MINIREVIEW

Perspectives on Renal Blood Flow Autoregulation (43321E)

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The purpose of this review is to discuss the control systems involved in renal blood flow (RBF) autoregulation and, in particular, the role of tubuloglomerular feedback (TGF) and myogenic mechanisms. We used primarily a comparison of studies from the rat versus the dog in an attempt to provide some new perspectives that may help our understanding of this subject. Most of the discussion focuses on the autoregulation of renal cortical blood flow as derived from *in vivo* studies. In this minireview, we were unable to discuss all of the pertinent studies that have been performed due to the extensive literature on this subject. Consequently, some of the more recent and perhaps key observations that have not been covered in other recent reviews will be highlighted. For other related topics not discussed in this paper, we refer the reader to other excellent reviews (1–5). Before discussing the mechanisms involved with RBF autoregulation, however, a brief overview is necessary.

The regulation of renal hemodynamics is a vital component in the overall control of renal function. Changes in renal vascular resistance and, therefore, in RBF can have profound effects on the rate of filtration, on tubular function and excretion, and ultimately on the fluid-volume state of the organism. Consequently, the influence of intrinsic and extrinsic mechanisms that influence renal hemodynamics is an important topic and one that has been widely studied for many years.

Among the different possibilities for hemodynamic control of kidney function, there are several mechanisms thought to be of prime importance. One variable that can influence the glomerular filtration rate (GFR), particularly in the condition of filtration pressure equilibrium, is the rate of plasma flow through the renal cortex (6). The degree to which the nephron finely regulates the excretion of a variety of different solutes is critically dependent upon the amount of the solute filtered. Hemodynamic control of excretory function is also thought to occur via the phenomenon known as pressure natriuresis, which is characterized by a direct relationship between renal perfusion pressure and sodium excretion. Guyton and his colleagues (7) contend that arterial blood pressure, through pressure-induced increases in sodium excretion, is a critical factor in the regulation of extracellular fluid volume and, therefore, of arterial blood pressure homeostasis. Another factor that is influenced by renal hemodynamics is the renin-angiotensin system; below a threshold pressure of approximately 95 mm Hg in the dog (8) and 85 mm Hg in the rat (9), the renin secretion rate and plasma renin activity are inversely proportional to renal perfusion pressure. Details concerning the role of the renin-angiotensin system in the control of renal hemodynamics were recently reviewed by Hall (10).

The regulation of RBF and GFR acts as an initial buffering system which, in turn, allows for a fine control of tubular reabsorptive events. The mechanism(s) responsible for the maintenance of normal RBF and GFR has not been fully elucidated, but it is generally believed that a constant volume of blood flow through the renal cortex is essential for the maintenance of a normal GFR.

Autoregulation: Myogenic versus Tubuloglomerular Feedback Mechanisms

Selkurt (11) and Shiply and Study (12) were among the first investigators to fully describe autoregulatory events in the kidney. These authors demonstrated that the organ was characterized by an ability to maintain a

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comparatively stable blood flow and glomerular filtration within a relatively large mean arterial pressure range (approximately 80–180 mm Hg). Representative data for both the dog and rat are shown in Figure 1. Two systems are now generally considered to be of fundamental importance in the phenomenon of renal autoregulation: one is based on an inherent myogenic response of vascular smooth muscle and the other is related to events in the macula densa region of the nephron, a tubuloglomerular feedback mechanism. The former mechanism is based on the ability of arterial vessels to alter endogenous tone in response to changes in transmural pressure, a well-known phenomenon described as long ago as 1902 (13). With increases or decreases in luminal pressure, vascular smooth muscle contracts or relaxes to produce the appropriate changes in resistance and thereby maintains a constant flow. Tubuloglomerular feedback is a phenomenon unique to the kidney, in which changes in the composition of tubular fluid flowing past the macula densa region of the nephron result in alterations in preglomerular resistance (and possibly postglomerular resistance as well) and, thus, maintain a constant glomerular capillary pressure. The mechanisms involved in the signal-sensing and transduction steps have not been fully elucidated and have been the subject of some controversy (for a recent review, see Ref. 14). Nonetheless, based on theoretical as well as anatomical considerations, the TGF mechanism appears well suited to play a major role in controlling preglomerular (and possibly postglomerular) resistance via pressure-induced changes in tubular flow past the macula densa. There is, however, considerable disagreement among investigators with regard to the degree to which each of these mechanisms participates in the control of autoregulation. The debate relating to TGF versus myogenic control of the renal circulation has evolved primarily from studies in which TGF is either intact or inhibited.

Several lines of evidence support the conclusion that an inherent myogenic capacity of the renal vasculature is an important, and perhaps is the primary, mechanism by which the kidney maintains a constant blood flow in the face of changing pressure (15–19). Based on studies in the rat, Aukland (15) has proposed that when renal perfusion pressure is slightly reduced, larger vessels (interlobar arteries) initially respond by vasodilating, a process that extends into smaller vessels as perfusion pressure is further reduced. By contrast, available evidence in the dog suggests that there are no pressure-induced changes in tone of larger renal vessels (20). Further support for a non-TGF mechanism in the renal autoregulatory phenomenon arises from studies in rats by Arendshorst and colleagues (21–23) in which TGF was inhibited yet autoregulation was maintained at full efficiency. Changes in glomerular capillary pressure estimated by stop-flow techniques are significantly inhibited during infusion of atrial natriuretic peptide or furosemide or during acute ureteral occlusion, while autoregulatory adjustments in blood flow during acute changes in renal perfusion pressure remain intact (21–23).

Perhaps the strongest evidence that TGF mediates autoregulation in the kidney comes from findings in which inhibition of TGF produced by plasma volume expansion in the rat is associated with a concurrent decrease in autoregulatory efficiency (24). Holstein-

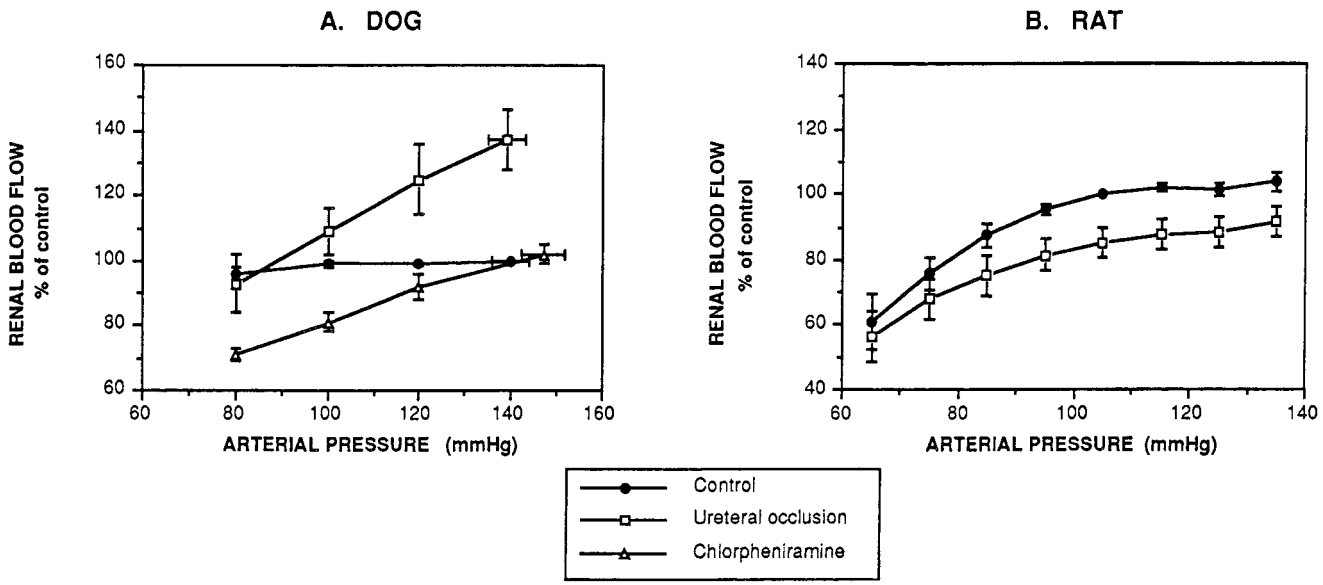

Figure 1. Effects of changes in mean arterial pressure on renal blood flow during control periods and following acute ureteral obstruction in (A) the dog and (B) rat. The effect of intra-arterial infusion of the histamine H_1 receptor antagonist, chlorpheniramine (10^{-5} mole/min), is also depicted for the dog. Renal blood flow data are expressed as a percentage of flow at baseline pressure (dog) or at 105 mm Hg (rat) during the control period. Figure is redrawn from References 22, 26, and 29. Values represent mean ± SE.

Rathlou and co-workers (25) recently used a mathematical model of TGF and standard linear techniques in rats to support a primary role for TGF in the dynamic regulation of renal blood flow (25), although other investigators using similar methods have not come to the same conclusion in terms of the relative contribution of TGF and myogenic components (22).

Although a role for TGF as a critical component in the maintenance of autoregulation remains a possibility, the true functional role of TGF has not been elucidated. While TGF is well-designed to have a direct influence on afferent arteriolar resistance and thus, perhaps, autoregulation, a more significant role may be in the control of fluid delivery to the distal tubule and collecting duct; thus, TGF may function as a regulator of excretory function. Since TGF is unique to the kidney, autoregulatory mechanisms would have to be different among vascular beds that obviously do not display this phenomenon. In other words, it has yet to be determined whether the mechanisms that control autoregulation are similar in all vascular beds.

Species Differences in the Control of Renal Hemodynamics

A comparison of data obtained from different animals, primarily from the dog and rat, supports the concept that there may be significant differences in the degree to which the TGF and myogenic components contribute to autoregulation of renal blood flow among species. In general, the renal circulation of the dog is characterized by a greater basal renal vascular resistance compared with the rat. Thus, RBF in the dog averages about 3–4 ml/min/g kidney wt, whereas in the rat, the value is at least 7 ml/min/g kidney wt; representative data are summarized in Table I. These data predict that the dog kidney should be more sensitive to the action of vasodilator agents, which is indeed the case. Many vasodilators can increase RBF in the dog as much as 100%, whereas the range is 20–30% in normal rats. Also, a higher vasodilatory reserve in the dog compared with the rat is indicated by the fact that the canine kidney is capable of a much larger autoregulation-induced vasodilation. Typically, the lower limit of autoregulation in the dog is in the range of 65–80 mm Hg of renal arterial pressure, whereas in the rat, this limit is roughly 10–20 mm Hg higher; the pressure at which renal blood flows begins to decline tends to be lowest in conscious animals (8, 9).

Table I. Typical Renal Blood Flow and Renal Vascular Resistance Values for the Dog and Rat[a]

	Dog	Rat
Renal blood flow (ml/min/g kidney wt)	2.9 ± 0.3	7.1 ± 0.8
Renal vascular resistance (mm Hg/[ml/min/g kidney wt])	53.4 ± 4.0	19.5 ± 1.7

[a] Average values (± SE) were derived from data included in References 26 and 29 for the dog and 23, 38, and 50 for the rat.

Ureteral Occlusion. Ureteral occlusion is a maneuver that can be used to block TGF and thereby evaluate its role in RBF autoregulation. When the flow of tubular fluid through the macula densa region of the nephron is reduced or absent, the TGF system cannot efficiently respond to signals that might be transmitted as a result of alterations in renal perfusion pressure. In the dog, acute ureteral occlusion results in a 30–40% increase in RBF within 10–15 min and, as depicted in Figure 1 (26), causes a concurrent loss in renal blood flow autoregulation; similar results have been reported by Navar and Baer (27) and by Blackshear *et al.* (28). Presumably, during ureteral occlusion, there is a reduction of tubular flow rate resulting in a TGF-induced decrease in renal vascular resistance. It is possible that the loss of autoregulation during ureteral occlusion in the dog could be related to the degree of renal vasodilation. However, a 40% increase in RBF produced by histamine, similar to the increase produced by ureteral occlusion, does not affect autoregulation; an increase in RBF of at least 60% is required before autoregulation becomes compromised (26, 29). By contrast, as is also illustrated in Figure 1, ureteral occlusion in the rat causes little change in either renal blood flow or renal blood flow autoregulation (22). Indeed, RBF actually decreases to a small degree during ureteral occlusion in the rat. Collectively, these results indicate that TGF is an important element in RBF autoregulation in the dog, but not in the rat.

Experiments using chronic disruption of TGF also support the existence of species differences in the mechanisms of autoregulatory control. Chronic ureteral obstruction in the dog or rat results in damage to the renal tubules and renders the kidney nonfilterable (30–32). In the nonfiltering kidney of the dog, autoregulatory efficiency is significantly decreased, although not totally abolished (30, 31). However, autoregulation is maintained in nonfiltering kidneys of rats as assessed by visualizing changes in preglomerular vessel diameter (32).

Histamine. Another indication that different mechanisms account for renal autoregulatory events in the dog and rat derives from experiments in which the potential role of histamine (H) in this process has been evaluated. Both H_1 and H_2 receptors mediate the renal hemodynamic actions of histamine in the dog (33). Moreover, virtually every class of H_1 receptor antagonist blocks autoregulation of RBF in this species, indicating an important role of H_1 receptor activation in the renal vasodilatory response to decreases in renal perfusion pressure (29). By contrast, even though only H_1 receptors mediate the renal hemodynamic actions of histamine in the rat (34), intravenous infusions of

histamine receptor antagonists have no effect on GFR autoregulation in this species (Table II; R. O. Banks, previously unpublished observations). In the dog, H_1 receptor antagonists also severely attenuate reactive hyperemic responses following acute decreases in renal perfusion pressure (29; Fig. 1), inhibit the response to some vasodilatory prostaglandins (35), and, of particular relevance to the discussion of ureteral occlusion, block the vasodilatory response to acute ureteral occlusion (26). Only H_1 receptors appear to be involved, since specific H_2 receptor antagonists have no effect on these responses. Therefore, in the dog, reductions in renal perfusion pressure and/or decreases in flow past the macula densa may result in afferent arteriolar vasodilator events that are mediated by histamine.

Autoregulation during Changes in Preglomerular Vascular Tone

Schnermann *et al.* (36) have provided evidence in rats that there is a direct relationship between the degree of vasodilation and the inhibition of TGF. It is intuitively obvious that if both the TGF and autoregulatory events are localized to the same site within the preglomerular vasculature, then alterations in vascular tone such as those which occur with infusion of exogenous vasodilators should interfere with TGF and autoregulatory signals. In the dog, vasodilator-induced increases in RBF of 30–40% do not compromise the ability of the kidney to autoregulate (29, 37). By contrast, when RBF in the dog is increased to values approaching 100% that of baseline, i.e., within the RBF range (on a per gram kidney weight basis) normally observed in the rat, autoregulation is markedly attenuated. As noted above, Aukland (15) has provided evidence in the rat that decreases in renal arterial pressure are associated with an initial dilation of large, preglomerular vessels, a response that is transmitted further along the vasculature to the smaller vessels with continued pressure reductions. Therefore, agents that affect TGF and the resistance of small vessels may not have a readily discernible effect on autoregulation in the range of renal perfusion pressures in which RBF is more efficiently controlled. The possibility of segmental differences in autoregulatory responsiveness is also evidenced by the observations that dopamine$_1$ receptor activation in the rat produces relatively large increases in RBF (23%) and decreases in resistance, yet has minimal influence on TGF responsiveness and autoregulation (38).

Atrial natriuretic peptide (ANP) is often cited as a specific renal vasodilator. However, even at pharmacological doses, ANP infusion into the renal artery of dogs is associated with only a transient renal vasodilation (39); when infused in physiological doses, it actually causes a small decrease in RBF (40). The actions of ANP are unique in that ANP decreases preglomerular resistance while increasing postglomerular resistance (23, 41). During infusion of exogenous ANP into the rat, autoregulatory responses associated with decreases in renal perfusion pressure are unaffected, while TGF is attenuated (23). In addition, autoregulation is maintained during ANP infusion in the hydronephrotic kidney, a condition in which the TGF mechanism is completely inoperative (32). The direct effects of ANP on TGF in the dog have not been evaluated, but ANP does not affect either GFR autoregulation or RBF autoregulation in this species (42). In summary, these findings suggest that the kidney has the ability to call upon a "vasodilatory reserve" and thereby maintain normal autoregulation, despite ANF-induced preglomerular vasodilation and TGF inhibition. Furthermore, these studies are consistent with the idea that TGF and ANP may be regulators of fluid-volume conditions rather than hemodynamic variables *per se.*

Both Davis *et al.* (43) and Schnermann and Briggs (44) reported that autoregulation of single-nephron GFR in the rat remains intact during supramaximal stimulation of the feedback signal (by maintaining loop of Henle flow rates above 40 nl/min) to presumably block any feedback control. They also observed that autoregulation was not maintained under conditions of maximal inhibition of TGF (zero flow through the loop of Henle). Nonetheless, these authors concluded that single nephrons can autoregulate efficiently without contribution of the TGF mechanism, suggesting that TGF has a relatively minor role in terms of autoregulatory control in the rat. Similarly, Daniels and Arend-

Table II. Glomerular Filtration Rate at Baseline and Reduced Renal Arterial Pressure in Rats with and without Histamine Receptor Antagonists[a]

	Baseline RAP (mm Hg)	GFR at baseline RAP (ml/min)	GFR at 80 mm Hg (ml/min)
Control (n = 7)	109 ± 5	1.5 ± 0.2	1.3 ± 0.1
Chlorpheniramine alone (n = 6)	120 ± 8	1.6 ± 0.1	1.7 ± 0.3
Chlorpheniramine + cimetidine (n = 6)	119 ± 4	1.1 ± 0.2	1.4 ± 0.4

[a] The H_1 antagonist, chlorpheniramine, was infused intravenously at 0.1 mg/kg/min and the H_2 antagonist, cimetidine, was infused intravenously at 10 mg/hr. Renal arterial pressure (RAP) was reduced to 80 mm Hg by means of an adjustable clamp positioned around the abdominal aorta proximal to the renal arteries. Values represent mean ± SE.

shorst (21) have recently demonstrated in the rat that furosemide inhibits TGF without affecting RBF autoregulation. By contrast, a more critical role seems plausible for the dog, since there is only a transient loss of RBF autoregulation during the first 15 min of furosemide infusion (45), a time interval in which there is also a loss of TGF activity (46).

Effector Site of Autoregulatory Resistance Changes: Potential Role of the Renin-Angiotensin System

No matter whether myogenic and/or TGF mechanisms mediate pressure-induced changes in renal vascular resistance, the effector site must involve preglomerular vessels or a combination of pre- and postglomerular elements. Many investigators often assume that the autoregulation of RBF and GFR are controlled solely by changes in preglomerular resistance. However, there is evidence that autoregulation of these variables can be dissociated and, therefore, must have some dissimilar control mechanisms (8, 47). Based on data obtained from dogs maintained on a relatively low sodium diet, Hall *et al.* (47) have reported that the GFR during reduced renal arterial pressure is maintained by at least two mechanisms: one is independent of the renin-angiotensin system and the other involves angiotensin II-mediated arteriolar constriction, possibly localized to the efferent arteriole. The basic observation centers on the fact that at reduced renal arterial pressures, the inhibition of angiotensin-converting enzyme (ACE) is associated with a decrease in the GFR. The mechanism by which angiotensin II stabilizes the GFR in the dog with renal artery stenosis has been reviewed by Anderson *et al.* (48). One of the unresolved issues has been related to the fact that ACE inhibition following several weeks of renal artery stenosis is not associated with an increase in renal blood flow, i.e., there is no net decrease in total resistance (renal vascular resistance plus the resistance of the stenosis). Anderson *et al.* (49) reported recently, however, that there is a net decrease in renal vascular resistance in these long-term stenotic kidneys following ACE inhibition, but that there is an offsetting, flow-induced increase in the resistance of the stenosis. Thus, the effect of ACE inhibition on the GFR can be obtained within minutes to hours of reducing renal arterial pressure, a time interval during which plasma renin concentrations are elevated, or following a reduction in renal arterial pressure of several weeks, a time interval during which plasma renin concentrations have usually returned to within the normal range. Whether an intrarenal renin-angiotensin mechanism still exerts the same effect after several weeks of a functional renal artery stenosis or whether other factors such as an enhanced kinin system following ACE inhibition are involved in these responses will require further investigation.

Conclusion

There is a continuing debate in regard to the relative contribution of TGF and myogenic mechanisms in the control autoregulation in the kidney. Much of the controversy relating to the relative importance of each mechanism can possibly be explained simply by considering the species from which evidence is derived. Differences in autoregulatory behavior between the rat and the dog, as noted by Navar *et al.* (20), have been attributed by some investigators to the effects of anesthesia (8). However, in the current review, most of our comparisons were based on data obtained from anesthetized rats and dogs. Thus we propose that the factors responsible for autoregulatory control are indeed different between the dog and the rat. It is our opinion that based on the available evidence, TGF plays a substantial role in the autoregulation of RBF in the dog, while having only a minimal influence in the rat.

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1. Brenner BM, Troy JL, Ballermann BJ. Endothelium-dependent vascular responses. Mediators and mechanisms. J Clin Invest **84**:1373–1378, 1989.
2. Carmines PK, Fleming JT. Control of the renal microvasculature by vasoactive peptides. FASEB J **4**:3300–3309, 1990.
3. Chou S-Y, Porush JG, Faubert PF. Renal medullary circulation. Hormonal control. Kidney Int **37**:1–13, 1990.
4. Navar LG. Renal autoregulation. Perspective from whole kidney and single nephron studies. Am J Physiol **234**:F357–F370, 1978.
5. Stein JH. Regulation of the renal circulation. Kidney Int **38**:571–576, 1990.
6. Dean WM, Robertson CR, Brenner BM. A model of glomerular ultrafiltration in the rat. Am J Physiol **223**:1178–1183, 1972.
7. Guyton AC, Coleman TG, Cowley AW Jr, Manning RD Jr, Norman RA Jr, Ferguson JD. A systems analysis approach to understanding long-range arterial blood pressure control and hypertension. Circ Res **251**:159–176, 1974.
8. Kirchheim HR, Ehmke H, Hackenthal E, Lowe W, Persson P. Autoregulation of renal blood flow, glomerular filtration rate and renin release in conscious dogs. Pflugers Arch **410**:441–449, 1987.
9. Conrad KP, Brink-Johnson T, Gellai M, Valtin H. Renal autoregulation in chronically catheterized conscious rats. Am J Physiol **147**:F229–F233, 1984.
10. Hall JE. Control of sodium excretion by angiotensin II: Intrarenal mechanisms and blood pressure regulation. Am J Physiol **250**:R960–R972, 1986.
11. Selkurt EE. The relation of renal blood flow to effective arterial pressure in the intact kidney of the dog. Am J Physiol **147**:537–549, 1946.
12. Shipley RE, Study RS. Changes in renal blood flow, extraction of inulin, GFR, tissue pressure and urine flow with acute alteration of renal blood pressure. Am J Physiol **167**:676–688, 1951.
13. Bayliss WM. On the local reactions of the arterial wall to changes of internal pressure. J Physiol **28**:220–231, 1902.
14. Briggs JP, Schnermann J. The tubuloglomerular feedback mechanism: Functional and biochemical aspects. Annu Rev Physiol **49**:251–273, 1987.

15. Aukland K. Myogenic mechanisms in the kidney. J Hyperten 7(suppl 4):S71–S76, 1989.
16. Aukland K, Oien AH. Renal autoregulation: Models combining tubuloglomerular feedback and myogenic response. Am J Physiol **252**:F768–F783, 1987.
17. Jensen PK, Steven K. A model study of the tubuloglomerular feedback mechanism: Effector site and influence on renal autoregulation. Acta Physiol Scand **115**:295–300, 1982.
18. Kaskel F, Devarajan P, Birzgalis A, Moore LC. Inhibition of myogenic autoregulation in cyclosporine nephrotoxicity in the rat. Renal Physiol Biochem **12**:250–259, 1989.
19. Lush DJ, Fray JCS. Steady-state autoregulation of renal blood flow: A myogenic model. Am J Physiol **247**:R89–R99, 1984.
20. Navar LG, Bell PD, Burke TJ. Role of a macula densa feedback mechanism as a mediator of renal autoregulation. Kidney Int **22**(suppl 12):S157–S164, 1982.
21. Daniels FH, Arendshorst WJ. Autoregulation of renal blood flow in the rat is not impaired by abolition of tubuloglomerular feedback. FASEB J **3**:A1016, 1989.
22. Daniels FH, Arendshorst WJ, Roberds RG. Tubuloglomerular feedback and autoregulation in spontaneously hypertensive rats. Am J Physiol **258**:F1479–F1489, 1990.
23. Pollock DM, Arendshorst WJ. Native tubular fluid attenuates ANF-induced inhibition of tubuloglomerular feedback. Am J Physiol **258**:F189–F198, 1990.
24. Navar LG, Bell PD, Ploth DW. Role of feedback mechanism in renal autoregulation and sensing step in feedback pathway. Fed Proc **40**:93–98, 1981.
25. Holstein-Rathlou N-H, Wagner AJ, Marsh DJ. Tubuloglomerular feedback dynamics and renal blood flow autoregulation in rats. Am J Physiol **260**:F53–F68, 1991.
26. Banks RO, Jacobson ED. Renal vasodilation with ureteral occlusion and prostaglandins: Attenuation by histamine H_1 antagonists. Am J Physiol **249**:F851–F857, 1985.
27. Navar LG, Baer PG. Renal autoregulatory and glomerular filtration responses to graded ureteral occlusion. Nephron **7**:301–316, 1970.
28. Blackshear JL, Edwards BS, Knox FG. Autoregulation of renal blood flow. Miner Electrolyte Metab **2**:130–136, 1979.
29. Banks RO, Inscho EW, Jacobson ED. Histamine H_1 receptor antagonists inhibit autoregulation of renal blood flow in the dog. Circ Res **54**:527–535, 1984.
30. Gotshall R, Hess T, Mills T. Efficiency of canine renal blood flow autoregulation in kidneys with or without glomerular filtration. Blood Vessels **22**:25–31, 1985.
31. Osborn JL, Hoversten LG, DiBona GF. Impaired blood flow autoregulation in nonfiltering kidneys: Effects of theophylline administration. Proc Soc Exp Biol Med **174**:328–335, 1983.
32. Steinhausen M, Blum M, Fleming JT, Holz FG, Parekh N, Weigman DL. Visualization of renal autoregulation in the split hydronephrotic kidney of rats. Kidney Int **35**:1151–1160, 1989.
33. Banks RO, Fondacaro JD, Schwaiger MM, Jacobson ED. Renal histamine H_1 and H_2 receptors: Characterization and functional significance. Am J Physiol **235**:F570–F575, 1978.
34. Ichikawa I, Brenner BM. Mechanisms of action of histamine and histamine antagonists on the glomerular microcirculation in the rat. Circ Res **45**:737–745, 1979.
35. Banks RO. Vasoconstrictor-induced changes in renal blood flow: Role of prostaglandins and histamine. Am J Physiol **254**:F470–F476, 1988.
36. Schnermann J, Todd KM, Briggs JP. Effect of dopamine on the tubuloglomerular feedback mechanism. Am J Physiol **242**:F790–F798, 1990.
37. Baer PG, Navar LG, Guyton AC. Renal autoregulation, filtration rate, and electrolyte excretion during vasodilation. Am J Physiol **219**:615–619, 1970.
38. Pollock DM, Arendshorst WJ. Tubuloglomerular feedback and blood flow autoregulation during DA_1-induced renal vasodilation. Am J Physiol **258**:F627–F635, 1990.
39. Burnett JC, Granger JP, Opgenorth TJ. Effects of synthetic atrial natriuretic factor on renal function and renin release. Am J Physiol **247**:F863–F866, 1984.
40. Banks RO. Effects of a physiological dose of ANP on renal function in dogs. Am J Physiol **255**:F907–F910, 1988.
41. Dunn BR, Ichikawa I, Pfeffer JM, Troy JL, Brenner BM. Renal and systemic hemodynamic effects of synthetic atrial natriuretic peptide in the anesthetized rat. Circ Res **59**:237–246, 1986.
42. Paul RV, Kirk KA, Navar LG. Renal autoregulation and pressure natriuresis during ANF-induced diuresis. Am J Physiol **253**:F424–F431, 1987.
43. Davis JM, Kawata T, Haberle DA. The influence of renal autoregulation on TGF control of glomerular filtration rate. In: Persson AEG, Boberg U, Eds. Eric K. Fernstrom Symposium on The Juxtaglomerular Apparatus, Amsterdam: Elsevier, pp393–406, 1988.
44. Schnermann J, Briggs JP. Interaction between loop of Henle and arterial pressure as determinants of glomerular pressure. Am J Physiol **256**:F421–F429, 1989.
45. Duchin KL, Peterson LA, Burke TJ. Effect of furosemide on renal autoregulation. Kidney Int **12**:379–386, 1977.
46. Bell PD, Thomas C, Williams RH, Navar LG. Filtration rate and stop flow pressure feedback responses to nephron perfusion in the dog. Am J Physiol **234**:F154–F168, 1978.
47. Hall JE, Guyton AC, Jackson TE, Coleman TC, Lohmeier TE, Trippodo NC. Control of glomerular filtration rate by renin-angiotensin system. Am J Physiol **233**:F366–F372, 1977.
48. Anderson WP, Dinton KM, Woods RL, Alcorn D. Angiotensin II and the maintenance of GFR and renal blood flow during renal artery narrowing. Kidney Int **38**:S109–S113, 1990.
49. Anderson WP, Ramsey DE, Takata M. Development of hypertension from unilateral renal artery stenosis in conscious dogs. Hypertension **16**:441–451, 1990.
50. Pollock DM, Arendshorst WJ. Effect of atrial natriuretic factor on renal hemodynamics in the rat. Am J Physiol **251**:F795–F801, 1986.