Dynamics of Glomerular Ultrafiltration in the Rat

VIII. EFFECTS OF HEMATOCRIT

By Bryan D. Myers, William M. Deen, Channing R. Robertson, and Barry M. Brenner

ABSTRACT

This study was undertaken in an effort to examine the effects of selective variations in systemic hematocrit on the preglomerular, glomerular, and postglomerular microcirculation in the rat. By isovolemic exchange transfusions, systemic hematocrit (control 51 ml/100 ml) was either reduced (21 ml/100 ml, N = 7 rats) or elevated (62 ml/100 ml, N = 7). Single nephron glomerular filtration rate varied inversely and filtration fraction varied directly with the changes in hematocrit. The fall in filtration fraction with decreased hematocrit was due to a decline in the measured glomerular transcapillary hydraulic pressure difference (ΔP) and to a marked increase in the initial glomerular plasma flow rate. Afferent (R_a) and efferent (R_e) arteriolar resistances declined with the fall in hematocrit; R_a fell proportionately more than did R_e. The rise in filtration fraction with the elevation in hematocrit was due to a marked increase in ΔP in part due to a relatively greater rise in R_e than in R_a. These findings provide an attractive explanation for the general tendency for filtration fraction to vary directly with hematocrit in anemic and polycythemic states in man.

KEY WORDS

viscosity
anemia
vascular resistance
filtration fraction
polycythemia
afferent arteriole
efferent arteriole
glomerular filtration

It has long been recognized that the resistance to the flow of blood through the kidney is offered almost totally by the combined resistances of the afferent and efferent muscular arterioles (1–9). Quantitative measurements in both the rat and the monkey indicate that, during normal hydropenic conditions, afferent arterioles contribute roughly two-thirds of the total arteriolar resistance (9–12). Moreover, of the roughly 100-mm Hg hydraulic pressure drop that normally occurs from the renal artery to the renal vein, some two-thirds of this axial pressure drop takes place along afferent arterioles; the bulk of the remaining pressure drop occurs along efferent arterioles. Indeed, only a very small (probably less than 3 mm Hg) pressure drop occurs along the glomerular capillary network (10).

Recent studies in this laboratory have been concerned with a systematic assessment of the contributions of these arteriolar resistances to the intrarenal control of blood flow and glomerular filtration rate. Essential to such an analysis is the need to examine the influences on arteriolar resistance resulting from alterations in effective blood viscosity. The effects of variations in blood hematocrit, a major determinant of blood viscosity, are the subject of the present report.

Methods

LIST OF SYMBOLS

AP = Mean femoral arterial blood pressure (mm Hg).
C = Protein concentration (g/100 ml).
EABF = Efferent arteriolar blood flow (nliters/min).
GBF, GPF = Glomerular blood flow and plasma flow, respectively (nliters/min).
GFR = Glomerular filtration rate (whole kidney).
Hct_a = Blood hematocrit in the femoral artery or afferent arteriole.
k = Effective hydraulic permeability (nliters/sec mm Hg cm²).
K_t = Ultrafiltration coefficient (nliters/sec mm Hg) or nliters/(min mm Hg).
P = Hydraulic pressure (mm Hg).
P_{uf} = Net ultrafiltration pressure (mm Hg).
ΔP = Transmembrane hydraulic pressure difference, P_{oc} – P_f (mm Hg).
R = Resistance to blood flow (dynes/sec-cm⁻²).
R_{ra} = Total arteriolar resistance, R_a + R_e (dynes/sec-cm⁻²).
GENERAL PROCEDURES

Studies were performed on 14 adult Wistar rats (202-290 g) that were allowed free access to a rat pellet diet and water. The rats were anesthetized with Inactin (100 mg/kg, ip), placed on a temperature-regulated micropuncture table, and subjected to a tracheostomy. Indwelling polyethylene catheters were inserted into the left jugular vein for infusion of saline, into the right femoral vein for infusion of blood or plasma, and into the left femoral artery for withdrawal of blood during isovolemic exchange, periodic collections of blood samples, and periodic estimations of mean arterial blood pressure (MAP). MAP was monitored by an electronic transducer (Statham Instruments, model P23Db) connected to a chart recorder (Hewlett-Packard, model 7702B). The left kidney was exposed by a left subcostal incision separated from the adrenal gland and the surrounding perirenal fat. The kidney was suspended on a Lucite holder, and its surface was illuminated with a fiber-optic light source and bathed with isotonic NaCl heated to 37°C. Excessive fluid losses during preparative surgery were painstakingly avoided; the small losses that were incurred were not replaced.

Sixty minutes before micropuncture, the rats received an intravenous infusion of isotonic NaCl at the rate of 0.02 ml/min. Inulin was present in a concentration of 10%, thereby resulting in final plasma concentrations of about 100 mg/100 ml. After this 60-minute equilibration period, exactly timed (1-2 minutes) samples of fluid were collected at random sites from a surface proximal convolution from each of two nephrons for determination of flow rate and inulin concentration and calculation of SNGFR. The rate of fluid collection was adjusted to maintain a column of polymer oil (3M Co, Kel F polymer oil) 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 validated for this laboratory (13), minimum changes in tubule diameter and in the position of the distal oil block were produced. Coincident with these tubule fluid collections, femoral arterial blood samples, 100 µl in volume, were obtained for determination of hematocrit and plasma inulin concentration.

Hydraulic pressures were measured in single capillaries within surface glomeruli using a continuous-recording, servomunich micropipette transducer system (14-16). Micropipettes with outer tip diameters of 2-3 µm and containing 2.0M NaCl were used. Penetration of Bowman’s capsule and entry into glomerular capillaries were performed under stereomicroscopic control. The hydraulic output from the servosystem was channeled via an electronic transducer (Statham Instruments, model P23Db) to a second channel of the recorder. Accuracy, frequency response, and stability features of this servosystem have been reported previously (16).

Direct measurements of hydraulic pressure in single glomerular capillaries (P<sub>GC</sub>), proximal tubules (P<sub>T</sub>), efferent arterioles (P<sub>E</sub>), and third-order peritubular capillaries (P<sub>C</sub>) were recorded in each rat.

To obtain estimates of colloid osmotic pressure (π) of plasma entering and leaving glomerular capillaries, protein concentrations (C) in femoral arterial (d) and efferent arteriolar (Ce) blood plasma were measured as described previously (17). Protein concentrations (C) in femoral arterial (d) and efferent arteriolar (Ce) blood plasma were measured as described previously (17). Colloid osmotic pressures were calculated from these measured values of C using the equation

\[ \pi = 1.63C + 0.294C^2. \]

This equation has been shown (18) to agree to within 1% with the empirical equation derived by Landis and Pappenheimer (19). Eq. 1 assumes an albumin-globulin ratio (A/G) of unity, the value found in normal hydropenic rats in this laboratory. These estimates of pre- and postglomerular protein concentration permitted calculation of single nephron filtration fraction (SNFF) and GPF (see Eqs. 3 and 4). From direct measurements of the decline in hydraulic pressure along single afferent and efferent arterioles and estimates of blood flow through these vessels, vascular resistances to blood flow through these individual vessels were calculated (see Eqs. 7-9).

EFFECT OF REDUCTION OF ARTERIAL HEMATOCRIT

Following the measurements just described in normal hydropenic, we examined the effects of a reduction in arterial hematocrit on glomerular capillary pressures and flows and on resistances to blood flow along single afferent and efferent arterioles in seven rats. The reduction in hematocrit was achieved by isovolemic exchange transfusion. A volume of homologous rat plasma (obtained by exsanguination of a littermate on the morning of the study) equal to 4% of body weight was infused intravenously. Simultaneously, an equal volume of whole blood was withdrawn at the same rate from the femoral arterial catheter. The total duration of the isovolemic exchange was 15-20 minutes. Inulin was added to the infused plasma to yield a final concentration of approximately 100 mg/100 ml. Following a 15-minute equilibration period, collections of tubule fluid and efferent arteriolar and femoral arterial blood and measurements of MAP, P<sub>GC</sub>, P<sub>T</sub>, P<sub>E</sub>, and P<sub>C</sub> were repeated in each rat.

<sup>1</sup>Values for P<sub>GC</sub> used in the present study represent time averages. Peak-to-valley amplitudes of single glomerular capillary pressure pulses averaged approximately 10 mm Hg and generally bracketed these time-averaged values equally during systole and diastole. The term P<sub>GC</sub> represents P<sub>GC</sub> averaged over the length of the glomerular capillary; the justification for this assumption has been discussed previously (10).
EFFECTS OF HEMATOCRIT ON GLOMERULAR FILTRATION

In the remaining group of seven rats, following initial measurements and fluid collections in normal hydropenia, each rat underwent an isovolemic exchange transfusion to effect an elevation of its arterial hematocrit. In these rats, a volume of high-hematocrit blood (75-80 ml/100 ml) equal to 4% of body weight was infused intravenously (in a 15-20-minute period) in exchange for an identical volume of whole blood withdrawn simultaneously from the femoral arterial catheter. The infused blood, collected on the morning of study into lightly heparinized polyethylene syringes, was centrifuged at 3,500 rpm for 20 minutes, and the red blood cells were resuspended in appropriate volumes of homologous rat plasma to yield final hematocrits of 75-80 ml/100 ml. Care was taken in preparing these high-hematocrit infusates to avoid clotting, clumping, or hemolysis of red blood cells; if any of these untoward effects were observed, the blood was discarded. Inulin was present in this infusate in final concentrations of approximately 100 mg/100 ml. Following a 15-minute equilibration period, the measurements carried out in the normal hydropenic period were repeated.

ANALYTICAL PROCEDURES

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 the tubule fluid was measured, usually in duplicate, by the microfluorescence method of Vurek and Pegram (20). Inulin concentration in plasma was determined by the macroanthrone method of Fihr et al. (21). Protein concentrations in efferent arteriolar and femoral arterial blood plasmas were determined, usually in duplicate, with an ultramicrocolorimeter using a recently described (17) microadaptation of the method of Lowry et al. (22).

CALCULATIONS

Single nephron glomerular filtration rate was calculated as

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

where \((TF/P)_\text{IN}\) and \(V_{\text{TR}}\) refer to the transtubular inulin concentration ratio and the tubule fluid flow rate, respectively.

Single nephron filtration fraction was determined by the equation

\[ \text{SNFF} = 1 - \frac{C_{\text{A}}}{C_{\text{P}}} \]

where \(C_{\text{A}}\) and \(C_{\text{P}}\) denote afferent and efferent arteriolar protein concentrations, respectively.

Initial glomerular plasma flow rate was derived from the formula

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

Blood flow rate per single afferent arteriole or glomerulus was calculated as

\[ \text{GBF} = \text{GPF}/(1 - \text{Hct}_{A}) \]

where \(\text{Hct}_{A}\), the hematocrit of afferent arteriolar blood, was taken to be equal to the femoral arterial hematocrit.

Efferent arteriolar blood flow rate was determined as

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

Resistance per single afferent arteriole was derived from the formula

\[ R_{A} = \frac{[\bar{A}P - \bar{P}_{\text{oc}}]/\text{GPF}}{(1.962 \times 10^{19})} \]

where the factor \(7.962 \times 10^{19}\) is used to give resistance in units of dynes·sec·cm⁻¹ when \(\bar{A}P\) and \(\bar{P}_{\text{oc}}\) are expressed in mm Hg and GPF in nliters/min.

Resistance per single efferent arteriole was derived from the formula

\[ R_{E} = \frac{[\bar{P}_{\text{oc}} - P_{\text{E}}]/\text{EBF}}{(1.962 \times 10^{19})} \]

Total arteriolar resistance for a single pre- to postglomerular vascular unit was

\[ R_{TA} = R_{A} + R_{E} \]

An estimate of the net ultrafiltration pressure at the afferent end of the glomerular capillary \((P_{U\text{FA}})\) was determined using the expression

\[ P_{U\text{FA}} = \bar{P}_{\text{oc}} - P_{\text{T}} - \pi_{A} \]

An estimate of the net ultrafiltration pressure at the efferent end of the glomerular capillary \((P_{U\text{FE}})\) was determined using the equation

\[ P_{U\text{FE}} = \bar{P}_{\text{oc}} - P_{\text{T}} - \pi_{E} \]

Eqs. 10 and 11 assume that the colloid osmotic pressure of fluid in Bowman’s space \(\pi_{T}\) is negligible. This assumption has been validated by the finding in this strain of rats that the protein concentration of fluid in Bowman’s space is less than 200 mg/100 ml (23); accordingly, \(\pi_{T}\) is well below 1 mm Hg.

Mean glomerular transcapillary hydraulic pressure difference was calculated as

\[ \Delta P = \bar{P}_{\text{oc}} - P_{\text{T}} \]

The ultrafiltration coefficient \((K_{U})\) was calculated using a differential equation that gives the rate of change of protein concentration with distance along an idealized glomerular capillary. This equation, its derivation, and the method for its solution are given in detail elsewhere (18).

Results

EFFECT OF A REDUCTION OF ARTERIAL HEMATOCRIT

Individual and mean values for body weight, left kidney weight, \(\bar{A}P\), and a number of pertinent measures of single nephron function in seven normal hydropenic rats studied before and after an acute reduction of arterial hematocrit are summarized in Table 1 and Figure 1. Prior to the reduction in hematocrit, SNGFR averaged 30.4 ± 2.1 (se) nliters/min. SNFF averaged 0.38 ± 0.01 with individual values ranging from 0.34 to 0.40. Therefore, GPF, calculated using Eq. 4, averaged 81 nliters/min. \(\bar{P}_{\text{oc}}\) averaged 43.7 ± 1.2 mm Hg with individual values ranging from 40 to 49 mm Hg. Pressures measured at random sites along surface proximal convoluted tubules averaged nearly 11 mm Hg, yielding a mean value for the glomerular
**TABLE 1**

Effects of Reduction in Arterial Hematocrit on the Measured Determinants of Glomerular Ultrafiltration

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<th>Rat no.</th>
<th>Body wt (g)</th>
<th>Kidney wt (g)</th>
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<th>$P_{oc}$ (ml/100ml)</th>
<th>$P_{T}$ (g/100ml)</th>
<th>$P_{C}$ (g/100ml)</th>
<th>$Hct$ (ml/100ml)</th>
<th>$C_{A}$ (mm Hg)</th>
<th>$C_{E}$ (mm Hg)</th>
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See text for definition of abbreviations.

**TABLE 2**

Effects of Elevation in Arterial Hematocrit on the Measured Determinants of Glomerular Ultrafiltration

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<th>$Hct$ (ml/100ml)</th>
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See text for definition of abbreviations.

Circulation Research, Vol. 36, March 1975
### EFFECTS OF HEMATOCRIT ON GLOMERULAR FILTRATION

**Normal Hematocrit**

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**Low Hematocrit**

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### High Hematocrit

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+11.6 | -0.6 | +0.02 | -8.9 | -39 | -51 | -42 | +0.3 | +1.8 | +2.1 | -0.11 |

< 0.001 | > 0.5 | > 0.2 | < 0.025 | < 0.001 | < 0.001 | < 0.005 | < 0.025 | < 0.005 | < 0.005 | < 0.005 |

**Circulation Research, Vol. 36, March 1975**
Summary of the effects of isovolemic reductions and elevations in systemic hematocrit on several measures of surface nephron and microvascular function. Definition of symbols and abbreviations: HCT = systemic arterial hematocrit, \( R_a \) and \( R_e \) = afferent and efferent arteriolar resistances, respectively, \( \pi_a \) and \( \pi_e \) = afferent and efferent arteriolar oncotic pressures, respectively, \( \Delta P \) = mean glomerular transcapillary hydraulic pressure difference, SNFF = single nephron filtration fraction, GPF = initial glomerular capillary plasma flow rate, and SNGFR = single nephron glomerular filtration rate.

Transcapillary hydraulic pressure difference, \( \Delta P \), of 33 ± 1.4 mm Hg. Values for \( P_c \) and \( P_e \) averaged 8 ± 0.3 mm Hg and 13 ± 0.8 mm Hg, respectively. The hydraulic pressure drop along surface afferent arterioles (\( \Delta P - P_{GC} \)) averaged 73 ± 3 mm Hg, compared with a uniformly smaller pressure drop along surface efferent arterioles (\( P_{OC} - P_c \)) averaging 36 ± 1 mm Hg (\( P < 0.001 \)). Mean values for \( R_a \) and \( R_e \) are summarized in Table 1 and Figure 1. On the average, afferent arterioles contributed nearly two-thirds of the total resistance to blood flow to the level of the smallest peritubular capillaries, a finding in close quantitative accord with that reported by us previously (9-12).

Microassay measurements of total protein concentration in afferent (\( C_a \)) and efferent (\( C_e \)) arteriolar plasmas yielded values averaging 5.2 ± 0.1 g/100 ml and 8.3 ± 0.2 g/100 ml, respectively. \( \pi_a \) and \( \pi_e \) calculated from these protein concentrations are given in Table 1. Using Eqs. 10 and 11, it is possible, from measurements of \( P_{GC} \) and \( P_T \) and these estimates of \( \pi_a \) and \( \pi_e \), to determine the magnitude of the transcapillary pressure difference favoring ultrafiltration across afferent- and efferentmost points along the glomerular capillary network in each rat. \( P_{GC} \) exceeded the sum of the opposing pressures (\( P_T + \pi_a \)) at afferent ends of the glomerular capillaries by an average of 16.7 ± 1.3 mm Hg. At the efferent ends, this imbalance of pressures had essentially disappeared, averaging -0.5 ± 0.6 mm Hg, a value not significantly different from zero (\( P > 0.5 \)). For all seven rats, the ratio \( \pi_e / \Delta P \) averaged 1.02 ± 0.02, a value not significantly different from unity (\( P > 0.5 \)), indicating that filtration pressure equilibrium was achieved under these conditions of normal hydropenia. All values for single nephron and microvascular function are in close accord with values for similarly hydropenic Wistar rats reported from this laboratory previously (9-11, 23).

Isovolemic plasma exchange resulted in a uniform and large fall in arterial hematocrit from a mean value of 50.5 ± 1.1 ml/100 ml to 21.3 ± 0.8 ml/100 ml (\( P < 0.001 \)). This fall was attended by a modest and statistically significant reduction in \( \Delta P \) (Table 1). As shown in Table 1 and Figure 1, despite this reduction in \( \Delta P \), SNGFR increased in every rat (range +4 to +20 nliters/min), on the average, to 40.5 ± 3.1 nliters/min (\( P < 0.005 \)). Although we measured considerable variation in mean SNGFR values from rat to rat (Table 1), in any individual rat there was close agreement among SNGFR values from nephron to nephron. SNFF declined in each rat (range -0.10 to -0.15), on the average, to 0.26 ± 0.01. Hence, following the isovolemic reduction in hematocrit, GPF increased proportionately more than did SNGFR (Table 1 and Fig. 1). The rise in SNGFR occurred despite small but significant reductions in \( \Delta P \), on the average, to 30.1 ± 0.9 mm Hg (\( P < 0.025 \)). Values for \( P_c \) and \( P_e \) remained essentially unchanged from control. With the reduction in hematocrit, the hydraulic pressure drop along afferent arterioles declined in each rat, on the average, to 57 ± 4 mm Hg (\( P < 0.001 \)). In contrast, no important alteration in the magnitude of the pressure drop along efferent arterioles was observed (mean 33.7 ± 1.4 mm Hg, \( P > 0.2 \)). Rates of blood flow through single afferent (\( GBF \)) and efferent (\( EABF \)) arterioles increased uniformly from average values of 165 ± 13 nliters/min and 134 ± 11 nliters/min before the isovolemic reduction in hematocrit to 205 ± 16 nliters/min and 164 ± 13 nliters/min after the hematocrit reduction, respectively. It follows from Eq. 7 that, since this increase in \( GBF \) occurred in association with a substantial and highly significant decline in the axial hydraulic pressure drop along the afferent arteriole, \( R_a \) declined signifi-
cantly (Fig. 1 and Table 1). The lesser decline in $R_A$ was the consequence of a modest rise in $EABF$ without an appreciable change in the efferent arteriolar hydraulic pressure drop. Since the decline in $R_A$ was substantially greater than that in $R_E$, the contribution of $R_A$ to the total arteriolar resistance, $R_{TA}$, fell significantly with the hematocrit reduction so that it represented only slightly more than half of the total resistance to arteriolar blood flow to the level of the smallest peritubular capillaries.

With the hematocrit reduction, $C_A$, on the average, remained unchanged (Table 1), whereas $C_E$ declined in each rat (range $-1.0$ to $-2.1$ g/100 ml), on the average, to $6.8 \pm 0.1$ g/100 ml ($P < 0.001$). Corresponding values of $\pi_A$ and $\pi_E$ are given in Table 1 and Figure 1. Thus, the large increases in $SNGFR$ occurred in association with a slight, but significant, decline in net ultrafiltration pressure at afferent ($P_{UFA}$) and a larger and more highly significant rise at efferent ($P_{UFE}$) ends of glomerular capillaries (Table 1). Accordingly, the ratio $\pi_E/\Delta P$ averaged $0.82 \pm 0.02$ (difference from unity, $P < 0.001$), indicating that under these conditions filtration pressure equilibrium failed to obtain. A unique value of the glomerular capillary ultrafiltration coefficient, $K_f$, can be determined only under conditions in which filtration pressure equilibrium is not reached (18), i.e., when $\pi_E/\Delta P < 1$. This situation was obtained in all seven rats following the hematocrit reduction. Individual values for $K_f$ ranged from 0.057 to 0.107 nliters/(sec·mm Hg) and averaged $0.070 \pm 0.007$ nliters/(sec·mm Hg), a value very nearly the same as that reported by us previously for normal Wistar rats (24).

**EFFECT OF AN ELEVATION OF ARTERIAL HEMATOCRIT**

Individual and mean values for body weight, kidney weight, $\Delta P$, and the various measures of single nephron and microvascular function in a second group of seven normal hydropenic rats, studied before and after an acute elevation of systemic hematocrit, are summarized in Table 2 and Figure 1. These rats were similar to those in the preceding group with respect to body weight, kidney weight, and $\Delta P$. Values for each measure of single nephron and microvascular function prior to the elevation of hematocrit were also essentially the same as those reported for the corresponding period in the group of rats already discussed. Isovolemic exchange with high-hematocrit donor blood resulted in a uniform elevation of arterial hematocrit, on the average, from $31.1 \pm 0.6$ ml/100 ml to $62.5 \pm 1.0$ ml/100 ml ($P < 0.001$). Without changing $\Delta P$, the elevation in hematocrit resulted in a nearly uniform decline in $SNGFR$, on the average, from $32.6 \pm 3.4$ nliters/min to $23.7 \pm 3.3$ nliters/min ($P < 0.025$). Once again, although considerable variation was noted in $SNGFR$ values from rat to rat, close agreement among nephrons was found in each individual rat. $SNFF$ increased markedly in each rat from a mean value of $0.37 \pm 0.01$ to $0.49 \pm 0.01$ ($P < 0.001$). Hence, $SNGFR$ declined proportionately less than did $GPF$ (Fig. 1), the latter falling from $88 \pm 9$ nliters/min to $49 \pm 7$ nliters/min ($P < 0.001$) (Table 2). The elevation in hematocrit was associated with a large and uniform rise in $P_{OC}$, on the average, from $45.1 \pm 0.6$ mm Hg to $56.3 \pm 1.2$ mm Hg ($P < 0.001$). Since $P_{f}$ remained essentially unchanged (Table 2), the increment in $\Delta P$ was essentially the same as that for $P_{OC}$, averaging $+10.6 \pm 1.2$ mm Hg ($P < 0.001$). Following the elevation in hematocrit, the drop in hydraulic pressure along surface afferent arterioles declined significantly from a mean of $73 \pm 3$ mm Hg to $59 \pm 5$ mm Hg ($P < 0.005$). In contrast, the axial pressure drop along surface efferent arterioles increased appreciably, on the average, from $38 \pm 1$ mm Hg to $45 \pm 2$ mm Hg ($P < 0.01$). $GBF$ declined substantially with the hematocrit elevation (Table 2), but since this decline was very nearly proportional to the magnitude of the decline in the hydraulic pressure drop along afferent arterioles, $R_A$ remained essentially unchanged from control values (Fig. 1). In contrast, the impressive reduction in $EABF$ associated with a relatively large rise in the axial pressure drop along efferent arterioles resulted in a large and highly significant increase in $R_E$ (Fig. 1). Accordingly, the contribution of $R_E$ to the total arteriolar resistance, $R_{EA}$, increased from 39% before to 50% after the elevation of hematocrit ($P < 0.005$).

Following the hematocrit elevation, $C_A$ remained unchanged, on the average, from values measured during the control period (Table 2), whereas $C_E$ uniformly rose (range $+1.0$ to $+1.9$ g/100 ml), on the average, to $10.0 \pm 0.2$ g/100 ml ($P < 0.001$). Corresponding values of $\pi_A$ and $\pi_E$ are given in Table 2 and Figure 1. Thus, measured declines in $SNGFR$ occurred despite large and highly significant elevations in $P_{UFA}$ from a mean value of $17.8 \pm 0.6$ mm Hg before to $29.4 \pm 1.3$ mm Hg after the elevation in hematocrit ($P < 0.001$). Nevertheless, as in the period before the hematocrit elevation, net ultrafiltration pressure still declined essentially to zero by the efferent end of the glomerular capillary network ($P_{UFE}$), averaging $0.1 \pm 0.7$ mm Hg before and $-1.2 \pm 1.1$ mm Hg after the
elevation in hematocrit. Accordingly, the ratio \( \pi_e / \Delta P \) approximated unity, averaging 1.00 ± 0.02 and 1.02 ± 0.02, respectively, before and after the elevation of hematocrit (Table 2), indicating that filtration pressure equilibrium was achieved during both periods. Given filtration pressure equilibrium, unique values of \( K_f \) could not be calculated for this group of rats.

**Discussion**

The effects of variations in systemic hematocrit on renal function, particularly intrarenal hemodynamics, have long been of interest to renal physiologists (25-36). In many studies, however, alterations in hematocrit have been induced in concert with alterations in plasma and extracellular volume, thereby precluding an assessment of the effects of selective alterations in hematocrit alone. By contrast, Nashat and Portal (33) and Schrier and Earley (35) in studies in the dog and Brenner and Galla (36) in studies in the rat have examined the effects of selective reductions in systemic hematocrit on renal hemodynamics induced by isovolumetric exchange transfusions. In accord with the findings in the present study, these investigators have uniformly observed declines in the filtration fraction, the result of proportionately greater increases in renal plasma flow than in filtration rate.

Insight into the mechanisms governing the decline in the filtration fraction is provided by the results obtained in the present study. As summarized in Figure 1, with an isovolumetric reduction in hematocrit, SNFF decreased due to both the most decline in \( \Delta P \) and the marked increase in GPF, the latter to a value large enough to prevent achievement of filtration pressure equilibrium, the equality of \( \Delta P \) and \( \pi_e \). As is clearly evident in the present study, this increase in GPF was the result of a marked decline in total arteriolar resistance, \( R_{Ta} \) (Table 1). Moreover, as shown in Figure 1, the decline in afferent arteriolar resistance, \( R_s \), was far greater than that in efferent arteriolar resistance, \( R_e \).

The results obtained in the present study also permit examination of the mechanisms responsible for the observed increase in SNFF following an isovolumetric reduction in hematocrit. The rate of glomerular ultrafiltration can be expressed as

\[
SNFF = K_f \cdot P_{UF} = k \cdot S \cdot P_{UF},
\]

(13)

where \( P_{UF} \) is the mean net ultrafiltration pressure (\( P_{UF} \) averaged along the length of the capillary) and \( K_f \), the ultrafiltration coefficient, is the product of the effective hydraulic permeability (\( k \)) and the total surface area (\( S \)) of the glomerular capillaries. It is evident from Eq. 13 that the increase in SNFF following a reduction in hematocrit could have resulted from an increase in \( K_f \), \( P_{UF} \), or both. The first possibility, an increase in \( K_f \), can be excluded, since following the reduction in hematocrit \( K_f \) averaged 0.070 nliters/(sec-mm Hg), a value in close accord with that reported by us previously (0.078 nliters/[sec-mm Hg]) for this same strain of rats (24). For reasons discussed in detail elsewhere (18, 24), the existence of filtration pressure equilibrium, which obtained prior to the reduction in hematocrit, precludes the determination of a unique value of \( K_f \) for the initial, pre-plasma exchange period. It was confirmed, however, using a recently reported model of glomerular ultrafiltration (18) that the value of \( K_f \) of 0.070 nliters/sec-mm Hg determined after the reduction in hematocrit is also consistent with the equilibrium data obtained in the period prior to exchange transfusion. Therefore, the present evidence is interpreted to indicate that the increase in SNFF induced by the isovolemic reduction in hematocrit resulted solely from the increase in \( P_{UF} \).

As discussed in detail elsewhere (18, 37), for constant \( K_f \), changes in \( P_{UF} \) and hence SNFF are determined solely by changes in \( C_A \), \( \Delta P \), and GPF. Changes in GPF modify the average glomerular transcapillary colloid osmotic pressure difference: increases in GPF tend to reduce this average transcapillary osmotic pressure difference, and the opposite is true for decreases in GPF. Following the reduction in hematocrit, we noted no significant change in \( C_A \) relative to initial hydropenic values. Moreover, \( \Delta P \) declined to a small but statistically significant extent following the exchange transfusion, an effect that would tend to diminish SNFF. Therefore, the observed increase in SNFF following the reduction in hematocrit could only have resulted from the measured large increase in GPF.

Only two studies to date have been concerned with the effects of isovolemic elevations in systemic hematocrit on renal hemodynamics. Nashat and Portal (33), in studies in the dog, and Brenner and Galla (36), in studies in the rat, both demonstrated a rise in filtration fraction; in both studies this rise was due to a proportionately greater fall in plasma flow than in filtration rate. Of interest, Spencer (28) was able to demonstrate a rise in filtration fraction even when the elevation in systemic hematocrit was induced in volume-expanded dogs.
Whereas in these studies the elevation in hematocrit was induced acutely, it has also been shown that similar increases in filtration fraction result when hematocrit is elevated in a more chronic fashion, namely, following sustained hypoxia (30, 32).

Once again, the findings in the present study provide insight into the mechanisms governing the high-hematocrit-induced elevations in filtration fraction. As shown in Figure 1, the rise in SNFF with the isovolemic elevation in systemic hematocrit, the result of a proportionately greater fall in GPF than in SNGFR, was the result of the marked increase in \( \Delta P \), the latter entirely due to the rise in \( T_{GC} \). These changes were associated with a marked increase in total arteriolar resistance, \( R_{TA} \) (Table 2); this increase was almost entirely the consequence of the rise in efferent arteriolar resistance, \( R_E \).

Filtration pressure equilibrium, the equality between \( \pi_F \) and \( \Delta P \), was obtained both before and after the isovolemic elevation in hematocrit. As discussed elsewhere (18, 37), given filtration pressure equilibrium, \( SNGFR \) is determined by just three factors, namely, \( C_A \), \( \Delta P \), and \( GPF \). \( C_A \) was essentially unchanged following the elevation in hematocrit, whereas \( \Delta P \) and \( GPF \) changed in opposite directions; the increase in the former tended to raise \( SNGFR \), and the decline in the latter tended to lower \( SNGFR \) by increasing the length-averaged glomerular transcapillary oncotic pressure difference. Clearly, the measured decline in \( GPF \) was sufficient to more than offset the measured increase in \( \Delta P \), hence the decline in \( SNGFR \). Had \( \Delta P \) not increased, \( SNGFR \) would have fallen in proportion to the decline in \( GPF \), \( SNNF \) remaining constant. Since \( \Delta P \) was instead found to increase, \( SNNF \) likewise increased.

The findings in the present study provide an attractive explanation for the general tendency for glomerular filtration rate to vary inversely with and filtration fraction to vary directly with systemic hematocrit in a wide variety of anemic and polycythemic states in man (29, 38-41). Thus, in children with sickle cell, nutritional, and hemolytic anemias (38, 39), GFR is usually greater than normal and renal plasma flow is much greater than normal so that the filtration fraction is reduced. In adults, however, the effects of chronic anemia on renal hemodynamics are more variable (39, 42, 43) in part due to the intrinsic alterations in renal structure and function often associated with clinical anemias (e.g., sickle cell nephropathy). Conversely, in polycythemic states in man, whether due to primary (polycythemia vera) or secondary (congenital, cyanotic heart disease, or acclimatization to high altitude) erythrocytosis, elevations in the filtration fraction and reductions in the filtration rate have been observed consistently (29, 40, 41).

It is apparent from the present findings that alterations in renal arteriolar resistance are of fundamental importance in bringing about the observed changes in glomerular plasma flow rate and filtration rate in response to variations in systemic hematocrit. Resistances to blood flow in single afferent and efferent arterioles (\( R_A \) and \( R_E \), respectively) may be altered by changes in luminal diameter, effective blood viscosity, or both (assuming that arteriolar lengths remain constant). Of interest is whether changes in effective blood viscosity alone, induced by changes in systemic hematocrit, are sufficient to account for the alterations in \( R_A \) and \( R_E \) observed in the present study or whether it must be inferred that changes in luminal diameter also occurred. The effect of hematocrit on blood viscosity measured in macroscopic systems (e.g., Couette or cone and plate viscometers) is well known: viscosity increases with hematocrit (44). A similar trend has been observed in glass capillary tube viscometers, in which the non-Newtonian, particulate nature of blood flow becomes especially apparent for tube diameters less than \( \sim 500 \mu \) (45-47). In these small glass capillary tubes, the tube diameter is an important determinant of the extent to which changes in hematocrit result in changes in effective blood viscosity. Although effective blood viscosity has been found to be relatively insensitive to hematocrit for tube diameters less than \( \sim 10 \mu \) (45, 47, 48), profound effects occur in tubes larger than \( 10 \mu \) (45-47). For tube diameters in the range expected for afferent and efferent arterioles, roughly 15-25\( \mu \) (49, 50), the only data available to date indicate that an increase in hematocrit from a value of 43 to 63 ml/100 ml results in almost a doubling of effective blood viscosity (47), the percent increase being somewhat greater in 25\( \mu \) than in 15\( \mu \) diameter tubes. Since in the present study \( R_A \) and \( R_E \) changed by less than a factor of two (Tables 1 and 2 and Fig. 1), it is possible that these changes in arteriolar resistance resulted almost entirely from changes in effective blood viscosity. Unfortunately, this conclusion can only be qualitative, since the dependence of effective blood viscosity on hematocrit is likely to be highly nonlinear and since data for tubes similar in diameter to renal arterioles are available only at two hematocrit values, 43 and 63 ml/100 ml (47).
is therefore essential that future studies be carried out to provide these much-needed viscometric data for tube diameters in the 15µ to 25µ range and for a wide range of hematocrits.

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