Dynamics of glomerular ultrafiltration in the rat. II. Plasma-flow dependence of GFR

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BRENNER, B. M., J. L. TROY, T. M. DAUGHARTY, W. M. DEEN, and C. R. ROBERTSON. Dynamics of glomerular ultrafiltration in the rat. II. Plasma-flow dependence of GFR. Am. J. Physiol. 223(3): 1184–1190. 1972.—Using servo nulling transducers and microanalytical protein assay methods in rats with surface glomeruli, we measured glomerular capillary and proximal tubule hydrostatic pressures and estimated afferent and efferent arteriolar oncotic pressures during conditions deliberately designed to increase GFR (acute expansion of plasma and extracellular volume). Single-nephron (SN) GFR and initial glomerular capillary plasma flow (GPF) both increased approximately twofold. Net ultrafiltration pressure at the afferentmost point in the glomerulus was essentially the same prior to and following volume expansion. Similarly, filtration pressure equilibrium was achieved both prior to and following expansion. The roughly proportional changes in SNGFR and GPF provide evidence that glomerular ultrafiltration is a plasma flow dependent process. Under these conditions (filtration pressure equilibrium), SNGFR is determined entirely by GPF, transcapillary hydrostatic pressure difference, and initial capillary protein concentration. The possible physical mechanisms underlying the plasma-flow dependence of SNGFR have been examined using a recently developed model.

glomerular filtration rate; glomerular capillary pressure; intrarenal vascular resistances; intrarenal plasma flow; micropuncture methods; colloid osmotic pressure; Bowman’s space; afferent arteriole; efferent arteriole

THE DEVELOPMENT of highly sensitive servo-nulling micropipette transducer techniques (5, 9, 20) and reliable methods for estimating colloid osmotic pressure (σ) in pre- and postglomerular plasma (2, 6), together with the discovery of a mutant strain of Wistar rats which possess glomeruli as surface structures, have recently made possible direct measurements of several of the forces governing ultrafiltration in a mammalian kidney (4). In addition to serving as a frame of reference for comparing the effects of a variety of maneuvers aimed at experimentally altering these glomerular transcapillary forces, and hence glomerular filtration rate (GFR), these studies have provided the stimulus for the development of a mathematical model capable of predicting the profile of variations in net ultrafiltration pressure with distance along the glomerular capillary (8). We report here a series of measurements related to the dynamics of glomerular ultrafiltration during one such experimental maneuver, that of deliberately elevating GFR by means of acute expansion of plasma and extracellular volume. The results of these measurements provide evidence to indicate that glomerular ultrafiltration is a plasma-flow-dependent process. Using these measurements and the relationships derived from the mathematical model (8), it has been possible to examine the relative influences of several factors potentially responsible for this phenomenon of flow dependence.

METHODS

Experiments were performed in 19 normally hydropenic adult Munich-Wistar rats weighing 202–343 g and allowed free access to food and water prior to study. Rats were anesthetized with Inactin (100 mg/kg) and prepared for micropuncture as described previously (2, 3, 5).

Glomerular Dynamics During Normal Hydropenia

Beginning 60 min prior to study, each rat received an intravenous infusion of isotonic NaCl at the rate of 0.02 ml/min. Inulin was present in a concentration of 10% whereby resulting in final plasma concentrations of about 100 mg/100 ml. Mean femoral arterial pressure (MAP) was monitored by means of a transducer (model P23AA, Statham Instruments, Los Angeles, Calif.) connected to a direct-writing recorder (model 7702B, Hewlett-Packard Co., Palo Alto, Calif.). Late surface convolutions of proximal tubules were located by observing the passage of lissamine green dye which was injected rapidly (0.05 ml of a 5% solution) into the right jugular vein catheter. Following this 60-min equilibration period, exactly timed (1–2 min) samples of fluid were collected from each experimental tubule for determination of flow rate and inulin concentration, and calculation of single-nephron (SN) GFR. The rate of fluid collection was adjusted to maintain a column of polymer oil (Kcl F polymer oil, Minnesota Mining and Manufacturing Co., St. Paul, Minn.) three to four tubule diameters in length, in a relatively constant position just distal to the site of puncture. Using the collection technique of controlled suction recently validated for this laboratory (1), minimal
PLASMA-FLOW DEPENDENCE OF GFR

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changes in tubule diameter or the position of the distal oil block were produced. Coincident with these tubule fluid collections, femoral arterial blood samples were obtained for determinations of hematocrit and plasma insulin concentration.

Hydrostatic pressure measurements were obtained in glomerular capillaries using continuous-recording, servo-nulling micropipette transducer techniques (5, 9, 20). Micropipettes with outer-tip diameters of 2–3 μ and containing 1.5 M NaCl were used. Penetration of Bowman’s capsule and entry into single glomerular capillaries was performed under stereomicroscopic control. Hydraulic output from the servo system was channeled via a transducer (Statham, P23Db) to a second channel of the recorder. Accuracy, frequency response, and stability features of this servo system have been described in detail elsewhere (5). In addition to direct measurements of glomerular capillary hydrostatic pressure2 (ΦH), and proximal tubule pressure (ΦP), pressures also were recorded in Bowman’s space (Φb), efferent arterioles (ΦAA), and second- and third-order branch peritubular capillaries (ΦC) in each rat.

To obtain estimates of oncotic pressure of plasma entering and leaving glomerular capillaries, protein concentrations in femoral arterial and efferent arteriolar blood plasmas were measured as described recently (2). Colloid osmotic pressures were calculated from the equation for plasma derived by Landis and Pappenheimer (15) and recently validated for the rat (6). π calculated for femoral arterial plasma will be taken as representative for the afferent arteriole (πAA). These estimates of pre- and postglomerular protein concentration permit calculation of single-nephron filtration fraction (SNFF) and initial glomerular capillary plasma flow (GPF) (see equations below).

Since direct measurements of the segmental pressure differentials across single afferent and efferent arterioles were obtained together with estimates of local rates of blood flow through these vessels, this study also provides heretofore unavailable direct estimates of resistances across these pre- and postglomerular channels in the renal cortex of the rat.

Glomerular Dynamics After Elevations in GFR and GPF

After measurements in hydropenia, 11 rats received an intravenous infusion of fresh homologous rat plasma at the rate of 0.1 ml/min until a total volume equal to 2.5% of body weight had been given. In eight other rats, volume expansion was achieved by intravenous infusion of isosmotic bicarbonate-Ringer solution, in millimoles per liter: NaCl 115, KCl 5, NaHCO3 25, Na acetate 10, Na2HPO4 1.2, MgSO4 1.2, CaCl2 2.5) injected at the rate of 0.375 ml/min until an amount equal to 10% of body weight had been given. On completion of these infusions, all rats were given maintenance bicarbonate-Ringer infusions at the rate of .075 ml/min. Inulin was added in amounts adequate to maintain plasma concentrations of approximately 100 mg/100 ml. After the onset of these maintenance infusions, measurements of each of the variables studied in the pre-expansion period were repeated. Separate, rather than the same proximal tubules, glomeruli, efferent arterioles, and peritubular capillaries were punctured following volume expansion.

Analytical Determinations

The volume of tubule fluid collected from individual nephrons was estimated from the length of the fluid column in a constant-bore capillary tube of known internal diameter. The concentration of inulin in tubule fluid was measured, usually in duplicate, by the microfluorescence method of Vurek and Pegram (19). Inulin concentration in plasma was determined by the macro-anthrone method of Führ, Kaczmarkcy, and Krütgen (10). Protein concentrations in efferent arteriolar and femoral arterial blood plasmas were determined, usually in duplicate, with an Amicon ultramicrocolorimeter (American Instrument Co., Silver Spring, Md.) using a recently described (2) microadaptation of the technique of Löwy et al. (17).

Calculations

Single-nephron glomerular filtration rate:

\[ \text{SNGFR} = \frac{(\text{TF}/\text{P})_{\text{In}} \cdot V_{\text{TF}}}{\Pi_{\text{TF}}} \]  

where \((\text{TF}/\text{P})_{\text{In}}\) and \(V_{\text{TF}}\) refer to transtubular inulin concentration ratio and tubule fluid flow rate, respectively. Flow rates in this and subsequent equations are given in units of nanoliters per minute.

Single-nephron filtration fraction:

\[ \text{SNFF} = 1 - \frac{[\text{protein}]_{\text{AA}}}{[\text{protein}]_{\text{EA}}} \]  

where AA and EA denote afferent and efferent arteriole, respectively.

Initial glomerular capillary plasma flow:

\[ \text{GPF} = \frac{\text{SNGFR}}{\text{SNFF}} \]  

Initial glomerular capillary blood flow:

\[ \text{GBF} = \frac{1}{1 - \text{Hct}_{\text{AA}}} \]  

Plasma flow per efferent arteriole:

\[ \text{EAPF} = \text{GPF} - \text{SNGFR} \]  

Blood flow per efferent arteriole:

\[ \text{EABF} = \text{GBF} - \text{SNGFR} \]  

RESULTS

Glomerular Dynamics During Normal Hydropenia

Table 1 summarizes average values for the various determinants of glomerular transcapillary exchange during normal hydropenia. SNGFR averaged 20.5 nl/min. Individual values for each rat are shown in Fig. 1. SNFF, and

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2 Values for ΦP as given in the present study represent time averages. As reported previously (4), peak-to-valley amplitudes of single glomerular capillary pressure pulses average approximately 8 mm Hg and generally bracket these time-averaged values equally during systole and diastole. The term (ΡΦ) represents ΦΦ averaged over the length of the glomerular capillary, the justification for which is discussed below. Values for ΦP are very close to zero and have therefore been neglected.
TABLE 1. *A summary of some measured determinants of glomerular ultrafiltration*

<table>
<thead>
<tr>
<th>Condition</th>
<th>(AP)</th>
<th>(PGo)</th>
<th>PT</th>
<th>Pc</th>
<th>Protein</th>
<th>πAA</th>
<th>πEA</th>
<th>(πGo - PT)</th>
<th>SNFGR</th>
<th>GPF</th>
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<tbody>
<tr>
<td>Normal hydropenia</td>
<td>109</td>
<td>45.1</td>
<td>9.8</td>
<td>6.2</td>
<td>5.8</td>
<td>8.7</td>
<td>19.4</td>
<td>36.7</td>
<td>1.04</td>
<td>20.5</td>
</tr>
<tr>
<td></td>
<td>±2.6</td>
<td>±0.8</td>
<td>±0.2</td>
<td>±0.2</td>
<td>±0.1</td>
<td>±0.2</td>
<td>±0.5</td>
<td>±1.1</td>
<td>±0.03</td>
<td>±1.3</td>
</tr>
<tr>
<td>Plasma loading</td>
<td>99</td>
<td>46.9</td>
<td>11.5</td>
<td>10.8</td>
<td>6.0</td>
<td>8.9</td>
<td>20.2</td>
<td>36.3</td>
<td>1.02</td>
<td>39.5</td>
</tr>
<tr>
<td></td>
<td>±4.6</td>
<td>±1.3</td>
<td>±0.5</td>
<td>±0.6</td>
<td>±0.1</td>
<td>±0.2</td>
<td>±0.5</td>
<td>±1.4</td>
<td>±0.02</td>
<td>±3.0</td>
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<tr>
<td></td>
<td>(11)</td>
<td>(11)</td>
<td>(11)</td>
<td>(11)</td>
<td>(10)</td>
<td>(10)</td>
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<td>(10)</td>
</tr>
<tr>
<td>P value</td>
<td>&gt;.4</td>
<td>&lt;.05</td>
<td>&lt;.025</td>
<td>&lt;.001</td>
<td>&gt;.5</td>
<td>&gt;.2</td>
<td>&gt;.5</td>
<td>&gt;.5</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
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<tr>
<td>Ringer loading</td>
<td>105</td>
<td>38.3</td>
<td>11.1</td>
<td>7.2</td>
<td>4.6</td>
<td>7.2</td>
<td>13.9</td>
<td>26.7</td>
<td>1.00</td>
<td>39.5</td>
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<td></td>
<td>±3.2</td>
<td>±2.6</td>
<td>±0.7</td>
<td>±1.0</td>
<td>±0.2</td>
<td>±0.3</td>
<td>±0.9</td>
<td>±1.6</td>
<td>±0.05</td>
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<tr>
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<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&gt;.5</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

Values are means ± 1 se. Number in parentheses denote number of animals. P values calculated from paired data in each rat using the Student t test.

The mean values for femoral arterial and efferent arteriolar protein concentration from which SNFF was calculated, were similar to values reported previously (2-4). GPF, therefore, averaged 65.0 nl/min.4 Individual values for each rat are also given in Fig. 1. (PGo) measured in 21 glomeruli of 19 rats during normal hydropenia, as shown in Fig. 2 (abscessa), ranged from 39 to 52 mm Hg. The average value of 45.1 mm Hg corresponds nearly exactly to that reported previously (4). The ratio (PGo)/(AP) averaged 0.42 ± 0.01 se, indicating that nearly 60% of the systemic mean arterial pressure is dissipated prior to the glomerular capillary. Values for PT (Table 1) were similar to pressure measured directly in Bowman's space (9.4 mm Hg ± 0.2 (n = 13)). The pressure difference of some 3.6 mm Hg recently demonstrated (5, 9) between PT and Pc in normal hydropenia was again observed (Table 1). Surface efferent arterioles, like glomerular capillaries, uniformly exhibited pulsatile pressures and averaged 11.0 mm Hg ± 0.6. πEA and πAA (Table 1) were typical of values previously reported (4) for normal hydropenia. The effective ultrafiltration pressure (PUF) at the afferentmost point in the glomerular capillary (PUF = (PGc) - Pfl - πAA)4 averaged 16.2 mm Hg ± 0.9. In accord with previous observations, during normal hydropenia the ratio πAA/(PGo - PT) was close to unity (Table 1, Fig. 3, closed circles), indicating that ultrafiltration pressure approaches zero and filtration pressure equilibrium obtains by the terminal end of the glomerular capillary network.

Glomerular Dynamics After Elevations in GFR and GPF

Isoncotic expansion. Expansion with homologous plasma in 11 rats (2.5% body wt) resulted in a uniform fall in systemic hematocrit, on average from 52.4 to 37.5 vol%. SNGFR increased nearly twofold in each rat (Table 1 and Fig. 1, 4). The use of this equation assumes the axial variation in PGo to be zero.
Since SNFF changed little, if any, from control, GPF likewise increased approximately twofold, thereby maintaining a roughly constant relationship to SNGFR. This is illustrated by the tendency in Fig. 1 for each slope to parallel the hypothetical line which describes a constant filtration fraction of 0.33. As seen in Table 1 these changes in SNGFR and GPF were accompanied by small and insignificant changes in (AP), pre- and postglomerular protein concentrations, and oncotic pressures. (PGC) tended to increase only slightly, if at all (Fig. 2, solid circles); paired differences from hydropenia to plasma loading averaged +1.8 mm Hg ± 0.7 (P < .05). Thus, P_{T/R} exhibited little overall change from control, averaging 13.5 mm Hg ± 1.4. P_T, P_{EA} (Table 1), and P_{EA} (mean = 14.3 mm Hg ± 0.9) increased significantly (for all, P < .025). These increments in hydrostatic pressure, together with this tendency toward abolition of the normal proximal tubule-peritubular capillary pressure difference (Table 1), have previously been observed following plasma expansion. Line of identity is indicated. 

(Fig. 3) Comparison of values for (P_{EA}) and (P_{GOC} - P_T) obtained in each rat during hydropenia (solid circles) and after plasma (half-filled circles) and Ringer (open circles) loading. Filtration pressure equilibrium is given by line of identity.

In accord with recent observations from this laboratory (4), the present study demonstrates that the formation of glomerular ultrafiltrate occurs coincident with a progressively diminishing imbalance of pressures across the glomerular capillary wall. During normal hydropenia this imbalance averages approximately 16 mm Hg at the proximal portion of the glomerular capillary network and approaches zero by the end. Of interest is the fact that this progressive diminution in P_{T/R} results primarily from the rise in π plasma, reflecting thereby the protein-free composition of the ultrafiltrate. That P_{GOC} remains relatively constant throughout the length of the glomerular capillary is suggested by the finding that the sum of the pressures opposing filtration in the distalmost portions of the glomerular capillary (as inferred from the sum of π_{EA} + P_T) reach a value which, on average, balances P_{GOC}. Since P_{GOC} is measured at sites along the glomerular capillary which are presumed to be statistically random, the finding that π_{EA} + P_T ≈ P_{GOC} makes it likely that P_{GOC} changes little along capillary segments at this level of the renal vascular circuit. This situation for the glomerular capillary is to be contrasted with that for peripheral capillaries (12-14, 16,
with the observation of filtration pressure equilibrium, the equation progresses decline in capillary hydrostatic pressure (from a level slightly in excess of, to a level slightly below, \( \pi \) plasma).

The experimental aim of the present study was to examine glomerular dynamics under conditions of enhanced rates of ultrafiltration. The fundamental requirement of a rise in SNGFR was achieved in each rat by means of acute expansion of plasma and extracellular volume. Following volume expansion, GPF approximately doubled. Despite these marked increments in GPF, filtration pressure equilibrium persisted. Under these conditions (that is, filtration pressure glomerular dynamics under conditions of enhanced rates of ultrafiltration. The fundamental requirement of a rise in SNGFR, for any given (\( \Delta P \)) and [protein]_{AA}. Since SNFF remained constant it follows, using the rearranged form of equation 3:

\[
\text{SNGFR} = \text{GPF} \cdot \text{SNFF}
\]

that SNGFR will increase in proportion to the increase in GPF.

Since equation 7 is only a statement of mass conservation, it cannot yield insight into the mechanisms whereby changes in GPF bring about changes in SNGFR. In order to relate these changes in SNGFR to the responsible transcapillary driving forces, SNGFR may be expressed as the product of the mean driving pressure for ultrafiltration (\( \langle P_{UF} \rangle \)) and an ultrafiltration coefficient (\( K_f \)):

\[
\text{SNGFR} = K_f \langle P_{UF} \rangle
\]

where \( K_f \) is the product of the effective hydraulic permeability (\( k \)) of the capillary membrane and the surface area for filtration (\( S \)). Accordingly, the dependence of SNGFR on GPF might be mediated by changes in \( k, S, \langle P_{UF} \rangle \), or some combination thereof, so long as the resultant values for these quantities are sufficient to still yield filtration pressure equilibrium. The data obtained in the present study do not allow the accurate estimation of these three quantities because at filtration pressure equilibrium, only their product can be determined (8). Consequently, the extent, if any, to which these factors contribute to this flow-dependent phenomenon cannot be directly assessed from these data.

Using a recently developed mathematical model of glomerular ultrafiltration, we analyzed the means by which changes in plasma flow might influence these quantities to bring about the observed changes in SNGFR. In applying this model, assume first that \( k \) and \( S \) are unchanged by volume expansion. It is then possible to ascertain whether the predicted effect of GPF on \( \langle P_{UF} \rangle \) is sufficient to account for the observed changes in SNGFR. The use of the model to compute \( P_{UF} \) as a function of distance along a glomerular capillary requires the specification of four parameters: \( A_1 \) and \( A_2 \), the coefficients in the osmotic pressure equation; \( \epsilon \), the nondimensional axial pressure drop; and \( F \), the permeability-plasma flow rate parameter, expressed in the present notation as:

\[
F = \frac{K_f (P_{OC} - P_T)}{GPF}
\]

Employing a value of \( K_f \) (0.10 nl/(sec \cdot mm Hg)) consistent with the observation of filtration pressure equilibrium, the model was used to compute [protein]_{AA} and hence SNFF and SNGFR from measured values of [protein]_{AA}, \( P_{OC} \), \( P_T \), and GPF given in Table 1. Using this value of \( K_f \), \( F = 3.26 \) in normal hydropenia, \( F = 1.62 \) for plasma loading, and \( F = 1.47 \) for Ringer loading. Further, assume that \( \epsilon = 0.030 \) for normal hydropenia. Since the glomerulus may be viewed as a branched network of tubes in which the rate of fluid loss varies as a nonlinear function of distance along the tubes, the dependence of the axial pressure drop (\( \epsilon \)) in the glomerulus on GPF is not immediately apparent. We will therefore consider two extreme cases: a) the axial pressure drop is independent of GPF (\( \epsilon = 0.030 \) for plasma and Ringer loading as well as for normal hydropenia), and b) the pressure drop is proportional to GPF (\( \epsilon = 0.060 \) for plasma loading and \( \epsilon = 0.051 \) for Ringer). Values of \( A_1 \) and \( A_2 \) for the normal and volume-expanded rat are readily determined (8) from the available data.

Calculation of glomerular hydrostatic and osmotic pressure profiles for the conditions specified above leads to the results shown in Table 2. Agreement between predicted and observed values of SNGFR is excellent, the mean difference being 4%. As shown, the effect of GPF on \( \langle P_{UF} \rangle \) can account for essentially all of the observed plasma-flow dependence of SNGFR. Note that these results are relatively insensitive to the specific assumption made about the axial pressure drop, \( a \) or \( b \). It should also be emphasized that these increases in \( \langle P_{UF} \rangle \) occur without concomitant increases in P_{UF,AA}.

Increases in \( K_f \) associated with elevations in GPF might also account for the plasma-flow dependence of SNGFR. Assuming now that \( \langle P_{UF} \rangle \) is not altered following volume expansion, the model may be used to estimate the change in \( K_f \) required to produce the observed degree of flow dependence. It can be shown (8) that predicted values of \( \langle P_{UF} \rangle \) are relatively insensitive to the differences in \( A_1 \),

<table>
<thead>
<tr>
<th>Condition</th>
<th>SNFF (Predicted)</th>
<th>SNFF (Observed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal hydropenia</td>
<td>( \epsilon = 0.030 )</td>
<td>20.5 ( \pm 1.3 )</td>
</tr>
<tr>
<td>Plasma loading</td>
<td>( A) \ \epsilon = 0.030 )</td>
<td>36.8</td>
</tr>
<tr>
<td></td>
<td>( B) \ \epsilon = 0.050 )</td>
<td>36.4</td>
</tr>
<tr>
<td>Ringer loading</td>
<td>( A) \ \epsilon = 0.030 )</td>
<td>39.5 ( \pm 2.5 )</td>
</tr>
<tr>
<td></td>
<td>( B) \ \epsilon = 0.051 )</td>
<td>37.0</td>
</tr>
</tbody>
</table>

- Observed SNGFR values are means \( \pm SE \). Predicted refers to values calculated using the model, assuming \( K_f = 0.100 \) nl/(sec \cdot mm Hg). Observed refers to the mean values obtained in the present study.
- \( A_1 = 0.268, A_2 = 0.280, F = 3.26 \).
- \( A_1 = 0.276, A_2 = 0.299, F = 1.62 \).
EFFERENT RESISTANCE

Therefore, if $F$ is constant, $(PUF)$ will remain approximately unchanged. Inspection of equation 9 and Table 1 indicates that $F$ will remain constant provided that $K_f$ approximately doubles as a result of volume expansion. Thus, the observed increases in $S$ are then not required.

APPENDIX

To date, the calculation of peripheral vascular resistance in single afferent and efferent arterioles based on direct measurements of $\Delta P$ and flow through these vessels has not been accomplished. Since mean pressures were recorded in a systemic artery, and in glomerular and peritubular capillaries, the segmental resistances for single afferent and efferent arterioles could be calculated, using the following equations:

$$R_A = \frac{\langle AP \rangle}{\langle P_G \rangle} \times 1,327 \quad (10)$$

where $\langle AP \rangle$ (mean arterial pressure) and $\langle P_G \rangle$ are in units of millimeters Hg, and GBF is in units of cubic centimeters per second (cm$^3$ sec$^{-1}$). The factor 1,327 is obtained by multiplying the weight of 0.1 ml baryes (dynes cm$^{-2}$). This, when divided by flow (cm$^3$ sec$^{-1}$), yields a value for resistance in units of dyne cm$^{-2}$/sec. These units for pressure, flow, and resistance apply in succeeding equations as well.

Resistance per single efferent arteriole:

$$R_E = \frac{(P_{GO} - P)}{EABF} \times 1,327 \quad (11)$$

Total arteriolar resistance for a single series-linked vascular unit:

$$R_{TA} = R_A + R_E \quad (12)$$

Table 3 summarizes average blood flows for single surface afferent and efferent arterioles. Absolute values for afferent and efferent resistance together with values for total arteriolar resistance (the sum of these series-linked resistances) are also given. During hydropenia, slightly more than half the total arteriolar resistance is contributed by the afferent arteriole. Plasma expansion resulted in uniform increases

Table 3. Summary of blood flows and resistances for single afferent and efferent arterioles

<table>
<thead>
<tr>
<th>Condition</th>
<th>GBF</th>
<th>EABF</th>
<th>$R_A$</th>
<th>$R_E$</th>
<th>$R_{TA}$</th>
<th>$R_A$</th>
<th>$R_E$</th>
<th>$R_{TA}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>134.0</td>
<td>113.5</td>
<td>4.34</td>
<td>3.96</td>
<td>7.36</td>
<td>0.57</td>
<td>0.43</td>
<td></td>
</tr>
<tr>
<td>hydropenia</td>
<td>±12.1</td>
<td>±10.9</td>
<td>±0.46</td>
<td>±0.37</td>
<td>±0.01</td>
<td>±0.02</td>
<td>±0.02</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± 1 se. Numbers in parentheses denote number of animals. $P$ values calculated from paired data in each rat using the Student $t$ test.
in GBF and EABF averaging 54.8 nl/min ± 12.0 (P < .005) and 38.1 ± 10.0 (P < .005), respectively. RA and RE each decreased in nine of ten rats (Figs. 4, left, and 5, left), the paired changes for all rats of −1.03 and −0.71 X 10^9 dynes-sec-cm^{-2} being significant at the .01 and .025 levels, respectively. Accordingly, RTA fell in these same 9 of 10 rats, with paired changes for all rats averaging −1.73 X 10^9 dynes-sec-cm^{-2} (P < .01). Note in Figs. 4 (left) and 5 (left) that although the absolute magnitude of the changes in RA, RB, and RTA tended to vary from rat to rat, the slopes of the lines from rat to rat were relatively uniform. Hence, the ratios RA/RTA and RE/RTA were not changed significantly by plasma loading (Table 3).

Ringer infusion resulted in uniform elevations in GBF and EABF (Table 3), the increments averaging 91.9 nl/min ± 22.2 (P < .005) and 69.6 ± 18.9 (P < .01), respectively. Ringer resulted in uniform and often large reductions in RA, RB, and RTA (Table 3 and Figs. 4, right, and 5, right) averaging 5.0, 2.5, and 5.60 X 10^9 dynes-sec-cm^{-2}, respectively. Again the slopes of the lines in each figure cluster in close and parallel fashion. Of interest was the finding that RA/RTA decreased (and therefore RE/RTA increased) in 8 of 10 rats following right, nine of ten rats (Figs. 4, left, and 5, left), the paired changes for all rats in GBF and EABF averaging 54.8 nl/min ± 12.0 (P < .005) and .01 and .025 levels, respectively. Accordingly, RTA fell in these same 9 rats contributed to the observed fall in (Pot) following expansion with plasma loading (Table 3). This latter difference very likely also contributes to the observed fall in (Pot) following expansion with Ringer but not with plasma (vide supra).

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