Vascular Capacitance in Rats Subjected to Chemical Renal Medullectomy

Mair E. Edmunds, Gavin I. Russell, Paul R. Burton, and John D. Swales

Selective renal medullary destruction is produced in rats by a single injection of 2-bromoethylamine hydrobromide. The object of these studies was to investigate whether destruction of the renal medulla in normal rats would alter vascular capacitance. Conscious bromoethylamine-treated rats \((n=15)\) were compared with control saline-injected rats \((n=12)\). Mean circulatory filling pressure was measured during a brief circulatory arrest caused by inflation of a right atrial balloon. Blood volume was determined from plasma volume (iodine-125-labeled albumin) and hematocrit. Mean circulatory filling pressure was measured at resting blood volume and after rapid blood volume changes. Vascular compliance was derived from the mean circulatory filling pressure–blood volume curve. The bromoethylamine-treated rats were significantly hypertensive compared with control rats (mean arterial pressure 133±2 and 114±3 mm Hg, respectively, \(p<0.001\)) and had a significant tachycardia (475±8 and 443±10 beats/min, respectively, \(p=0.02\)). Blood volume, plasma volume, hematocrit, and sodium excretion were no different. There was no significant difference in mean circulatory filling pressure (6.5±0.2 and 6.8±0.2 mm Hg, respectively, \(p=0.4\)) or vascular compliance (3.64±0.20 and 3.53±0.12 ml/kg/mm Hg, respectively, \(p=0.7\)). The position of the vascular pressure–volume curve was unchanged indicating no change in vascular capacity. This would suggest that the destruction of renal medullary vasodepressor mechanisms does not result in alterations in vascular capacitance. (Hypertension 1990;15:576–582)

Selective destruction of the renal medulla in normal rats can be produced by a single injection of 2-bromoethylamine hydrobromide and results in moderate but consistent elevation of the blood pressure.\(^{1,2}\) The severity of the hypertension is proportional to the degree of histological damage seen in the medulla.\(^1\) The mechanisms responsible for the increase in blood pressure are not fully understood. Urine volumes are grossly elevated,\(^2\) but exchangeable sodium is normal after 2 weeks; plasma renin is low or normal; vasopressin levels are slightly elevated but not to pressor levels, and infusion of arginine vasopressin (AVP) antagonists does not lower the blood pressure.\(^4\) In the absence of obvious candidates for increased pressor activity, attention has focused on the possible loss of medullary vasodepressor systems.

Muirhead and others\(^{5,6}\) have postulated that the renal medulla contains lipid substances that have vasodepressor potential.\(^5\) These are called renomedullary lipids (antihypertensive neutral renomedullary lipid [ANRL] and antihypertensive polar renomedullary lipid [APRL] or platelet activating factor) and are produced in the interstitial cells of the renal medulla.\(^6\) Transplants of renal medulla or infusion of renomedullary lipids have been shown to have a blood pressure-lowering effect in rats with experimental hypertension.\(^7,8\)

Hemodynamic studies have demonstrated that the elevated blood pressure seen in the chemical renal medullectomy model of hypertension is associated with an increase in peripheral vascular resistance.\(^9\) However, it is possible other mechanisms also contribute. It is known that changes in vascular capacitance have an important role in the regulation of arterial pressure by influencing the venous return to the heart, cardiac filling, and thus cardiac output.\(^10\) Accordingly, alterations in vascular capacitance have been described in other forms of hypertension.\(^11\)

Experiments were therefore designed to study the role of the renal medullary vasodilator system in blood pressure control, in particular its interaction with the venous side of the circulation. Mean circulatory filling pressure (MCFP) is the pressure that...
would be obtained throughout the circulation if the blood was instantaneously redistributed so that all the pressures were equal, and it is an index of the filling of the vascular system as it is dependent on the blood volume and venous capacity and compliance. Recently, Yamamoto et al devised a method of measuring vascular capacitance in conscious restrained rats, thus avoiding any complicating effects of the anesthetic agents. This technique was used in rats rendered hypertensive by bromoethylamine-induced renal papillary necrosis.

**Methods**

Female Wistar rats that weighed 200–220 g were given either a single injection of approximately 0.4 ml bromoethylamine hydrobromide (0.2 g/kg) (Sigma UK, London, England) as a solution containing 10 g/100 ml in isotonic sodium chloride solution (0.9 g/100 ml) or a control injection of a similar volume of isotonic saline intravenously via the tail vein during light ether anesthesia. The rats were then housed individually with free access to standard rat chow and water. Four to five weeks after injection, the rats were placed in metabolic cages and, after a 24-hour period of acclimatization, a 24-hour urine collection was made. Blood samples for measurement of plasma renin concentration (PRC) and creatinine were taken when urine collection was made. Blood samples were obtained under light ether anesthesia by amputation of the tail end, according to a previously established protocol. The blood sample was obtained at least 1 week before cannulation to allow recovery of the blood volume before further investigations. The volume of the blood sample taken was 0.8 ml (i.e., between 5 and 10% of the blood volume of the rat). Other studies in rats have shown that acute bleeding of this degree is followed by full recovery of the hematocrit within 8 days. Bromoethylamine-treated rats with 24-hour urine volumes of 25 ml or greater were selected for further study; the average urine volume of normal Wistar rats is 12 ml/24 hr.

**Operative Procedures**

At 6 weeks, the rats were cannulated under ether anesthesia. The left carotid artery was cannulated with a short length of polyethylene tubing PE-50 (0.58 mm i.d., 0.96 mm o.d.) and PE-90 tubing (0.86 mm i.d., 1.27 mm o.d.); two PE-10 catheters (0.28 mm i.d., 0.61 mm o.d.) and a PE-25 catheter (0.4 mm i.d., 0.8 mm o.d.) were inserted into the left femoral vein and the PE-25 catheter was advanced to the thoracic vena cava for recording of central venous pressure. A balloon-tipped PE-50 catheter was inserted via the jugular vein into the right atrium. The catheters in the carotid and thoracic vena cava were connected to Statham P23ID transducers (Statham Instr. Division, Gould Inc., Oxnard, California) and arterial and central venous pressures were recorded on a multichannel Grass recorder (Grass Instr. Co., Quincy, Massachusetts). Baseline pressure was set at cardiac level by inspection (two thirds of the distance between the anterior and posterior chest wall from the back). The transducer recorder channel used for measurement of the venous pressure was calibrated with a water manometer; sensitivity was set at 10.9 cm water (8 mm Hg)/2 cm deflection. The correct position of the catheters was determined by inflating the right atrial balloon with 0.3 ml saline and observing the characteristic smooth increase in venous pressure and simultaneous decrease in arterial pressure to less than 25 mm Hg. If this was not observed, the balloon was repositioned. (The position of the balloon in the right atrium was confirmed at the end of each experiment by postmortem examination after thoracotomy.) All catheters were exteriorized between the scapulae and protected by a light flexible metal coil attached to the rats by a linen jacket. The coil was attached to an overhead lightly counterbalanced arm to ensure minimal tension as described. The arterial catheter and the PE-25 venous catheter were each flushed with 0.2 ml heparinized dextrose (50 g dextrose/l; 10 IU heparin/ml). Heparinized dextrose (as above) was then constantly infused through the PE-10 venous catheters overnight and throughout the study at a rate appropriate to the previous 24-hour urine volume. All subsequent measurements were made in conscious, unrestrained rats.

**Plasma Volume, Hematocrit, and Blood Volume**

Plasma volume was measured using the distribution of radioiodine-labeled albumin (RISA) (Amersham International, Amersham, England); a single 0.6 ml arterial blood sample was taken at 5 minutes after injection of the isotope. Approximately 75 kilobecqueral (kBq) RISA in 0.2 ml 0.9% saline was injected intravenously via the PE-10 catheter and flushed with 0.2 ml dextrose (50 g/l). The number of counts injected was equal to initial counts in the syringe minus the counts remaining in the syringe and PE-10 tubing. At 5 minutes after injection, the arterial sample was taken, and the sample volume was then replaced with 0.6 ml fresh donor rat blood. Arterial hematocrit was measured. The remaining plasma was separated and duplicate aliquots of 0.1 ml plasma were counted in a Packard Auto-Gamma 5650 (Canberra-Packard, Pangbourne, Berks, UK); all counts were corrected for background activity. Plasma volume was calculated from the counts per minute injected divided by counts per minute per milliliter plasma. Blood volume was calculated from plasma volume and hematocrit by using an F cell factor of 0.81 to correct arterial hematocrit to total body hematocrit.

\[
BV = PV/(1 - 0.8 \text{ hct/100})
\]

where BV is blood volume, PV is plasma volume, and hct is hematocrit. Plasma volume was measured before and after the compliance studies in the same way with iodine-125 and iodine-131, respectively.
**Mean Circulatory Filling Pressure and Mean Circulatory Filling Pressure—Blood Volume Curve**

MCFP was measured by inflating briefly an atrial balloon with 0.3 ml 5% dextrose and recording arterial pressure and central venous pressure. During the circulatory arrest, central venous pressure increased reaching a plateau within 3–5 seconds (venous plateau pressure) and arterial pressure fell to a low plateau (final arterial pressure). The balloon was then deflated and the circulation rapidly returned to normal. MCFP was calculated as follows:

\[
\text{MCFP} = \frac{(\text{FAP} - \text{VPP})}{60}
\]

where VPP is the venous plateau pressure and FAP is the final arterial pressure, and an arterial/venous compliance ratio of 1:60 in the Wistar rat is assumed. Vascular capacitance describes the pressure–volume relation of the vasculature. This can be described in terms of the vascular capacity, which is the volume contained at any given filling pressure, and the vascular compliance, which is the slope of the pressure–volume curve at a specified capacity. To obtain a measure of vascular capacitance, MCFP was also measured after blood volume was increased and decreased by approximately 10% by rapid infusion or withdrawal of 6 ml/kg blood via the arterial catheter. MCFP was measured within 12 seconds of the start of the blood volume change, then blood volume was immediately restored to normal. Blood volume changes were made in random order. MCFP was measured again between blood volume changes and at the end of the experiment; baseline MCFP was the mean of these three readings. With small changes in blood volume (<15%), the pressure–volume relation may be considered linear. The MCFP–blood volume curve was obtained for each rat from these five data points by linear regression analysis. Compliance \((dV/dP)\) was the reciprocal of the slope of this curve and represented total vascular compliance. Unstressed vascular volume is defined as the volume contained in the vasculature at a distending pressure of zero. It was obtained by extrapolating the linear portion of the MCFP–blood volume curve to zero MCFP.

**Hemodynamic Measurements**

Baseline hemodynamic measurements of heart rate (beats/min), mean arterial pressure (diastolic+1/3 systolic—diastolic pressure) (mm Hg), central venous pressure (mm Hg), and MCFP (mm Hg) were determined in both groups of rats 15 hours after cannulation. Blood volume was measured and MCFP–blood volume curves constructed for control rats and bromoethane-amine-treated rats.

**Pathology**

At the end of the experiment, each rat was killed and the kidneys were inspected macroscopically. A rat was excluded if the damage was less than confluent necrosis of the papilla and inner medulla.

**Plasma Renin Concentration**

Blood was collected in a precooled tube containing 0.1 ml concentrated dipotassium EDTA. After centrifugation at 4° C, the plasma was stored at −20° C. PRC is a measure of the concentration of plasma renin in terms of its activity. This is accomplished by inactivation of the endogenous substrate in the form of plasma from binephrectomized rats. The generation of the product angiotensin I (Ang I) is then measured by radioimmunoassay, and the results expressed as the quantity of Ang I generated under standardized conditions over a fixed time (pmol Ang I/ml/hour).14–18

**Plasma and Urine Creatinine**

Plasma and urine creatinine were measured by the Jaffe colorimetric method without deproteinization with a test kit (Boehringer Mannheim GmbH Diagnostica, Mannheim, West Germany).19

**Urine Electrolytes**

Sodium and potassium concentrations (mmol) were determined by flame photometry.

**Statistics**

Unless otherwise stated, results are expressed as mean±SEM. Simple comparisons of group means were made using two-sample \( t \) tests and Student’s \( t \) test. Plasma renin concentration is not normally distributed and therefore was logarithmically transformed before parametric analysis. The variation among the three baseline measurements of MCFP in each rat was assessed with the coefficient of variation. Compliance and unstressed vascular volume were investigated with a two-stage regression analysis. In stage one, maximum likelihood estimates for the regression parameters representing the intercept and gradient of the MCFP–blood volume curve for each individual rat were obtained by using linear modeling (multiple linear regression). In view of the design of the experiments, MCFP was modeled as the dependent variable. The constant term and the main effect for “VOLUME” were forcibly removed from the regression model, the regression coefficients \((I, I_1, \ldots, I_2)\) for a 27-level “RAT” factor then provided the necessary intercept estimates, and the coefficients \((G_1, G_2, \ldots, G_2)\) for 27 “RAT \cdot VOLUME” interaction terms provided the required gradients. The compliance in rat “k” was then estimated as \(1/G_k\), and its unstressed vascular volume was estimated as \(-I_1/G_k\). During the first stage of the analysis, a formal test for nonlinearity of the individual MCFP–blood volume curves was performed by adding appropriate polynomial volume terms into the model. In stage two of the analysis, the estimates for compliance and for unstressed vascular volume that had been obtained during stage one were compared directly between bromoethane-amine-treated and control rats using two-sample \( t \) tests. This provided an appropriate means of testing for differences in capaci-
itorance or compliance. Data was analyzed with GLIM 3.77.20

The calculation of unstressed vascular volume necessa-

lied the extrapolation of regression relations well beyond the
bounds of the observed data. It was therefore recognized that
to find, or fail to find, a major difference in unstressed vascular
volume between the two groups of rats might not necessarily
 imply an analogous difference (or equivalence) in
capacity at MCFP values within the range actually
observed in the experiments. Therefore, vascular
capacity was also estimated at a range of more
"realistic" MCFP values (5.0, 6.6, 8.2) that fell within
the range of the observed data, and volume differ-
ences were investigated at these pressures.

Results

Fifteen bromoethylamine-treated rats were com-
pared with 12 control rats. There was no significant
difference in body weight between the bromoethyl-
amine-treated and control group (257±23 and
273±23 g, respectively, p=0.1) or in the number of
days after injection at the time of study (40±2 and
41±2, respectively, p=0.9).

Bromoethylamine-treated rats had a urine output of
37±2 ml/24 hr and normal rats 16±2 ml/24 hr.
There was no significant difference in urinary pH
(8.13±0.30 and 8.13±0.37, respectively, p=0.99) or
24-hour urinary sodium excretion (2.77±0.13 and
2.37±0.20 mmol/24 hr, respectively, p=0.1), but ur-
nary potassium excretion was increased in the
bromoethylamine-treated group (1.79±0.10 vs.
1.39±0.11 mmol/24 hr, respectively, p=0.01). Plasma
creatinine (63±4 and 55±2 µmol/l, respectively,
respectively, p=0.1) and creatinine clearances (0.92±0.09
and 0.78±0.09 ml/min, respectively, p=0.3) were not
significantly different.

PRC was similar in the two groups (122±36 in
bromoethylamine-treated rats vs. 80±22 pmol Ang
I/hr/ml, p=0.4). Bromoethylamine-treated rats were
significantly hypertensive compared with control rats
(mean arterial pressure 133±2 vs. 114±3 mm Hg,
respectively, p<0.001), and they had a significant
tachycardia (heart rate 475±8 vs. 443±10 beats/min,
respectively, p=0.02).

Blood Volume Measurements

There was no significant difference in initial
plasma volume (43.5±1.2 ml/kg in bromoethylamine-
treated compared with 40.1±0.7 ml/kg in control
rats, p=0.07) or in hematocrit (40.1±0.7% vs.
41.3±0.5%, respectively, p=0.2). Blood volume was
similar in both groups (64.0±1.5 vs. 60.8±0.8 ml/kg,
respectively, p=0.1).

Venous Capacitance Measurements

There was no difference in central venous pres-
sure. There was no significant difference in resting
MCFP between the two groups (6.5±0.2 and 6.8±0.2
mm Hg, respectively, p=0.4). The coefficient of vari-
ation for repeated baseline measurements within
each rat was 5%.

Maximum likelihood linear regression lines were
fitted to the MCFP and blood volume data points as
outlined under Methods. Formal testing for nonlinear-
earity suggested that it was acceptable to model the
MCFP-blood volume curves as straight lines. Esti-

imated vascular compliance was not significantly dif-
ferent (p=0.7) between the two groups of rats
(3.64±0.20 and 3.53±0.12 ml/kg/mm Hg, respective-
ly). Formal tests for capacity shift were carried out at
three different MCFP values that lay within the range
of the observed data. No test indicated a significant
shift (p>0.08). Similarly, there was no significant
difference in the unstressed vascular volume in
bromoethylamine-treated rats compared with con-
trols (40.2±2.0 vs. 37.6±1.3 ml/kg, respectively).
There was therefore no significant evidence of a
change of vascular capacitance (see Figure 1).

Discussion

The results reported here confirm that chemical
medullectomy results in hypertension but indicate
that this is not associated with changes in vascular
capacity or compliance.

The damage produced by bromoethylamine is
selective for the renal medulla, with little evidence of
cortical injury. The present study showed no differ-
ence in plasma creatinine or creatinine clearance.
Bromoethylamine-treated rats were markedly poly-
uric, but did not show a significant difference in
24-hour sodium excretion. Previous studies have
shown that, although sodium balance is negative over
the first week after bromoethylamine injection, it
then returns to normal,2 and by 5–7 weeks there is no
difference in total body exchangeable sodium in rats
subjected to chemical medullectomy compared with
control rats.3 The finding of unchanged blood vol-
ume, plasma volume, or hematocrit in rats subjected
to chemical medullectomy confirms the view that the
hypertension is not secondary to sodium retention

<table>
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<th>Table 1. Blood Volume, Mean Circulatory Filling Pressure, Vascular Compliance, and Other Variables in Bromoethylamine-Treated and Control Rats</th>
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<tr>
<td>Variable</td>
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<td>Hematocrit (%)</td>
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<td>Plasma volume (ml/kg)</td>
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<td>Blood volume (ml/kg)</td>
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<td>Mean arterial pressure (mm Hg)</td>
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<td>Unstressed vascular volume (ml/kg)</td>
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<td>Compliance (ml/kg/mm Hg)</td>
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Values are mean±SEM. BEA, bromoethylamine; MCFP, mean
circulatory filling pressure.

*p<0.001.

†p<0.05.
with a raised intravascular volume, and this is supported by the observation that no difference in plasma renin or body weight has been found. In the present experiment, the rats subjected to chemical renal medullectomy had a significant tachycardia compared with the control group. This finding has not been consistent in previous studies and may reflect increased susceptibility to sympathetic activation in response to stress. Plasma noradrenaline has not been found to be elevated in this model. At present, the involvement of the sympathetic nervous system in the hypertension in the renomedullary-damaged model remains inconclusive, although other studies of the renomedullary vasodepressor system have suggested an interaction with adrenergic vascular control.

As the hypertension observed in this model does not seem to be associated with sodium retention or obvious activation of pressor mechanisms, an alternative explanation would be the impairment of vasodepressor mechanisms, in particular deficiency of the renomedullary lipid described by Muirhead. In rabbits subjected to bilateral nephrectomy and a high sodium load, autotransplantation of fragmented renal medulla, consisting mainly of renal interstitial cells, initially blunted the rise in blood pressure and later reversed it despite a significant increase in extracellular fluid volume. This effect could be explained by alteration of vascular capacitance or interstitial compliance by substances produced by these cells (ANRL and APRL).

Hypertension seen in the chemical renal medullectomy model is associated with an increased peripheral vascular resistance. Reversal of two-kidney, one-clip hypertension in the rat is associated with a pronounced decrease in peripheral vascular resistance. This hypotensive effect of unclipping is attenuated by chemical renal medullectomy, indicating that the renomedullary vasodepressor mechanisms may be involved. In addition to changes in peripheral vascular resistance, previous studies have demonstrated alterations on the venous side of the circulation within 6 hours of unclipping with a reduction of MCFP indicating an increase in vascular capacity. Further, the renomedullary polar lipid has been shown to have α-blocking activity; if this action is nonselective, the renal medulla might therefore be involved in modulating capacitance vessel tone through venous α-adrenergic receptors. It was the object of these studies to investigate whether the removal of the renal medulla in a normal rat would have effects on the venous system and provide a possible explanation of the hemodynamic changes seen in the reversal model.

The method of measuring vascular capacitance in this study has the advantage in that it can be used in conscious unrestrained animals, thus avoiding any complicating effects of anesthetics on cardiac contractility and venous tone. There are, however, some possible sources of error. There are difficulties in obtaining a true measure of mean circulatory filling pressure. Circulatory arrest provokes intense cardiovascular reflex activity resulting in venoconstriction; thus, MCFP should be measured as far as possible before these powerful reflexes significantly alter venous tone. In lightly anesthetized rats, there is a delay of 6–8 seconds after pulmonary artery occlusion before there is a secondary rise in venous pressure. This secondary rise is abolished by spinal cord transection, indicating that it is due to the activation of vasoconstrictor reflexes. During measurement of MCFP, venous and arterial pressures reach plateaus within 4–5 seconds; thus, the measurements should be complete before significant interference from reflex venoconstriction.

Another problem is that the passive flow from the arteries to the veins is relatively slow after the heart has stopped pumping, and so the pressures throughout the circulatory system would not fully equilibrate within a few seconds. If no pump is used to transfer blood from the arterial side of the circulation to the venous side, the arterial and venous pressures seldom
converge exactly to a final equilibrium value. In the current study, the venous pressures were found to plateau at a value about 10–15 mm Hg below the lowest arterial pressure reached, suggesting that a small amount of blood remains trapped in the arterial system. To correct for this, it is possible to calculate the theoretical MCFP by calculating the additional change in venous pressure that would result from the transfer of this trapped blood from the arteries to the veins, taking into account the different compliances of the two sides of the circulation. To determine the arterial/venous compliance ratio in rats, Yamamoto et al measured the actual MCFP, venous plateau pressure, and final arterial pressure in anesthetized rats. The measurement of MCFP was then repeated, but blood was transferred from the arterial side to the venous side of the circulation by a reciprocal pump until arterial and venous pressure were equal, the final pressure was taken as the best estimate of the actual MCFP. The amount of blood transferred was less than 0.3 ml. The arterial/venous compliance ratio was then calculated as 1/60. This was found valid over a range of blood volumes. It is possible, in the hypertensive bromoethylamine-treated rats, that arterial compliance may be altered. However, because the volumes transferred to equilibrate the pressures were so small (less than 0.3 ml blood), the resulting venous plateau pressure was very close to the value obtained for MCFP by the blood transfer method, and the exact value of the arterial/venous compliance ratio was not very critical when MCFP was calculated. Using the arterial/venous compliance ratio value of 1/60, the small correction usually resulted in the addition of a maximum of 0.3 mm Hg to the venous plateau pressure. If arterial compliance was reduced and the arterial/venous compliance ratio was 1/100, the correction would have been even less at approximately 0.2 mm Hg. Thus, although not directly measured in these experiments, small changes in arterial compliance would have little effect on the measurements of MCFP.

Another possible source of error is that, with this method, the heart continues to beat during the measurement of MCFP, which may cause variable amounts of blood to shift from the cardiopulmonary to the systemic circulation. Because of this, theoretically, differences in compliance of the cardiopulmonary circuit or altered cardiac contractility may have influenced the values obtained for systemic MCFP. However, in previous studies in which MCFP was measured after inflation of a right atrial balloon in anesthetized rats, values obtained for MCFP with and without simultaneous occlusion of the aorta, which prevents blood shifts between thoracic and systemic circuits, were not significantly different (6.1 vs. 6.0 mm Hg). This suggests that the beating heart probably does not greatly affect the MCFP as measured in this model.

In constructing the blood volume–MCFP curve, blood volume changes were made as rapidly as possible (within 12 seconds), but it still remains a possibility that there was some activation of cardiovascular reflexes before measurements were completed. In addition, there may have been a degree of stress relaxation/reverse stress relaxation or transcapillary fluid shifts, although these should be quantitatively insignificant within 15 seconds. All of these factors would tend to rotate the MCFP–blood volume curve around the baseline data points, giving an overestimate of the vascular compliance. However, the values obtained for MCFP and compliance were similar to those obtained in other studies.

With these provisos, the present studies have clearly demonstrated no change in MCFP or vascular compliance in rats subjected to chemical renal medullectomy. The position of the vascular pressure–volume curve was not significantly shifted, indicating no significant change in vascular capacity (i.e., no change in venous tone). In view of the relatively small sample size (in statistical terms), there may have been inadequate power to detect subtle shifts, and therefore this study cannot be considered to have formally excluded a small shift of the curve to the right in the bromoethylamine-treated group. This finding would, if anything, strengthen our conclusions that, in this state of chronic renomedullary deficiency, the absence of the renomedullary vasodepressor mechanisms does not result in venoconstriction. It is, however, possible that after unclipping in experimental renovascular hypertension, renomedullary factors may have transient acute effects on venous tone. In summary, the hypertension in chemical renal medullectomy was not associated with any change in vascular capacitance. Thus, although the absence of the renomedullary vasodilator system may be responsible for an increase in arterial tone, as previously demonstrated, it appears that this effect is selective, as no alterations are seen on the venous side of the circulation.

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