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# Role of the efferent arteriole in glomerular hemodynamics of superficial nephrons

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**Role of the efferent arteriole in glomerular hemodynamics of superficial nephrons.** Hemodynamic pressure in glomerular capillaries (GCP) and in first order peritubular capillaries (EAP) in superficial nephrons of mutant Wistar rats with surface glomeruli was measured by micropuncture with a servo-nulling device in the following conditions: *1*) control; *2*) norepinephrine infusion (NE); *3*) epinephrine infusion (E); *4*) dopamine infusion (D); *5*) hemorrhagic hypotension (HH); *6*) HH + NE; *7*) HH + E; *8*) HH + D; *9*) acute hypertension secondary to bilateral cervical vagotomy and occlusion of both common carotid arteries. BP was also recorded. Both GCP/BP and EAP/GCP ratios averaged 0.40 in control conditions, but only the EAP/GCP ratio remained constant in all conditions under study, indicating that approximately 60% of the hydrostatic pressure in glomerular capillaries is constantly dissipated by the efferent arteriole. When all values of EAP were plotted against the respective values of GCP, a linear relationship was detected ($r$ = 0.843). These results indicate that changes of pressure in the first order peritubular capillaries of superficial nephrons are merely secondary to changes in glomerular capillary pressure.

**Rôle de l'artériole efférente dans l'hémodynamique glomérulaire des néphrons superficiels.** La pression hémodynamique dans les capillaires glomérulaires (GCP) et dans les capillaries péritubulaires de premier ordre (EAP) des néphrons superficiels de rats Wistar mutants, à glomérules superficiels, a été mesurée par microponction au moyen d'un dispositif à zéro asservi dans les conditions suivantes: *1*) témoins; *2*) perfusion de norépinéphrine (NE); *3*) perfusion d'épinéphrine (E); *4*) perfusion de dopamine (D); *5*) hypotension hémorragique (HH); *6*) HH + NE; *7*) HH + E; *8*) HH + D; *9*) hypertension aiguë secondaire à une vagotomie cervicale bilatérale et à l'occlusion des deux carotides primitives. La pression artérielle (BP) a été enregistrée. Les rapports GCP/BP et EAP/GCP sont en moyenne de 0,40 chez les témoins. Seul EAP/GCP demeure constant dans toutes les conditions étudiées, ce qui indique que 60% environ de la pression hydrostatique du capillaire glomérulaire sont constamment perdus par l'artériole efférente. Quand toutes les valeurs de EAP sont représentées graphiquement en fonction des valeurs correspondantes de GCP, on observe une relation linéaire ($r$ = 0,843). Ces résultats indiquent que les modifications de pression dans les capillaires péritubulaires de premier ordre des néphrons superficiels sont simplement secondaires aux modifications de la pression dans le capillaire glomérulaire.

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It has been postulated, on the basis of whole kidney clearance studies, that increases and decreases in filtration fraction are the result of constriction and dilatation, respectively, of the efferent arterioles [1]. Anatomical studies, however, have shown that the efferent arterioles of superficial nephrons have a very thin wall, with very few muscle cells [2, 3]. Furthermore, recent micropuncture studies have demonstrated that in several experimental conditions in which single nephron glomerular filtration rate was raised and lowered, glomerular hemodynamic pressures and single nephron filtration fraction in superficial nephrons remained constant, and changes in single nephron glomerular filtration rate were proportional to changes in glomerular plasma flow [4, 5]. These observations appeared to provide evidence against an active role of efferent arterioles in regulating filtration fraction in superficial nephrons.

The present study was undertaken to evaluate the role of the efferent arterioles in the control of the glomerular hemodynamics of superficial nephrons by studying the relationship between glomerular capillary pressure (GCP) and the pressure in the first order peritubular capillaries (so-called efferent arteriole pressure, EAP) in superficial nephrons of rat kidney in various conditions in which glomerular capillary pressure was varied experimentally.

## Methods

A mutant strain of Wistar rats was utilized in this study. This strain is identical to normal Wistar rats with the exception that a few glomeruli (between 1 and 15) can be seen on the kidney surface and are, therefore, accessible to renal micropuncture [6–8].

Thirty-six non-fasted female rats, weighing 200 to 300 g were anesthetized with sodium pentobarbital (Nembutal, 60 mg/kg body wt) i.p. The animal was placed on a heated and thermostatically controlled

micropuncture table. Tracheostomy was performed with PE 240 tubing. A catheter (PE 50) was inserted into the left femoral artery to monitor blood pressure; the left femoral vein and the left superficial jugular vein were cannulated (PE 50) for the infusion of solutions. The left kidney was exposed through a lateral abdominal incision ("ventral approach," [9]). The left ureter was cannulated with a length of PE 10 tubing for urine collection from the left kidney; another catheter (PE 50) was placed in the bladder to collect urine from the right kidney.

The adrenal gland, perirenal fat and connective tissue were dissected away from the left kidney, paying careful attention so as to avoid stretching of the pedicle or damage to the fibrous renal capsule. The kidney was then placed in a Lucite cup which was fixed to the micropuncture table. Cotton wicks were placed around the kidney, which was then covered with heated (37°C) physiological saline solution.

A servo-nulling pressure-measuring system, recently modified by Intaglietta, Pawula and Tompkins [10] (Instrumentation for Physiology and Medicine, San Diego, California, U.S.A.) was used to measure the renal cortical hemodynamic pressures.

Cortical hemodynamic pressures were measured in the glomerular capillaries (glomerular capillary pressure, GCP) and the large peritubular capillaries assumed to be the first ramifications of efferent arterioles (efferent arteriole pressure, EAP). The latter were identified easily because of their star shape around the welling point of the efferent arterioles [11–13].

Cortical hemodynamic pressures and arterial blood pressure (BP) were recorded simultaneously on a dual-channel recorder (7702 B Hewlett-Packard).

As soon as the left superficial jugular vein was cannulated, an i.v. infusion of bicarbonate-saline solution (NaCl, 110 mEq/liter; $NaHCO_3$, 28 mEq/liter; KCl, 5 mEq/liter) was begun at an infusion rate of 0.02 ml/min, and maintained thereafter [14].

After completion of the surgical procedures, at least one hour was allowed before starting the experimental periods.

Three experimental protocols were used to evaluate the role of the efferent arteriole in the hemodynamics of superficial nephrons.

*1) Effect of norepinephrine, epinephrine and dopamine in normal hydropenic rats (21 rats).* Cortical hemodynamic pressures and systemic BP were measured for 30 min in control conditions. Then an intravenous infusion of norepinephrine (0.5 to 0.8 μg/min; 11 rats) or epinephrine (0.5 to 0.8 μg/min; 5 rats) or dopamine (15 to 25 μg/min; 5 rats) was started; cortical pressures were measured again when BP had stabilized at the new levels for approximately 30 min.

In some rats a third measurement of pressures was performed again when BP had returned to control values after discontinuation of the drug.

*2) Effect of norepinephrine, epinephrine, and dopamine in rats during hemorrhagic hypotension (10 rats).* Cortical renal hemodynamic pressures and systemic blood pressure were measured for 30 min in control conditions. Then 1.5 to 2 ml of blood was withdrawn from the femoral arterial catheter in order to decrease blood pressure to approximately 70 mm Hg. The withdrawn blood was stored in heparinized vials. When BP had stabilized at the new low levels, cortical pressures were measured again. Then an intravenous infusion of norepinephrine (three rats), epinephrine (four rats) and dopamine (three rats) was started at such a dosage as to raise the BP to at least 85 mm Hg. The dosage of each drug, however, was actually the same as in the first experimental protocol. Cortical pressures were measured again during the infusion of the drug. The drug was then discontinued and the blood previously collected was transfused in the same animal; at the end of the transfusion cortical pressures were measured for the last time.

*3) Effect of acute hypertension secondary to bilateral cervical vagotomy and occlusion of both common carotid arteries (5 rats).* Systemic blood pressure and cortical renal hemodynamic pressures were measured for 30 min in control conditions. Then a bilateral cervical vagotomy was performed, and both common carotids were ligated. When BP had stabilized at the new higher levels, cortical renal hemodynamic pressures were measured again.

No bleeding on the kidney surface, or within the tubules, was observed upon withdrawal of the micropipette from the glomerular capillaries after each GCP measurement. This allowed several measurements of GCP in the same glomeruli in different experimental periods. When occasional bleeding was observed, the nephron was not used for further measurements.

## Results

*Effect of norepinephrine, epinephrine and dopamine in normal hydropenic rats (Tables 1, 2 and 3).* BP in the control period averaged 115 ± 9 SD mm Hg in the first group of 11 rats (Table 1), 124 ± 6 SD mm Hg in the second group of 5 rats (Table 2) and 125 ± 13 SD mm Hg in the third group of 5 rats (Table 3). These values are normal for hydropenic Wistar rats of the mutant strain with glomeruli on the kidney surface as reported in the recent literature [4, 7, 8]. In these three groups of rats, in the control periods, the GCP

**Table 1.** Effects of norepinephrine on arterial blood pressure and pressure in glomerular and peritubular capillaries in hydropenic rats[a]

| Observation | Control | Norepinephrine | Control |
|---|---|---|---|
| BP, *mm Hg* | 115 ± 9 | 149 ± 13 [b] | 116 ± 10 |
| GCP, *mm Hg* | 50.3 ± 6 | 37.7 ± 5.3[b] | 46.3 ± 6.7 |
| EAP, *mm Hg* | 19.8 ± 2.7 | 16.3 ± 2[b] | 19.9 ± 3.4 |

[a] BP = arterial blood pressure; GCP = glomerular capillary pressure; EAP = pressure in the first order peritubular capillaries (so-called efferent arteriole pressure). All values are mean ± SD.
[b] $P < 0.001$ (paired $t$ test).

averaged 50.3 ± 6 SD mm Hg, 47.8 ± 7.8 SD mm Hg and 47.8 ± 5.3 SD mm Hg, while the EAP averaged 19.8 ± 2.7 SD mm Hg, 18.4 ± 2.4 SD mm Hg and 20.1 ± 2.5 SD mm Hg, respectively. Also, these control values were similar to those previously reported by us and others [4, 8, 15–17]. During norepinephrine infusion (Table 1), while BP rose to 149 ± 13 SD mm Hg, GCP fell to 37.7 ± 5.3 SD mm Hg and EAP fell to 16.3 ± 2 SD mm Hg; these changes were highly significant ($P < 0.001$) using the paired $t$ test. After discontinuation of the drug, blood pressure fell and GCP and EAP rose to control values.

With epinephrine (Table 2) BP rose to 153 ± 12 SD mm Hg; similarly GCP and EAP showed a slight but significant increase.

Dopamine infusion (Table 3) effected a slight reduction of BP but GCP and EAP were increased.

*Effect of norepinephrine, epinephrine and dopamine in rats during hemorrhagic hypotension (Tables 4, 5 and 6).* The hemorrhage significantly decreased BP, GCP and EAP in all three groups of this protocol.

Norepinephrine infusion during hemorrhagic hypotension (Table 4) raised the BP to normotensive levels (108 mm Hg) but it did not change GCP (28.6 ± 2 SD vs. 28.3 ± 3.5 SD mm Hg) and EAP (12.0 ± 1.2 SD vs. 12.3 ± 0.9 SD mm Hg). After intravenous reinfusion of the previously withdrawn blood, BP and cortical hemodynamic pressures returned to normal values.

Epinephrine infusion during hemorrhagic hypotension (Table 5) significantly raised BP to normotensive levels, GCP from 26.5 ± 2.8 SD to 38.2 ± 5.7 SD mm Hg and EAP from 11.3 ± 3.2 SD to 16.3 ± 3.2 SD mm Hg. When the withdrawn blood was transfused after discontinuation of epinephrine, BP and cortical pressures returned to normal levels.

**Table 2.** Effects of epinephrine on arterial blood pressure and pressure in glomerular and peritubular capillaries in hydropenic rats[a]

| Observation | Control | Epinephrine | Control |
|---|---|---|---|
| BP, *mm Hg* | 124 ± 6 | 153 ± 12[c] | 122 ± 6 |
| GCP, *mm Hg* | 47.8 ± 7.8 | 51.8 ± 5.4[b] | 47.5 ± 5.0 |
| EAP, *mm Hg* | 18.4 ± 2.4 | 22.0 ± 5.3[b] | 18.9 ± 2.0 |

[a] Symbols as in Table 1. All values are mean ± SD.
[b] $P < 0.05$
[c] $P < 0.001$ (paired $t$ test).

**Table 3.** Effects of dopamine on arterial blood pressure and pressure in glomerular and peritubular capillaries in hydropenic rats[a]

| Observation | Control | Dopamine | Control |
|---|---|---|---|
| BP, *mm Hg* | 125 ± 13 | 112 ± 22[b] | 126 ± 10 |
| GCP, *mm Hg* | 47.8 ± 5.3 | 61.7 ± 8.5[b] | 49.0 ± 1 |
| EAP, *mm Hg* | 20.1 ± 2.5 | 25.0 ± 5.2[b] | 20.5 ± 5 |

[a] Symbols as in Table 1. All values are mean ± SD.
[b] $P < 0.05$ (paired $t$ test).

Dopamine infusion during hemorrhagic hypotension (Table 6) significantly increased BP (from 57 to 86 mm Hg), GCP (from 23.7 ± 4.5 SD to 44.3 ± 4 SD mm Hg) and EAP (from 7.5 ± 1.9 SD to 17.4 ± 1.5 SD mm Hg).

*Effect of acute hypertension secondary to bilateral cervical vagotomy and occlusion of both common carotid arteries (Table 7).* Bilateral vagotomy and carotid occlusion raised BP from 118 to 155 mm Hg; GCP and EAP increased only slightly.

## Discussion

It has been demonstrated recently that the filtration pressure within superficial nephrons achieves equilibrium in the most distal portion of the glomerular capillary network; i.e., effective filtration pressure (EFP) in the glomerular capillaries declines to zero before circulating blood has entered the efferent arteriole [1, 4, 7, 15, 16]. Under conditions of filtration pressure equilibrium at the efferent end of the glomerulus, single nephron glomerular filtration rate (SNGFR) depends entirely on glomerular plasma flow (GPF) for any given difference in transcapillary

**Table 4.** Effects of hemorrhage and hemorrhage + norepinephrine on arterial blood pressure and pressure in glomerular and peritubular capillaries in hydropenic rats[a]

| Observation | Control | Hemorrhage | Hemorrhage + NE | Blood transfusion |
|---|---|---|---|---|
| BP, *mm Hg* | 122 ± 6 | 71 ± 7[b] | 108 ± 14[c] | 155 ± 5[d] |
| GCP, *mm Hg* | 50.0 ± 6.2 | 28.6 ± 2[b] | 28.3 ± 3.5[b] | 45.2 ±4.3[d] |
| EAP, *mm Hg* | 20.5 ± 0.8 | 12.0 ± 1.2[b] | 12.3 ± 0.9[b] | 20.4 ± 2.5[d] |

[a] NE = norepinephrine. Other symbols as in Table 1. All values are mean ± SD.
[b] $P < 0.001$ (paired $t$ test vs. control).
[c] $P < 0.05$ (paired $t$ test vs. hemorrhage).
[d] $P < 0.001$ (paired $t$ test vs. hemorrhage).

**Table 5.** Effects of hemorrhage and hemorrhage + epinephrine on arterial blood pressure and pressure in glomerular and peritubular capillaries in hydropenic rats[a]

| Observation | Control | Hemorrhage | Hemorrhage + E | Blood transfusion |
|---|---|---|---|---|
| BP, *mm Hg* | 119 ± 7 | 62.5 ± 8[c] | 107 ± 6[b,d] | 105 ± 5[d] |
| GCP, *mm Hg* | 48.9 ± 4.5 | 26.5 ± 2.8[c] | 38.2 ± 5.7[b,d] | 47.4 ± 3.1[d] |
| EAP, *mm Hg* | 21.5 ± 2.4 | 11.3 ± 3.2[c] | 16.3 ± 3.2[b,d] | 20.9 ± 2.0[d] |

[a] E = epinephrine. Other symbols as in Table 1. All values are mean ± SD.
[b] $P < 0.05$ (paired $t$ test vs. control).
[c] $P < 0.001$ (paired $t$ test vs. control).
[d] $P < 0.001$ (paired $t$ test vs. hemorrhage).

**Table 6.** Effects of hemorrhage and hemorrhage + dopamine on arterial blood pressure and pressure in glomerular and peritubular capillaries in hydropenic rats[a]

| Observation | Control | Hemorrhage | Hemorrhage + D | Blood transfusion |
|---|---|---|---|---|
| BP, *mm Hg* | 128 ± 11 | 57 ± 2[b] | 86 ± 6[b,c] | 129 ± 15[c] |
| GCP, *mm Hg* | 45.1 ± 5.1 | 23.7 ± 4.5[b] | 44.3 ± 4[c] | 48.2 ± 1.7[c] |
| EAP, *mm Hg* | 17.6 ± 1.3 | 7.5 ± 1.9[b] | 17.4 ± 1.5[c] | 16.5 ± 0.1[c] |

[a] D = dopamine. Other symbols as in Table 1. All values are mean ± SD.
[b] $P < 0.001$ (paired $t$ test vs. control).
[c] $P < 0.001$ (paired $t$ test vs. hemorrhage).

hydrodynamic pressure [1]. This is supported by the recent finding, in rat kidney, of a rather constant filtration fraction in superficial nephrons (SNFF) prior to and following acute expansion of plasma and extracellular volume [4], and also during aortic constriction [5]. These results suggest that in superficial nephrons an active role of efferent arterioles is actually not necessary to account for changes in glomerular hemodynamics; i.e., neuro-humoral factors involved in regulation of glomerular hemodynamics may play their role without necessarily acting directly on efferent arterioles. In favor of this hypothesis are the anatomical studies in the rat which have shown that the media of efferent arterioles in superficial nephrons thins out shortly beyond the glomerulus, with complete disappearance of muscle cells, leaving the wall with only the basement membrane and endothelial cells [2, 3]. In other words, these efferent arterioles appear as thin-walled venules [3]. Moreover, histochemical studies in dog kidney have shown that adrenergic innervation of cortical nephrons is limited to afferent arterioles [18].

On the basis of these observations we have formulated the hypothesis that the efferent arterioles of superficial nephrons do not react directly to humoral and neural stimuli as afferent arterioles usually do.

This hypothesis was tested in the present studies by evaluating the percentage of pressure dissipated across the afferent and efferent arterioles in several experimental conditions in which BP and/or GCP were varied (Table 8). In reference to the efferent arterioles the occurrence of "active" constriction (i.e., contraction of the muscle fibers of the arteriolar wall) would have induced a fall in EAP in relation to GCP; whereas the occurrence of "active" dilatation (i.e., relaxation of the muscle fibers of the arteriolar wall) would have effected an increase of the EAP in relation to GCP.

In the experimental conditions that were chosen, a wide range of GCP's was obtained, ranging from 23.7 mm Hg during hemorrhagic hypotension to 61.7 mm Hg during dopamine infusion in hydropenic rats.

In control conditions the GCP/BP ratio and the EAP/GCP ratio averaged 0.39 and 0.40, respectively, thus indicating that in normal hydropenic rats approximately 60% of the systemic BP is dissipated across the afferent arteriole, and that 60% of the glomerular capillary pressure is dissipated by the efferent arteriole.

Norepinephrine and epinephrine infusion were both accompanied by a decrease of the GCP/BP ratio in both normal hydropenic and hemorrhagic rats, while dopamine increased the same ratio in both conditions. Hypertension secondary to bilateral vagotomy and carotid ligation decreased the GCP/BP ratio to 0.32, whereas hemorrhage did not change it.

In contrast with the wide variation of the GCP/BP ratio, the EAP/GCP ratio remained constant in all conditions under study (the slight changes were not statistically significant), in spite of the great changes in GCP. This indicates that approximately 60% of hemodynamic pressure in glomerular capillaries of superficial nephrons is constantly dissipated by the efferent arteriole, whatever the value of GCP.

When all values for EAP were plotted against the respective values of GCP, a linear relationship was detected as shown in Fig. 1.

**Table 7.** Effects of bilateral cervical vagotomy and occlusion of both common carotid arteries on arterial blood pressure and pressure in glomerular and peritubular capillaries in hydropenic rats[a]

| Observation | Control | Acute hypertension |
|---|---|---|
| BP, *mm Hg* | 118 ± 17 | 155 ± 15[c] |
| GCP, *mm Hg* | 44.6 ± 8.1 | 47.9 ± 7.1 |
| EAP, *mm Hg* | 16.7 ± 3 | 19.3 ± 3.5[b] |

[a] Symbols as in Table 1. All values are mean ± SD.
[b] $P < 0.05$
[c] $P < 0.001$ (paired $t$ test).

**Table 8.** Behavior of GCP/BP and EAP/GCP ratios in various experimental conditions[a]

| Observation | Control | NE | E | D | AH |
|---|---|---|---|---|---|
| GCP/BP | 0.39 | 0.26[b] | 0.34[b] | 0.56[c] | 0.32[c] |
| EAP/GCP | 0.40 | 0.42 | 0.41 | 0.40 | 0.40 |

| Observation | Control | Hemorrhage | Hemorrhage + NE | Hemorrhage + E | Hemorrhage + D |
|---|---|---|---|---|---|
| GCP/BP | 0.39 | 0.41 | 0.27[c] | 0.36 | 0.51[c] |
| EAP/GCP | 0.40 | 0.40 | 0.44 | 0.44 | 0.39 |

[a] NE = norepinephrine; E = epinephrine; D = dopamine; AH = acute hypertension secondary to bilateral cervical vagotomy and occlusion of both common carotid arteries. Other symbols as in Table 1.
[b] $P < 0.05$ (unpaired $t$ test).
[c] $P < 0.001$ (unpaired $t$ test).

These results indicate that changes of pressure in the first order peritubular capillaries of superficial nephrons are merely secondary to changes in glomerular capillary pressure. This does not mean that resistance across the efferent arterioles of superficial nephrons is constant. Resistance is given by the ratio between the pressure gradient (GCP−EAP) and the blood flow across the vessel. Changes in this ratio are not necessarily dependent on variations in the muscular activity of the arteriolar wall. At low perfusion pressure, in fact, the blood behaves as a non-Newtonian fluid; its viscous resistance to flow increases

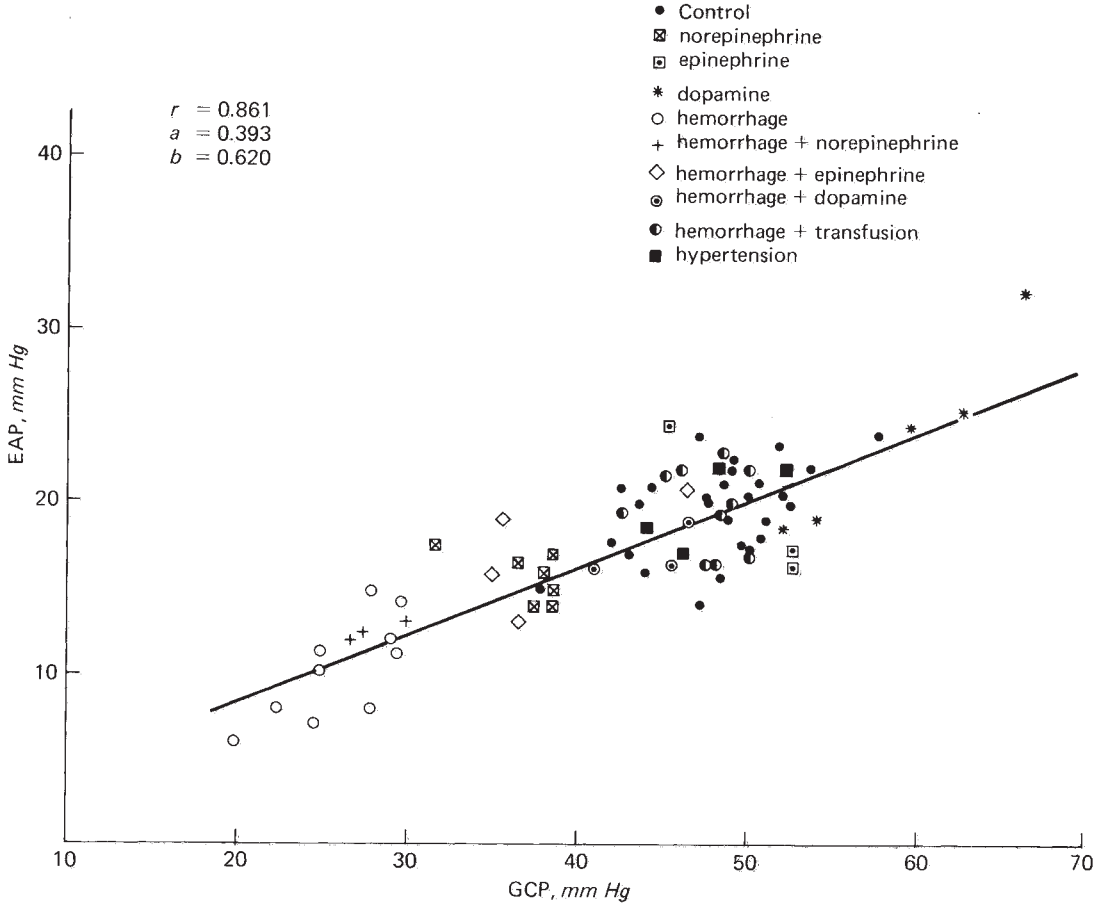


**Fig. 1.** *Relationship between GCP and EAP in all conditions under study.*

with decreasing perfusion pressure; hence at low GCP, resistance to blood flow across efferent arterioles may be higher than normal (i.e., than the resistance at normal GCP) because of the viscous properties of blood. Furthermore, the elastic tension of the arteriolar wall does not increase linearly with intraluminal pressure because of the greater stretching of collagen fibers with respect to the elastic ones. Both of these phenomena will affect resistance of efferent arterioles despite constancy of the GCP/EAP ratio.

The present study does not rule out the possibility that changes in vascular resistance beyond the early peritubular capillaries are also contributing to the absolute values for EAP. This appears, however, to be unfeasible in those conditions (e.g., norepinephrine infusion) in which only an opposite effect beyond the first order peritubular capillaries in comparison to the effect on afferent arterioles could account for the changes observed in EAP.

Our results fit well with the mathematical model elaborated by Deen, Robertson and Brenner [19]. According to these authors the shape of the EFP curve changes at different values of $F = Kf \cdot \Delta P / Q_0$ (where $Kf$ is the ultrafiltration coefficient, $\Delta P$ the glomerular transcapillary pressure and $Q_0$ the initial glomerular plasma flow). Thus, a fall in $F$ values (secondary to rise in $Q_0$ and/or fall in GCP) will shift the point of filtration pressure equilibrium towards and even beyond the efferent arteriole. In the latter condition (i.e., with the filtration pressure balance point beyond the efferent arteriole), any increase or decrease in GPF will be followed by minor changes in the rate of glomerular ultrafiltration, thus accounting for the change in SNFF without the need of an "active" role of efferent arterioles.

What we have shown in superficial nephrons may not be valid for deep and midcortical nephrons. Anatomical as well as functional differences have, in fact, been found among nephrons in relation to their depth in the cortex [20], which may account for the discrepancy between SNFF in superficial nephrons and whole kidney filtration fraction reported in the recent literature (see [1]).

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