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MECHANISM OF AUTOREGULATION IN THE INTACT KIDNEY

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FOREWORD

The kidney performs a crucial role in maintaining a stable environment for the various tissues and organ systems in the body. A great variety of extrinsic (extrarenal) factors are involved in the interplay between the kidney and its environment. Among these, neurohumoral influences, are of chief importance in various stress states. Some of these have been evaluated in a previous CARI report from this laboratory. Man in flight may encounter environmental conditions which markedly affect renal hemodynamics. Normally, extrarenal controlling influences, such as the carotid sinus apparatus, are involved in regulating the vascular resistances of important beds such as the kidney. The kidney is unique, however, in its peculiar intrinsic ability to control its own blood flow rate. This poorly understood characteristic, termed "autoregulation," has been under intensive study for over a half century. The present investigation is of crucial importance in that it is concerned with the mechanism of autoregulation in the intact kidney, and relates the roles of extrarenal humoral influences as modifying factors of the basic autoregulatory apparatus. These experiments serve to reveal the important links between intrinsic - extrinsic controlling mechanisms in renal hemodynamics operative in man during conditions of stress.

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ABSTRACT

The mechanism of renal autoregulation is unclear. The subject of the present investigation is the mechanism of autoregulation of blood flow in the intact kidney. The left kidney of the anesthetized dog was exposed via a flank approach. Renal venous outflow was measured during control periods and following separate elevations of renal artery pressure, renal vein pressure, and ureteral pressure. Extravascular pressure (tissue pressure) was estimated by obtaining values of (a) deep vein pressure; (b) needle pressure; (c) and (d) large vein pressure and ureteral pressure elevations resulting in significant decreases in renal blood flow. Results are in agreement with previously reported studies regarding the important role of extravascular pressure in autoregulation. Increases in renal artery pressure result in elevations of extravascular pressure which compress the renal vasculature. Increases in renal vein and ureteral pressures do not decrease renal blood flow until approximating or exceeding resting tissue pressure. Results from carotid artery occlusion experiments indicate that extrarenal humoral influences may be superimposed on the autoregulatory phenomenon.

Recent studies have accounted for autoregulation of renal blood flow on the basis of changes in extravascular pressure (1, 8-15, 21-22). Experiments carried out in this laboratory (8-15) have been concerned with autoregulation of flow in isolated perfused kidneys. The question has arisen as to the applicability of these findings to the intact kidney. The present investigation is therefore a logical extension of the earlier studies to explore the possible intrinsic renal mechanism(s) operating within the intact dog kidney which would account for blood flow regulation. Results from the present work are in agreement with previously reported isolated perfused kidney studies (8-15) regarding the important role of extravascular pressure in the autoregulatory phenomenon.

METHODS

Adult dogs were intravenously anesthetized with sodium pentobarbital, 30 mg./kg. The left kidney was exposed via a flank incision, care taken to avoid penetration of the peritoneal layer. The kidney was carefully freed from the peritoneal lining, the artery, vein, and ureter were cleared of all extraneous tissues. No ties were placed around the kidney in the region of the hilus. Several blood vessels were ordinarily ligated in the anterior polar region of the capsule. The kidney was denervated surgically by section of nerve fibers, and chemically, by placing a gauze saturated with procaine around the renal pedicle. Following heparinization of the dog (3 mg./kg. body weight), the renal artery was temporarily ligated to permit cannulation of the renal vein with a large bore polyethylene tubing. Renal venous outflow was collected in a plastic reservoir secured in a water bath and blood returned from the kidney to the femoral vein of the dog by means of a sigmamotor

pump. Renal vein outflow was measured with cylinder and stop watch. Large renal vein pressure, tissue pressure (needle pressure) and aortic pressure (orifice renal artery pressure) were measured via pressure transducers and recorded on a Sanborn recorder, as previously described (8-12). Twenty-three intact kidney preparations were carried out as described above. Renal blood flows averaged 4.0 cc/min./gm. (range, 2.3-5.5) at an average renal artery pressure of 122 mm. Hg (range, 90-155). Several dog-pump-perfused isolated kidney studies were also carried out to determine the effect of bilateral carotid occlusion on levels of blood borne vasoactive agents. Deep vein pressures in some intact kidneys were obtained as illustrated in Figure 1. The tip of a flexible polyethylene catheter was carefully maneuvered deeply into the kidney substance at the level of the arcuate or interlobular veins. Criteria for successful catheter tip placement were (a) a marked increase in pressure when the catheter tip was advanced into veins deep within the renal substance (b) a rapid return in pressure following catheter flushing, and (c) free withdrawal of blood through the catheter. Technical errors inherent in the procedure have been described in two reports. (13, 16). Large vein pressure was measured and maintained at zero mm. Hg. In some experiments large vein pressure was increased in steps by adjustment of a screw clamp placed on the venous outflow catheter distal to the pressure measuring needle. Tissue pressure was estimated by four independent procedures: (a) value of deep vein pressure, (b) value of needle pressure, (c) value of increased large vein pressure resulting in a detectable fall in renal blood flow, and (d) value of increased ureteral pressure bringing about a detectable decrease in renal blood flow. As in previous studies (11, 13-16), these values showed close agreement.

Systemic arterial pressure in some experiments was increased by transfusion or decreased by withdrawal of blood following transfusion. Results were compared to those in which arterial pressure was increased by bilateral carotid artery occlusion as carried out by Thurau and Wober (26). Experiments on the dog-pump perfused kidney were also carried out to assay levels of circulating constrictor agents following carotid artery occlusion.

RESULTS

Experiments were carried out in intact dog kidneys to obtain estimations of extravascular pressure (tissue pressure). *Table I* provides average values of systemic arterial pressure (orifice renal artery pressure) and extravascular pressure in twenty-one intact kidney preparations. Extravascular pressure values were obtained either by deep vein pressure measurement (4 kidneys)or by values obtained from two to four of the independent procedures described in the Methods section. Pressures in twenty-one kidneys averaged 37 mm. Hg (range, 18-50) at an average orifice renal artery pressure of 122 mm. Hg (range, 90-145). Renal blood flows averaged 4.0 cc./min./gm. (range, 2.3-5.5).

Deep vein pressures, obtained as described in *Figure 1*, were measured early in nine intact kidneys. This data with simultaneously measured renal artery pressures is shown in *Table II*. Deep venous pressure averaged 42 mm. Hg in nine kidneys with an average renal artery pressure of 129 mm. Hg (mean renal blood flow, 4.2 cc./min./gm.).

As an extension to the above experiments, systematic arterial pressures were increased in some experiments by transfusion of blood, or decreased following transfusion. The effect of the resulting change in renal artery pressure on extravascular pressure was determined. *Figure 2* provides raw and calculated data from one intact kidney experiment. It is seen that the change in total renal vascular resistance (RAP/F) was accounted for in large measure by alterations in venous segment resistance (DVP-LVP/F) (deep vein pressure minus large vein pressure/blood flow). *Table III* summarizes results from 7 intact kidney experiments. Arterial pressure is increased from an average of 101 to 155 mm. Hg and autoregulation is observed in each instance. Approximately two-thirds of the increase in total renal resistance (RAP/F) was accounted for by alterations in venous segment resistance (DVP-LVP/F). These data emphasize the role of extravascular pressure changes as an important component of the autoregulatory apparatus.

The effect of large vein pressure elevation on renal hemodynamics is summarized in 17 intact kidney experiments (*Table IV*). Large vein pressure was successfully increased by adjustment of screw clamp on the venous outflow catheter. On the average, renal blood flow is little influenced by increases in renal vein pressure (orifice vein pressure) through a wide range of venous pressure values (0-45 mm. Hg). Total resistance (RAP-LVP/F) progressively decreases as a function of increased venous pressure varying from 0 to 60 mm. Hg. Renal artery pressures are maintained relatively constant in each experiment through the entire range of venous pressure alteration. Deep venous pressures were measured in four experiments in which large vein pressures were increased as in the previous table. *Table V* demonstrates the effect of increased large vein pressure on total resistance (RAP-LVP/F) and venous segment resistance (DVP-LVP/F) in the intact kidney. Renal artery pressure is maintained relatively constant in each experiment. It is seen that renal blood flow remains relatively constant until large vein pressure approximates or exceeds resting deep vein pressure (extravascular pressure). Total resistance (R_T) decreases as a function of elevated large vein pressure (LVP), which is primarily due to the drop in venous segment resistance (R_V).

The effect of ureteral pressure elevation on renal blood flow in intact kidneys, with and without a diuresis is illustrated in *Table VI*. Control (pre-ureteral pressure elevation) extravascular pressures are shown in most experiments. Ureteral pressure was increased by occlusion of the ureteral catheter following catheter attachment to a pressure transducer. Results show that a decrease in renal blood flow ordinarily occurs when ureteral pressure approximates or exceeds resting extravascular pressure.

Arterial pressures in the present experiment were increased by transfusion of homologous blood. Other investigators (26) have achieved this by carotid artery occlusion. Several experiments were carried out utilizing the latter procedure to determine if extrarenal influences were present to interfere with renal autoregulation. *Table VII* illustrates the results from these studies. An isolated kidney from a donor dog was perfused at constant arterial pressure or flow with blood from an intact dog. The common carotid arteries of the dog were temporarily occluded and the vascular effects on the isolated kidney were measured. Changes in resistance in the perfused kidney could only be due to changing concentrations of vasoactive agents in the blood. Results from eight experiments show that when bilateral carotid occlusion occurs, constrictor agents are released into the blood which cause a marked rise in renal resistance. Conversely, when carotid occlusion is released, a marked drop in total resistance in the isolated perfused kidney occurs. Because of the magnitude of the resistance changes when compared to the corresponding systemic artery pressure alterations, autoregulation of a kidney may be grossly interfered with by the extrinsic humoral influences. These experiments were also carried out in intact kidneys, and similar complicating extrarenal influences were also noted. It was shown that intra-arterial injections of catechol amines in the intact kidney result in marked decreases in deep venous pressure (extravascular pressure). Therefore, the effect of carotid occlusion on the intact kidney is twofold: (a) renal artery vasoconstriction and (b) a tendency to reduce extravascular pressure. These carotid occlusion experiments demonstrate the important role of extra-renal humoral influences on renal vascular resistance.

DISCUSSION

Results from the present work on the intact denervated kidney are in agreement with previously reported isolated perfused kidney studies (8-15) regarding the important role of extravascular pressure in the autoregulatory phenomenon. Findings from four independent procedures in determining extravascular pressures show essential agreement. These procedures are: values of (a) deep vein pressure (arcuate vein or deeper); (b) needle pressure; (c) large vein pressure increase which results in a significant fall in renal blood flow and (d) ureteral pressure increase producing a drop in renal blood flow. These independent measurements appear to adequately substantiate needle pressure measurements previously reported in this laboratory (8-15), and by others

(25, 27, 28). It appears that if bleeding is produced by the tissue pressure needle, it is venous in origin. Since deep venous transmural pressures approximate zero mm. Hg (11, 13, 14), deep venous pressures are reflections of tissue pressure. A penetrating needle may be apt to puncture veins because of the large volume occupied by the venous segment (3, 19). Extravascular pressures obtained in the present study are of similar magnitude, although slightly higher, than those previously reported in isolated perfused kidneys (8-15).

Autoregulation was observed in the present study when both renal arterial and renal venous pressures were altered. "Renal arterial" autoregulation was found to be produced in large part by changes in extravascular pressure, whereas "renal venous" autoregulation was brought about by failure to alter resting extravascular pressures until approximated in magnitude by large vein pressure.

It has been shown (26) that capillary and tubular pressures are uninfluenced by changes in renal artery pressure. However, these reported results (26) may be accounted for by release of catechol amine-like agents following carotid artery occlusion, as demonstrated in the present study. Release of constrictor agents into the blood as a result of carotid occlusion would produce an exaggerated degree of autoregulation by afferent arterial constriction, and would prevent a rise in extravascular pressure. Compression of the aorta above the renal artery would act in reverse fashion to offset normal changes in extravascular pressure.

Values of directly measured deep venous pressures in the present study are corroborated by the other previously described procedures (8-16). Additional information validating this measurement is as follows: (a) obstruction of the venous bed by the catheter is unlikely because of the massive degree of collateralization (2, 3, 19); (b) the size of the catheter does not influence the magnitude of the pressure (16), and (c) the greater the degree of autoregulation when either arterial or venous pressures are increased, the higher is the recorded deep venous pressure value (12-15).

Obstruction of lymphatic vessels did not account for the high values of extravascular pressure in the present study. No lymphatic vessels were ligated and results in the intact study were similar to isolated perfused preparations (8-15). Lymphatic outflow, reportedly lesser in magnitude when arterial pressure is increased than during venous pressure elevation (5, 6) is entirely consistent with the present and past (8-15) investigations: increasing arterial pressure results in increased extravascular pressure which would compress lymphatic vessels. Lymphatic flow therefore autoregulates (6, 7). The magnitudes of tissue pressure and lymph flow rate are inversely related (20). On the other hand, increases in large vein pressure would increase peritubular transmural pressure but extravascular pressure tends to remain constant through wide ranges of venous pressure alteration (14). Lymphatic outflow would therefore increase. Caution should be exercised in the interpretation of the relationship of lymphatic pressures and flows and autoregulation, for example, lymphatic pressure should not be taken as a measure of tissue pressure (7) unless the tip of the measuring catheter is in the renal parenchyma (13). This relationship also exists between large vein and deep (arcuate, interlobular) venous pressures (13, 16). It is difficult to interpret the significance of changes in lymphatic pressures and flows for the following additional reasons: lymph vessels of capsule and parenchyma anastomose (20); acute lymphatic congestion has no effect on GFR and RBF (20); injections of epinephrine result in a decrease in urine flow but an increase in lymph flow (23). The latter observation could be explained by decreases in glomerular and tissue pressures.

There has been much recent interest in the effect of increased ureteral pressure on renal hemodynamics (13, 17, 24, 27). Results from the present study are in agreement with those reported by Kiil and Aukland (17), Winton (27), and Waugh (W. H. Waugh, personal communication). Renal blood flow falls when ureteral pressure rises to values approximating or exceeding resting (control) extravascular pressure. In several experiments, renal blood flow significantly increased following ureteral occlusion. This has also been reported by others (17, 24), and from the data of the present

study, initially occurs when ureteral pressure is well below the level of control extravascular pressure. A myogenic reflex resulting from a decrease in pre-glomerular transmural pressure (due to increased extravascular pressure) therefore appears improbable. Other possibilities have been pointed out by Selkurt (24).

Figure 3 is a schema of the proposed mechanisms operating to produce renal autoregulation. The several suggested components of resistance are depicted in a qualitative fashion. Generalized extravascular pressure (tissue pressure) is seen to account for approximately 2/3 of the total resistance change. Other components, each accounting for fractions of the total resistance are, Bowman's capsule extravascular pressure (10); changes in post-glomerular viscosity (10, 12); the myogenic response (4, 15, 16, 26) and metabolic factors (15). It is probable that the contributions of each of the proposed components of resistance may vary in magnitude in kidneys as is suggested by the results of the present and past (8-15) investigations.

REFERENCES

1. Bounous, G., M. Onnis, and H. B. Shumacker, Jr. The abolition of renal autoregulation by renal decapsulation. *Surg. Gynec. & Obstet.* 111: 682-690, 1960.
2. Brun, C., C. Crone, H. G. Davidsen, J. Fabricius, A. T. Hansen, N. A. Lassen, and O. Munck. Renal interstitial pressure in normal and in anuric man: Based on wedged renal vein pressure. *Proc. Soc. Exp. Biol. Med.* 91: 199-202, 1956.
3. Chatterjee, S. K., and A. K. Dutta. Anatomy of the intrarenal distribution of renal arteries of the human kidney. *J. Ind. Med. Assoc.* 40: 155-162, 1963.
4. Folkow, B. Transmural pressure and vascular tone—some aspects of an old controversy. *Arch. int. Pharmacodyn.* 139: 455-459, 1962.
5. Haddy, F. J., J. Scott, M. Fleishman, and D. Emanuel. Effect of change in renal venous pressure upon renal vascular resistance, urine and lymph flow rates. *Am. J. Physiol.* 195: 97-110, 1958.
6. Haddy, F. J., J. Scott, M. Fleishman, and D. Emanuel. Effect of change in flow rate upon renal vascular resistance. *Am. J. Physiol.* 195: 111-119, 1958.
7. Haddy, F. J., and J. B. Scott. The relation between renal lymph pressure and autoregulation of renal blood flow. *Physiologist* 6: 194, 1963.
8. Hinshaw, L. B., S. B. Day, and C. H. Carlson. Tissue pressure as a causal factor in the autoregulation of blood flow in the isolated perfused kidney. *Am. J. Physiol.* 197: 309-312, 1959.
9. Hinshaw, L. B., H. M. Ballin, S. B. Day, and C. H. Carlson. Tissue pressure and autoregulation in the dextran-perfused kidney. *Am. J. Physiol.* 197: 853-855, 1959.
10. Hinshaw, L. B., and C. H. Carlson. Mechanisms of autoregulation in isolated perfused kidney. *Proc. Soc. Exptl. Biol. Med.* 103: 373-375, 1960.
11. Hinshaw, L. B., R. D. Flaig, R. L. Logemann and C. H. Carlson. Intrarenal venous and tissue pressure and autoregulation of blood flow in the perfused kidney. *Am. J. Physiol.* 198: 891-894, 1960.
12. Hinshaw, L. B., R. D. Flaig, C. H. Carlson, and N. K. Thuong. Pre- and postglomerular resistance changes in the isolated perfused kidney. *Am. J. Physiol.* 199: 923-926, 1960.
13. Hinshaw, L. B., and D. M. Worthen. Role of intrarenal venous pressure in the regulation of renal vascular resistance. *Circulation Res.* 9: 1156-1163, 1961.
14. Hinshaw, L. B., C. M. Brake, P. F. Iampietro, and T. E. Emerson, Jr. Effect of increased venous pressure on renal hemodynamics. *Am. J. Physiol.* 204: 119-123, 1963.
15. Hinshaw, L. B., B. B. Page, C. M. Brake, and T. E. Emerson, Jr. Mechanisms of intrarenal hemodynamic changes following acute arterial occlusion. *Am. J. Physiol.* (in press)
16. Johnson, P. C., W. H. Waugh, and L. B. Hinshaw. Autoregulation of blood flow. *Science* 140: 203-205, 1963.
17. Kiil, F., and K. Aukland. Renal concentration mechanism and hemodynamics at increased ureteral pressure during osmotic and saline diuresis. *Scand. J. Clin. Lab. Invest.* 13: 276-287, 1961.
18. Montgomery, A. V., E. R. Mickle, H. G. Swann, and J. J. Coleman. A measure of intrarenal pressure. *Texas Reports Biol. Med.* 8: 262-270, 1950.
19. Morison, D. M. A study of the renal circulation with special reference to its finer distribution. *Am. J. Anat.* 37: 53-93, 1926.
20. Rusznyak, Istvan, Mihaly Foldi, and S. Gyorgy. Lymphatics and Lymph Circulation. New York; *Pergamon Press*, 1960; (pgs. 116, 688-703).
21. Samelius, U., and A. Astrom. On pressure flow relationships in the cat kidney. *Acta Physiol. Scand.* 50 (Suppl. 175): 139, 1960.
22. Scher, A. M. Mechanism of autoregulation of renal blood flow. *Nature* 184: 1322-1323, 1959.
23. Schmidt, C. F., and J. M. Hayman, Jr. A note upon lymph formation in the dog's kidney and the effect of certain diuretics upon it. *Am. J. Physiol.* 91: 157-160, 1929.
24. Selkurt, E. E. Effect of ureteral blockade on renal blood flow and urinary concentrating ability. *Am. J. Physiol.* 205: 286-292, 1963.
25. Swann, H. G., V. Moore, and A. V. Montgomery. Influence of arterial pressure on intrarenal pressure. *Am. J. Physiol.* 168: 637-641, 1952.
26. Thurau, K., and E. Wober. Zur Lokalisation der autoregulativen Widerstandsanderungen in der Niere. *Pflueger Arch. ges. Physiol.* 274: 553-566, 1962.
27. Winton, F. R. Hydrostatic pressures affecting the flow of urine and blood in the kidney. *The Harvey Lectures, Series XLVII*, New York, Acad. Press, 1951-52, pp. 21-52.
28. Winton, F. R. Intrarenal pressure and renal blood flow. *Transactions of the Third Conference, Renal Function*; New York, Josiah Macy, Jr. Foundation, 1951, pp. 51-102.

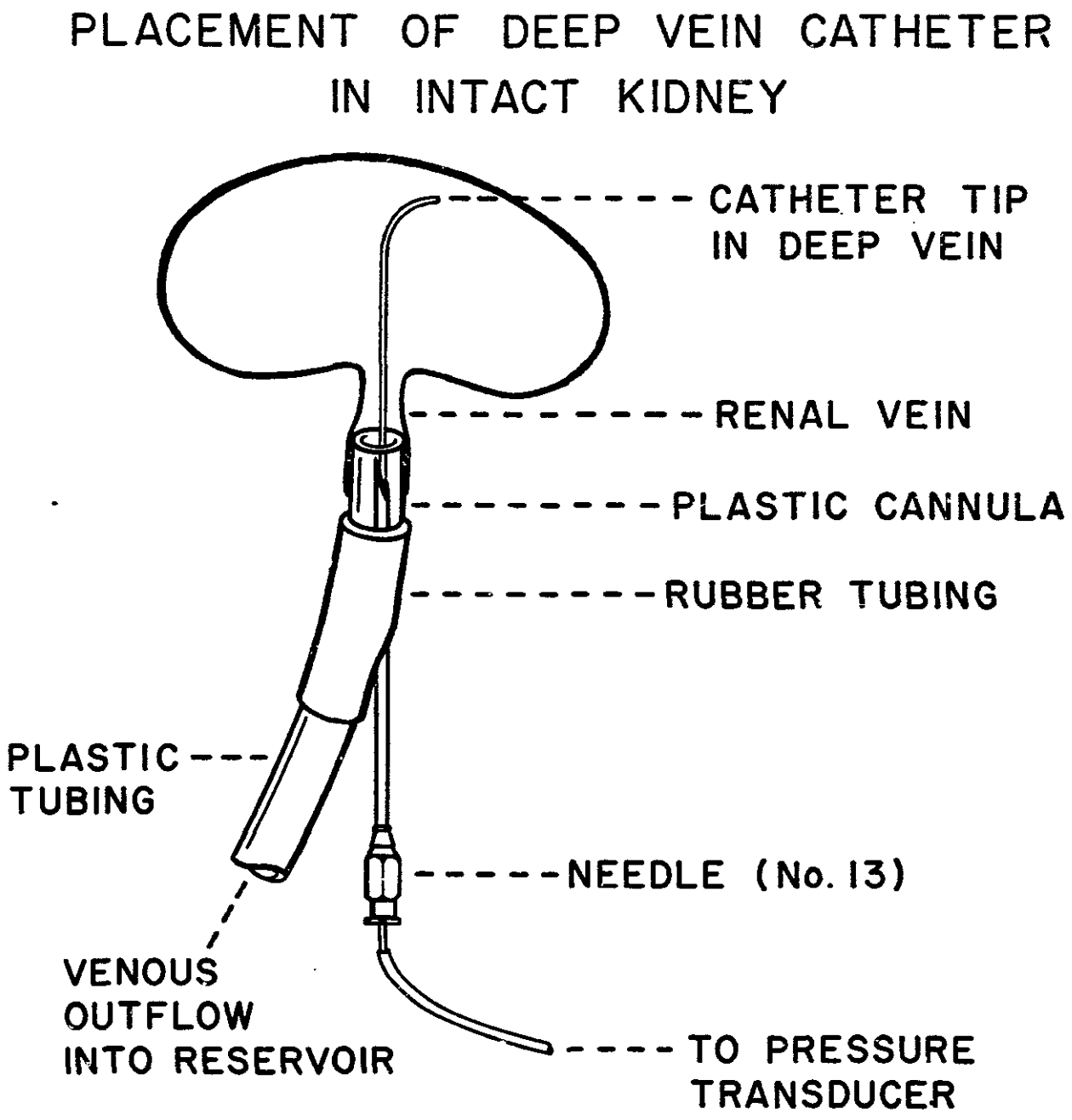

FIGURE 1. Placement of deep vein catheter in intact kidney.

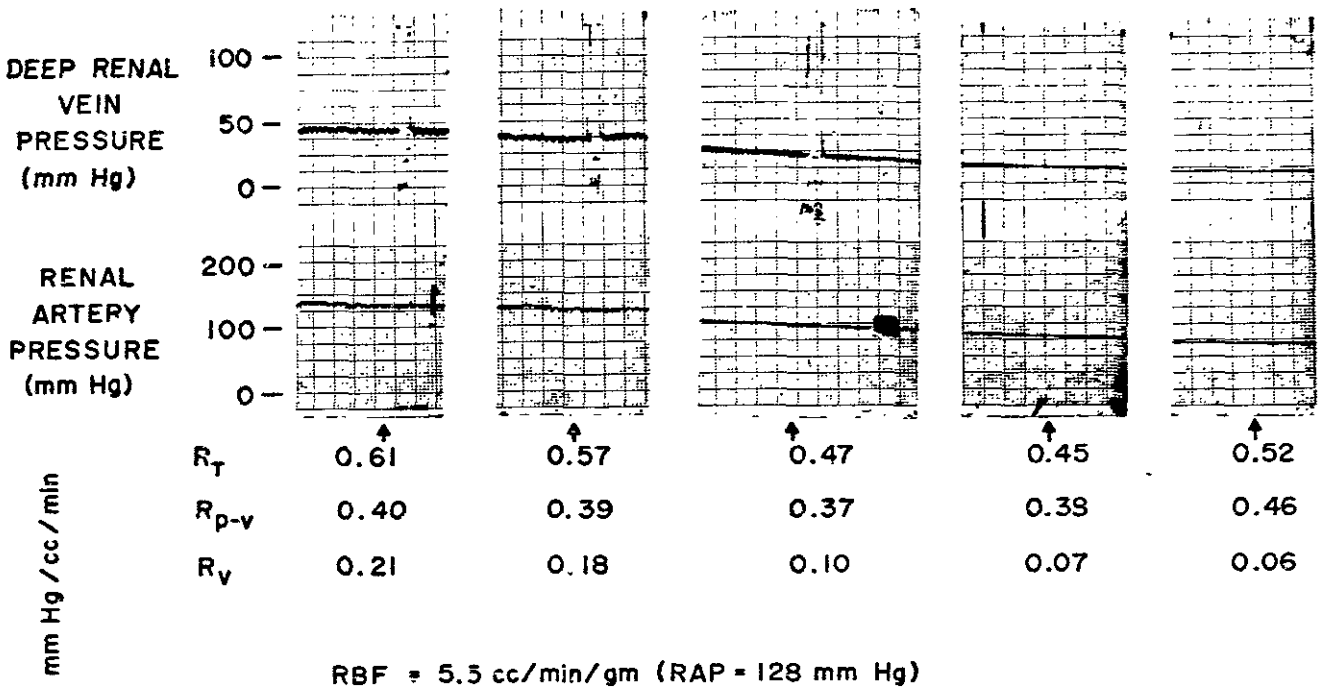

FIGURE 2. Relationships of deep renal vein pressure, renal artery pressure, and renal segmental resistance.
(one expt.)
R_T=TOTAL RESISTANCE (RAP/F) (LVP=ZERO mm Hg)
R_{p-v}=PRE-VENOUS SEGMENT RESISTANCE (RAP-DVP/F)
R_v=VENOUS SEGMENT RESISTANCE (DVP–LVP/F)

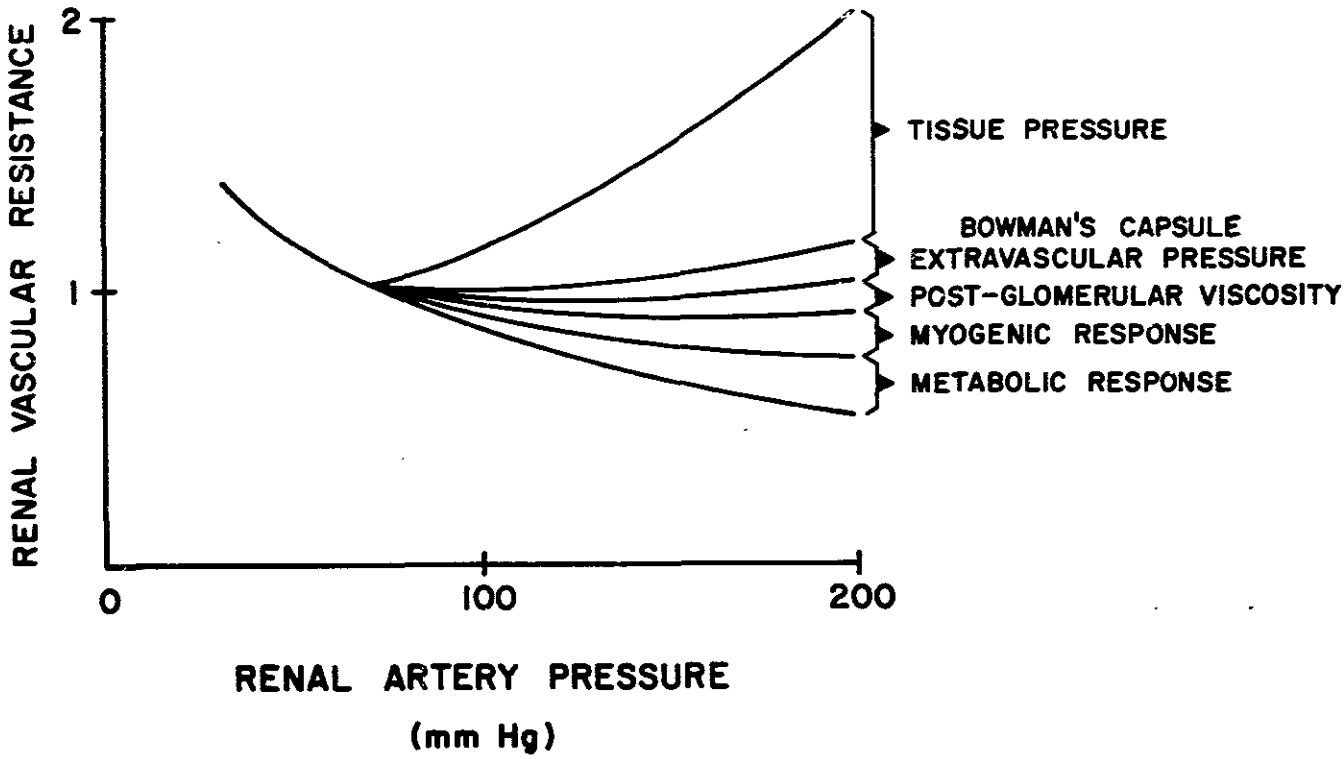

FIGURE 3. Mechanism of autoregulation: suggested components of resistance. (Qualitative relationships.)

TABLE I

EXTRAVASCULAR PRESSURE AND ARTERIAL PRESSURE IN THE INTACT KIDNEY

(21 KIDNEYS, 21 DOGS)

	EXTRAVASCULAR PRESSURE* (mm Hg)	MEAN ARTERIAL PRESSURE (mm Hg)	RENAL BLOOD FLOW (cc/min./gm)
MEAN	37	122	4.0
RANGE	18-50	90-145	2.3-5.5
S. E.	2	3	0.2

*Determined by two to four procedures described in text, in 17 kidneys.
Determined by value of deep vein pressure in 4 kidneys.

TABLE II

DEEP VEIN PRESSURE AND RENAL ARTERY PRESSURE IN INTACT KIDNEYS*

EXPT. NO.	DEEP VEIN PRESSURE (mm Hg)	RENAL ARTERY PRESSURE (mm Hg)
13	45	123
14	37	145
15	37	128
16	61	151
17	45	131
18	24	110
19	50	140
20	41	109
21	41	128
MEAN	42	129

*Pressure recorded early in experiment at time of equilibrium of renal blood flows. (Average RBF = 4.2 cc./min./gm.; range, 2.7-5.5)

TABLE III

AUTOREGULATION AND EXTRAVASCULAR PRESSURE IN INTACT KIDNEYS

EXPT. NO.	Δ RENAL ARTERY PRESSURE (mm Hg)	Δ EXTRAVASCULAR PRESSURE (mm Hg)	Δ RESISTANCE R_T*	Δ RESISTANCE R_V*	% Δ R_V OF TOTAL R_T
7	82→ 142	10→ 27	0.53→ 0.79	0.07→ 0.15	31
8	100→ 185	9→ 58	0.58→ 0.81	0.05→ 0.25	87
9	92→ 132	23→ 46	0.35→ 0.41	0.09→ 0.15	100
13	125→ 166	38→ 60	1.14→ 1.44	0.35→ 0.52	57
14	98→ 172	8→ 38	0.74→ 1.00	0.06→ 0.22	61
16	128→ 151	47→ 61	0.91→ 1.09	0.34→ 0.44	56
21	80→ 133	13→ 44	0.45→ 0.61	0.07→ 0.20	81
MEAN	101→ 155	21→ 48	0.67→ 0.88	0.15→ 0.28	
S. E.	7 8	6 5	0.12 0.13	0.05 0.06	

MEAN RBF = 4.9 cc./min./gm.
(range 2.7-6.8) ; at RAP 123-145 mm Hg.
*R_T = RAP/RBF
R_V = DVP—LVP/RBF

TABLE IV

HEMODYNAMIC EFFECTS OF LARGE VEIN PRESSURE ELEVATION IN THE INTACT KIDNEY (17 dogs)

RENAL VENOUS PRESSURE RANGE (mm Hg)	CONTROL 0		1 — 15		16 — 30		31 — 45		46 — 60		61 — 75	
	R_T*	RBF*	R_T	RBF	R_T	RBF	R_T	RBF	R_T	RBF	R_T	RBF
MEAN	0.95	156	0.78	162	0.78	153	0.77	151	0.64	146	0.73	126
S. E.	0.1	9	0.1	11	0.1	9	0.1	10	0.1	14	0.1	9
NO. EXPTS.	33	33	24	24	28	28	30	30	17	17	19	19

*R_T = TOTAL RESISTANCE (RAP — LVP/F) (mm Hg/cc/min)
RBF = RENAL BLOOD FLOW (cc/min)

TABLE V

EFFECT OF INCREASED LARGE VEIN PRESSURE ON TOTAL AND VENOUS SEGMENT RESISTANCES IN THE INTACT KIDNEY*

EXPT. NO.	LARGE VEIN PRESSURE RANGE (mm Hg)																							
	0				1 — 15				16 — 30				31 — 45				46 — 60				61 — 75			
	R_T†	R_V	DVP	RBF	R_T	R_V	DVP	RBF	R_T	R_V	DVP	RBF	R_T	R_V	DVP	RBF	R_T	R_V	DVP	RBF	R_T	R_V	DVP	RBF
13	1.1	0.4	48	104	1.0	0.3	50	106					0.9	0.2	55	100	0.8	0.1	65	90				
13	1.4	0.5	60	114					1.3	0.4	62	114	1.2	0.3	65	110	1.1	0.2	73	108	1.0	0.2	85	98
15	0.8	0.2	36	162	0.7	0.1	36	162	0.7	0.1	40	163	0.6	0.1	56	148								
16	0.7	0.2	27	194	0.6	0.1	29	194	0.5	0.1	35	184												
17	0.6	0.2	34	201	0.5	0.1	36	205	0.5	0.1	42	205	0.4	0.1	50	192								

*Renal artery pressure constant, each experiment.

†R_T = Total renal resistance; RAP-LVP/F (mm Hg/cc/min)

R_V = Venous segment resistance; DVP-LVP/F (mm Hg/cc/min)

DVP = Deep vein pressure (mm Hg)

RBF = Renal blood flow (cc/min)

TABLE VI

EFFECT OF INCREASED URETERAL PRESSURE ON RENAL BLOOD FLOW IN INTACT DENERVATED KIDNEYS

EXPT. NO.	WITHOUT DIURESIS		†E. P. (mm Hg)	WITH DIURESIS		†E. P. (mm Hg)
	△ UP* (mm Hg)	△ RBF (cc/min)		△ UP* (mm Hg)	△ RBF (cc/min)	
1				0→ 71→ 73	168→ 180→ 122	
2				0→ 75	276→ 213	
3				0→ 85	183→ 150	48
4				0→ 85	120→ 94	48
5	0→ 65	156→ 130	44			
7	0→ 24	276→ 255				
8	0→ 44	162→ 156	45			
	0→ 63	188→ 162				
9	0→ 24→ 33	264→ 246→ 237	23			
11	0→ 53	140→ 150	48			
12	0→ 17→ 42	150→ 158→ 138	16			
13	0→ 25→ 68	110→ 114→ 92	38			
	0→ 93	137→ 120	85			
17	0→ 35→ 59	216→ 228→ 198	38			
22	0→ 72	207→ 228		0→ 95	258→ 180	
23	0→ 31	144→ 141	25			

*Intermediate values of UP are to be associated with intermediate values of RBF.
†Determined by value of deep venous pressure and/or value of increased large vein pressure resulting in significant decrease in renal blood flow.

TABLE VII

EFFECT OF CAROTID ARTERY OCCLUSION ON VASCULAR RESISTANCE OF ISOLATED PERFUSED KIDNEY*

CAROTID OCCLUSION

8 EXPTS.	△ SYSTEMIC ARTERIAL PRESSURE (mm Hg)		△ RENAL RESISTANCE (mm Hg/cc/min)	
MEAN	132→	193	1.8→	2.4
S. E.	3.9	12.5	0.2	0.4

RELEASE OF CAROTID OCCLUSION

6 EXPTS.				
MEAN	171→	125	1.9→	1.6
S. E.	7.9	5.2	0.2	0.3

*Arterial pressure or renal blood flow of isolated kidney maintained constant.

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