at the exit of the vena cava at the diaphragm. This venous narrowing causes a resistance to flow which maintains an essentially normal venous distension in spite of the high extravascular pressure. The changes in renal resistance stem from the reduction of the effective (transmural) arterial pressure. The changes in renal resistance, flow, and size with elevated extrarenal pressure are exactly those seen when the arterial pressure is lowered in the simply perfused kidney.

If we may venture speculation as to the effects of increased intra-abdominal pressure upon urinary flow, it would seem that glomerular filtration and tubular function would be affected simply by the reduction in transmural (effective) arterial pressure; and (2) that the increased intra-abdominal pressure, acting upon the incompressible urine in tubule, pelvis, ureter, and bladder, would leave the mechanisms which normally move the urine along the urinary tract unaffected.

**SUMMARY**

The purely physical effects of elevated venous and extrarenal pressures on renal vascular resistance were studied in the oil perfused kidney preparation. This perfusate was chosen because it was a perfectly viscous liquid which remained intravascular and eliminated changes in vasomotor activity.

Renal resistance was found to be a function of the arteriovenous pressure difference rather than a function of the height of the mean intravascular pressure above atmospheric pressure. Increased venous pressure which lowered the pressure gradient for flow raised the total vascular resistance of the kidney by increasing the interstitial pressure through venous distension. When venous and arterial pressures were raised to the same extent, renal resistance remained constant. This constancy was due to an equal rise in a generalized subcapsular or interstitial pressure.

At normal arterial and venous pressures, the effluent intrarenal veins were narrowed but they were not in a collapsed state. With venous perfusion of the kidney, flows were restricted by a very high intrarenal pressure which collapsed the large arteries.

Increasing the pressure surrounding the kidney above that in the renal vein caused resistance changes within the kidney which were the same as the changes produced by lowering the arterial pressure to an amount equaling the difference between arterial and ambient pressure. During extrarenal pressurization, the pressure which distended the renal vein and its intrarenal tributaries was maintained at a low normal level.

**SOMMARIO IN INTERLINGUA**

Le effectos purmente physic que elevate pressiones venose e extrarenal exercite super le resistentia reno-vascular esseva studiate in preparatos de renes perfundite con oleo. Iste typo de perfusion esseva selegite proque le liquido in question es perfectemente viscoso, remane in sito intravascular, e elimina alteraciones de activitate vasomotori.

Esseva trovate que le resistentia renal esseva un function del differentia arterio-venose de pression plus tosto que del elevacion medie del pression intravascular supra le pression atmospheric. Augmentos del pression venose, que reduceva le gradiente de pression pro le fluxo, augmentava le total resistentia vascular del ren per augmentar le pression interstitial in consequentia de distension venose. Quando le pressiones venose e arterial esseva augmente per le mesme valor, le resistentia renal remaneva constante. Iste constantia esseva le effetto de un equal augmento in le generalisate pression subcapsular o interstitial.

A normal pressiones arterial e venose, le effluente venas intrarenal esseva restringite, sed illos non se trovava in un stato de collaps. Post perfusion venose del renes, le fluxo esseva restringite per un altissime pression intrarenal que resultava in le collaps del grande arterias.

Augmentos del pression circa le ren a nivellos supra le pression del vena renal effectuava alteraciones de resistentia intra le ren que esseva identic con le alterationes producire per
reduce the pressure arterial a un valor equal al differentia inter le pression arterial e le pression ambiente. Durante le application de pression extrarenal, le pression que distendeva le vena renal e su tributarios intrarenal se manteneva a un basse nivello normal.

REFERENCES

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