Morphology of Renal Afferent Arterioles and Glomeruli, Heart Weight, and Blood Pressure in Primates

Karin Skov, Pavel Hamet, Jens Randel Nyengaard, and Michael J. Mulvany

In a Caribbean outbred population of African green monkeys (Cercopithecus aethiops), 5 to 10% of feral adults have elevated blood pressure (BP). We have investigated whether the increased pressure is associated with abnormal renal afferent arteriole structure or glomerular number. In seven young adult (aged 7 to 13 years) male monkeys with consistently high BP (mean BP, 111 mm Hg; ketamine anesthesia) and seven controls (mean BP, 81 mm Hg), the morphology of the renal vasculature has been analyzed in three cortical zones. In each animal, the left kidney vasculature was fixed while relaxed and at known intravascular pressure, and afferent arteriolar diameter and media cross-sectional area were estimated. The right kidney was perfusion-fixed and prepared for unbiased stereologic estimation of glomerular number and size.

No difference was found in afferent arteriole lumen diameter or media cross-sectional area, or in glomerular number or size, between the high BP group and controls. There was no difference in heart weight between the two groups, but there was a negative correlation between left ventricle heart weight and afferent arteriole diameter (controls: $r = -0.81$, $P = .025$; all animals: $r = -0.70$, $P = .005$, slope about 3.5% reduction in lumen diameter for 10% increase in heart weight). The results suggest that cardiac mass and renal afferent arteriole structure may be controlled by a common mechanism unrelated to BP measured in anesthesia. However, the lack of conscious measurements prevents conclusions as to whether this mechanism involves ambulatory BP.

Key Words: Arteriole, glomerulus, blood pressure, left ventricular mass, African green monkey.

In human arterial hypertension, the renal circulation has long been considered as playing a primary role, because the renal vascular resistance is increased in normotensive offspring of hypertensive parents. Similarly, in young prehypertensive spontaneously hypertensive rats (SHR) renal vascular resistance is increased and renal plasma flow and glomerular filtration rate are decreased. Furthermore, when blood pressure (BP) increases, glomerular filtration rate and renal plasma flow are normalized, whereas renal vascular resistance is further increased. Therefore, abnormal renal vascular resistance could play an important role in the pathogenesis of hypertension.

Support for this concept comes from our study and studies from other investigators who have demonstrated that the renal afferent arteriole is structurally narrowed in young adult (12-week-old) SHR. Moreover, in F₂ generation SHR × Wistar-Kyoto (WKY) rat hybrids a narrowed lumen diameter at age 7 weeks is a predictor of later development of high BP, indicating that structural narrowing of the renal afferent arteriole could be an important link in the pathogenesis of hypertension. Furthermore, it has been suggested and demonstrated in several rat strains, including SHR, that a reduced number of nephrons or a decrease in glomerular filtration area, or both, leads to systemic hypertension, even in the absence of high sodium intake. Reduced number of glomeruli, and with that a reduced number of afferent arterioles, might together with narrowing of the afferent arterioles in SHR cause high renal vascular resistance and thereby increase the systolic blood pressure (SBP) in SHR.

The relevance of the rat studies to humans is unclear. Experimental studies of non-human primates should, therefore, offer important advantages. Besides offering optimal

Received January 18, 2000. Accepted September 19, 2000.

From the Department of Pharmacology, University of Aarhus (KS, MJM), Stereological Research Laboratory (JRN), Aarhus University Hospital, Aarhus, Denmark, and Clinical Research Institute of Montréal (PH), Hôtel-Dieu Hospital of Montréal, Montréal, Canada.

This work was supported by grants from the Danish Medical Research Council, the Danish Heart Foundation and the Medical Research Council of Canada (MRC MT-10803).

Address correspondence and reprint request to Karin Skov, MD, PhD, Department of Pharmacology, Aarhus University, University Park 240, 8000 Aarhus C, Denmark; e-mail: karin@farm.au.dk

© 2001 by the American Journal of Hypertension, Ltd.

Published by Elsevier Science Inc.
sampling and tissue preparations, primates are biologically and phylogenetically closer to humans than the rodents commonly used for experimental studies of hypertension.

In a Caribbean outbred population of African green monkeys, 5 to 10% of feral adults have spontaneously elevated BP and data have previously suggested that these animals provide a useful primate model of human essential hypertension.10,11 In preparation for the present studies, we have investigated afferent arteriolar and glomerular morphology in three cortical zones (superficial, midcortical, and juxtamedullary) in a group of normotensive young adult monkeys.12 The study demonstrated a coefficient of variance in the afferent arteriole diameter measurements (3%), which was small enough to make it reasonable to compare hypertensive and normotensive monkeys with only few animals in each group (eg, there would be a 98% probability of detecting a 10% difference in afferent arteriole diameter with only six animals in each group). For glomerular number, the coefficient of variance was larger (19%), therefore there would be a lower probability of observing differences, unless these were large.

The purpose of the present study was to test the hypothesis that renal afferent arteriolar diameter and glomerular number is decreased in monkeys with elevated BP compared to monkeys with normal BP. Blood pressure was measured under anesthesia (ketamine) as local conditions precluded measurements in conscious animals.

**Methods**

The methods used have previously been described in detail.10–15 The following gives the essential points.

**Monkeys**

African green monkeys (none of which were used in our previous study12) were housed routinely outdoors in social groups at the Behavioral Sciences Foundation, St. Kitts, Caribbean.10,11 The animals were fed ad libitum with Purina primate chow, supplemented with fresh fruit and produce; water was available continually from an automatic watering system. Animal facilities and animal care routines were reviewed and approved by the Canadian Council on Animal Care. From this outbred population we selected eight young adult (aged 7 to 13 years) male monkeys with high blood pressure (HBP) and eight monkeys with normal BP (controls).

**Blood Pressure**

The monkeys were anesthetized with ketamine hydrochloride, 10 mg/kg, intramuscularly. While positioned on the left side, a cuff was placed on the right arm and a Doppler stethoscope was placed over the brachial artery, SBP and diastolic blood pressure (DBP) were measured,11 and mean blood pressure (MBP) was calculated as: DBP + 1/3 (SBP − DBP). All animals had been screened for BP on at least four (median, 17; range, 4 to 33) separate occasions over periods ranging from 0.12 to 7.06 years (median, 4.3 years). The last four BP measurements, taken over a period of between 6 weeks and 18 months, were used (Fig. 1).

**Surgery**

The monkeys were anesthetized with a mixture of ketamine hydrochloride (63 to 90 mg) and xylazine (1.4 to 2 mg) intramuscularly. The major abdominal vessels and the

![FIG. 1. Mean blood pressure (MBP) measurements of monkeys with high (HBP, □) and normal (control, ○) BP. "Before entry" shows mean of the MBP measurements (●, ■) before the first of the last four measurements (1 to 4).](https://academic.oup.com/ajh/article-abstract/14/4/331/187659)
left kidney were cleaned free of fat and connective tissue. The aorta was cannulated retrogradely with a polyethylene tube and advanced into the aorta just below the origin of the renal arteries. A hole was cut in the renal vein to let out blood and perfusate. The perfusate was a plasma solution given under constant pressure (100 mm Hg). After 3 min, the left renal artery was catheterized with a polyethylene tube. The left renal artery and vein were then cut and together with the kidney removed to a beaker, while transfusion with plasma solution under constant pressure was continued. Meanwhile the right kidney was perfused with a formaldehyde/glutaraldehyde solution through the aortic catheter at 100 mm Hg pressure for 10 min after 3 min of plasma perfusion.

For measurement of afferent arteriole diameter and morphology, the technique described previously was used to ensure that the vessels were relaxed and subjected to a transmural pressure of 100 mm Hg during fixation. The left kidney vasculature was, after 10 min of plasma perfusion, relaxed by changing the perfusate to a papaverine solution. Then the perfusate was changed to a silicone rubber solution (Microfil, MV-130, Canton-Bio-Medical Products, Boulder, CO) containing ultrasonically dispersed microspheres (diameter, ~12 μm). The perfusions were all done under constant infusion pressure, 100 mm Hg. The microspheres lodged in the glomerular capillaries, and together with a closing of the venous outflow, thus stopping the flow and increasing the pressure within the afferent arterioles to a constant level of 100 mm Hg. After the Microfil had hardened, the left kidney was removed and stored with the right kidney in primary fixative. Further kidney preparation and evaluation were performed in laboratories at the University of Aarhus.

All animals were decapitated immediately after initiation of plasma perfusion. The heart was taken out, rinsed, cleaned free of major vessels and fat, gently blotted and weighed. The atria were removed and the ventricles were weighed. Finally the right ventricle was removed and the left ventricle was weighed alone.

Estimation of Afferent Arteriolar Lumen and Media Wall

The left kidney (Microfil perfused) was prepared for histologic examination. In the sections, it was observed that the microspheres were trapped at the entrance of the glomeruli, confirming that all preglomerular vessels containing Microfil were inflated under the same pressure. Using two microscopes equipped with mirrors, the longest inner diameter was found, then the inner (ID) and outer (OD) diameter of the border between media and adventitia of the vessel, perpendicular to the longest axis, were measured. Media thickness and media cross-sectional area were calculated as $\frac{\text{OD} - \text{ID}}{2}$ and $\pi\left(\frac{\text{OD}/2}{2}\right)^2 - \left(\frac{\text{ID}/2}{2}\right)^2$, respectively. Only arterioles in apposition to a glomerulus and that fulfilled the criteria for being afferent arterioles were included (“distal arterioles,” see Skov et al). On average, 88 (range, 76 to 112) profiles of such arterioles were measured in each kidney.

Estimation of Glomerular Number and Size

Unbiased estimates of glomerular number were made using the dissector method in a known and predetermined fraction of the whole kidney. The right perfusion-fixed kidney was sliced and every fourth slice was sampled and embedded. The tissue was serially sectioned into 20-μm sections. Every 10th and 11th section was sampled and prepared for histologic examination. Using two light microscopes equipped with mirrors, the fields of vision were selected using a motor-driven stage mounted on the first microscope. A counting grid was applied to the first microscope, corresponding to an area of the section. The fraction of the section area covered by the grid was $f_a$. The total number of glomeruli in a kidney was estimated from $N_{\text{glomer}} = 4 \times 10 \times \left(\frac{1}{f_a}\right) \times \left(\frac{Q^2}{2}\right)$, where $Q$ and $10$ are the inverse of, respectively, the slice and the section sampling fraction. $Q^2$ is the number of counted glomeruli. More than 15 sections pairs were used and, on average, 242 (range, 154 to 321) glomeruli were counted in each kidney. This provided a glomerular number with a coefficient of error (= SEM/mean) of 6.6%.

The number-weighted mean glomerular volume $v_{\text{glomer}}$ is equal to the volume fraction of glomeruli in cortex divided by the numerical density of glomeruli in the counted cortical tissue estimated with a combination of fractionator sampling of glomeruli on the 20-μm thick sections and point counting of glomeruli on 2-μm thick sections. Cortical volume ($V_{\text{cortex}}$) was estimated using a combination of fractionator and point counting.

Statistical Analysis

All results are presented as mean ± SEM. Significance of differences between HBP and control parameters in Tables 1 and 2 were assessed by Student’s two-tailed $t$ test. In Table 2, differences in afferent arteriole lumen diameter and media cross-sectional area and glomerular size with respect to cortical localization were tested among all three groups by one-way ANOVA (modified by the Bonferroni method, INSTAT), separately, for HBP and controls. The null hypothesis that slope and intercepts equaled zero for left ventricle heart weight versus BP or renal morphologic parameters was evaluated with Student’s two-tailed $t$ test. $P < .05$ was considered statistically significant.

Results

Of the 16 monkeys used, 1 HBP and 1 control were excluded without further examination because of poor Microfil perfusion. Fig. 1 shows that MBP was consistently increased over the measurement period. The MBP in the last four measurements of HBP (111 ± 1 mm Hg) was 37% greater than that of controls (81 ± 3 mm Hg). The
SBP was 139 mm Hg versus 110 mm Hg, HBP and control, respectively. The DBP was 98 mm Hg versus 67 mm Hg, HBP and control, respectively. The was no difference in pulse pressure (SBP − DBP) in the two groups (41 mm Hg v 43 mm Hg, HBP and control, respectively). Table 1 shows that body weight was higher in HBP, but left ventricle (and right ventricle) heart weight did not differ (P > .29), leading to the same heart-to-body weight ratio in HBP and controls. No difference was found in ipsilateral kidney weights between the two groups of animals. However, right kidney weight corrected for body weight was lower in HBP. Table 2 presents the results of the structural estimates of the afferent arterioles and glomeruli. There was no difference in afferent arteriolar lumen diameter between the two groups (P = .42). Mean afferent arteriolar lumen diameter varied little within either group (coefficient of variation (CV) = SD/mean was below 7% in both HBP and controls, separately), but was largest in the juxtamedullary cortex in both groups. No difference in afferent arteriolar media area was found between the two groups (P = .38). There was no difference in total glomerular number or mean glomerular volume between the two groups. Mean glomerular volume was variable between animals, but largest in the juxtamedullary zone. No sclerotic glomeruli were found in any of the animals.

Table 1. Characteristics of monkeys with high and normal blood pressure

<table>
<thead>
<tr>
<th>Parameter</th>
<th>HBP (n = 7)</th>
<th>Controls (n = 7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>10.8 ± 2.7</td>
<td>9.9 ± 1.7</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>5.4 ± 0.2</td>
<td>4.8 ± 0.1</td>
</tr>
<tr>
<td>Left ventricular (LV) heart weight (g)</td>
<td>16.4 ± 0.8</td>
<td>14.8 ± 1.3</td>
</tr>
<tr>
<td>Right ventricular heart weight (g)</td>
<td>6.0 ± 0.5</td>
<td>5.7 ± 0.4</td>
</tr>
<tr>
<td>LV heart weight/body weight (g/kg)</td>
<td>3.0 ± 0.1</td>
<td>3.1 ± 0.3</td>
</tr>
<tr>
<td>Left kidney weight (Microfil casted) (g)</td>
<td>16.2 ± 0.1</td>
<td>15.9 ± 0.1</td>
</tr>
<tr>
<td>Right kidney weight (perfusion fixed) (g)</td>
<td>10.9 ± 0.1</td>
<td>11.3 ± 0.1</td>
</tr>
<tr>
<td>Right kidney weight/body weight (g/kg)</td>
<td>2.0 ± 0.1*</td>
<td>2.4 ± 0.1</td>
</tr>
</tbody>
</table>

HBP = monkeys with high blood pressure; controls = monkeys with normal blood pressure. Values are presented as mean ± SEM. *P < .05.

Table 2. Characteristics of afferent arterioles and glomerular parameters in three cortical zones in monkeys with high and normal blood pressure

<table>
<thead>
<tr>
<th>Zone</th>
<th>HBP</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left kidney</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AA lumen diameter (µm)</td>
<td>33.5 ± 0.8</td>
<td>33.9 ± 0.9</td>
</tr>
<tr>
<td>Superficial</td>
<td>31.5 ± 0.8</td>
<td>32.1 ± 0.8</td>
</tr>
<tr>
<td>Midcortical</td>
<td>33.7 ± 0.8</td>
<td>34.0 ± 0.9</td>
</tr>
<tr>
<td>Juxtamedullary</td>
<td>37.6 ± 1.0*†</td>
<td>37.8 ± 1.4*†</td>
</tr>
<tr>
<td>AA media area (µm²)</td>
<td>370 ± 11</td>
<td>387 ± 21</td>
</tr>
<tr>
<td>Superficial</td>
<td>348 ± 11</td>
<td>370 ± 13</td>
</tr>
<tr>
<td>Midcortical</td>
<td>362 ± 15</td>
<td>385 ± 25</td>
</tr>
<tr>
<td>Juxtamedullary</td>
<td>427 ± 28*</td>
<td>432 ± 32</td>
</tr>
<tr>
<td>Right kidney</td>
<td></td>
<td></td>
</tr>
<tr>
<td>V(cortex) (cm³)</td>
<td>62.2 ± 1.7</td>
<td>68.5 ± 3.1</td>
</tr>
<tr>
<td>Superficial</td>
<td>17.5 ± 0.6</td>
<td>19.1 ± 0.6</td>
</tr>
<tr>
<td>Midcortical</td>
<td>29.7 ± 0.8</td>
<td>34.0 ± 2.0</td>
</tr>
<tr>
<td>Juxtamedullary</td>
<td>15.0 ± 0.9</td>
<td>15.4 ± 0.9</td>
</tr>
<tr>
<td>N(glom)  (×10⁶)</td>
<td>113 ± 10</td>
<td>113 ± 8</td>
</tr>
<tr>
<td>Superficial</td>
<td>46 ± 6</td>
<td>43 ± 4</td>
</tr>
<tr>
<td>Midcortical</td>
<td>56 ± 5</td>
<td>59 ± 5</td>
</tr>
<tr>
<td>Juxtamedullary</td>
<td>11 ± 1</td>
<td>11 ± 1</td>
</tr>
<tr>
<td>vH(glom) (×10⁶ µm³)</td>
<td>3.1 ± 0.3</td>
<td>2.7 ± 0.5</td>
</tr>
<tr>
<td>Superficial</td>
<td>2.4 ± 0.3</td>
<td>2.2 ± 0.4</td>
</tr>
<tr>
<td>Midcortical</td>
<td>3.3 ± 0.3</td>
<td>2.7 ± 0.7</td>
</tr>
<tr>
<td>Juxtamedullary</td>
<td>5.3 ± 0.4*†</td>
<td>4.7 ± 0.9*†</td>
</tr>
</tbody>
</table>

AA = afferent arteriole; V(cortex) = estimate of cortical volume; N(glom) = estimate of glomerular number per kidney; vH(glom) = estimate of number-weighted mean glomerular volume; other abbreviation as in Table 1.

Values are expressed as mean ± SEM. There was no significant difference between HBP and controls. P < .05: *to superficial, †to midcortical.
pressure (P). In morbid obesity, there was a negative correlation between left ventricle heart weight and afferent arteriole lumen diameter (all animals, \(r = -0.58, P = .17\)) (Fig. 2). Similar correlations obtained when left ventricle heart weight was corrected for body weight (all animals, \(r = -0.60, P = .023\); controls, \(r = -0.76, P = .049\); HBP, \(r = -0.37, P = .42\)). There was no correlation between right ventricular heart weight and afferent arteriole diameter (all animals, \(r = -0.13, P = .66\); controls, \(r = -0.38, P = .41\); HBP, \(r = 0.04, P = .92\)). Body weight did not correlate to either heart weight, or MBP, or afferent arteriole lumen diameter for the two groups separately (\(P > .17\) in all cases). There was no correlation between left ventricle heart weight and glomerular number (HBP, \(r = 0.55, P = .20\); controls, \(r = -0.07, P = 0.87\), SBP \(P > .34\), DBP \(P > .29\) or pulse pressure \(P > .41\).

**Discussion**

This study presents a stereologic evaluation of kidney structures in non-human primates with either normal or elevated BP, as measured under anesthesia. On the basis of unbiased stereologic techniques, a negative correlation between left ventricle heart weight and afferent arteriole diameter was observed. Surprisingly, we found no difference in left ventricle heart weight, afferent arteriole lumen diameter, media cross-sectional area, or in glomerular number or size between the two groups.

**Blood Pressure**

The use of ketamine anesthesia to allow cuff measurements of BP in the present study is not ideal, as there is concern that such measurements are not true indications of normal BP. The cuff method as such has been validated by Kraft-Schreyer and Angelakos\(^1\) who showed that the present BP method using a Doppler ultrasound stethoscope and standard cuff with ketamine anesthesia correlates closely with direct femoral artery measurements under these conditions in African Green monkeys. Further evidence that the method can be used to identify hypertensive animals comes from a study of feral baboons.\(^2\) Here 456 animals were screened for BP using percutaneous arterial cannulation while the animals were under ketamine sedation and hypertensive and normotensive animals were identified. These animals were then used as a basis for a breeding program to produce hypertensive and normotensive progeny, in which the BP of the progeny was confirmed under unanesthetized conditions. Therefore, although it would have been preferable to have obtained unanesthetized BP measurements in the animals studied, it seems unlikely that the 37% greater measured BP in the HBP, consistently measured (Fig. 1), compared to controls did not reflect at least a degree of hypertension in the HBP. Whether the presented BP difference is due to biological differences or merely is caused by environmental factors such as stress factors (capturing, hierarchy in the social groups) is not known. Importantly, because local conditions prevented the use of telemetry techniques, we do not know the relation between the measurements made and conscious, ambulatory BP.

**Left Ventricle Heart Weight**

High BP increases the incidence of cardiovascular events and death. Before severe complications occur, less dramatic manifestations of target organ damage such as concentric left ventricular hypertrophy may develop. The increased pressure load and wall tension can lead to thickening of the left ventricular wall, which tends to normalize the elevated wall stress.\(^2\) One might, therefore, expect a close relationship between left ventricular mass and BP. It was surprising that in our study there was no difference in left ventricular mass between HBP and controls (with the given coefficient of variation of the measurements [21%], there was a 70% chance of finding a difference of 15%). Therefore, as in humans,\(^2\) there appear to be factors other than BP (genetic, humoral, environmental) that determine left ventricular mass in these animals. A possible reason for the lack of increase in left ventricular mass is the previously determined lower plasma angiotensin level in HBP compared to controls,\(^1\) which might counteract any effect of the pressure. Alternatively, as indicated above, the results are confounded by...
the BP measurements under anesthesia not reflecting ambulatory BP.

In the present study, despite the lack of correlation between BP (MBP, SBP, DBP, or pulse pressure) and left ventricle heart weight (or index), there was a negative correlation between left ventricle heart weight (and index) and afferent arteriolar lumen diameter among all animals. We are aware that pooling data from the two groups should be treated with caution. However, in controls alone, there was also a significant negative correlation between left ventricle heart weight and afferent arteriole diameter. Furthermore, there was a similar, but nonsignificant, relation among HBP. This suggests that cardiac mass and renal afferent arteriole structure are controlled by a common mechanism. Our experiments do not allow us to identify this mechanism.

Positive relations between heart weight and vascular structure have also been reported by other researchers. Thus, Muiesan et al. found a correlation between left ventricular mass index and carotid wall thickness estimated with B-mode ultrasound in a general population of unselected middle-aged subjects. Another study found a significant correlation between left ventricular mass index and media:lumen ratio (r = 0.74, P < .001) in resistance arteries from gluteal biopsies in patients with untreated hypertension.

Moreover, after 3 years of treatment with the angiotensin converting enzyme inhibitor lisinopril, the changes in left ventricular mass index were also correlated significantly with the media:lumen ratio (r = −0.51, P < .05) suggesting that the regression of left ventricular hypertrophy is also associated with smaller media:lumen ratio.

Whether left ventricular mass index is correlated with renal resistance vessel structure in humans remains to be determined. However, it has been assumed that the correlation between heart mass and resistance vessel structure is mediated through BP, this could not be confirmed in the present study.

Afferent Arteriolar and Glomerular Stereology

In the present study, there was a low coefficient of variation (7%) in the afferent arteriole diameter measurements, as found previously. It could be speculated that the low variation in lumen diameters between animals is due to the importance of strict control of renal vascular resistance, as resistance varies inversely with the fourth power of the radius according to Poiseulle’s equation. Furthermore, statistical power calculations indicate that the variation of the measurements in the present study was small enough to detect a potential difference of 15% in diameter with 98% probability. Nevertheless, although previous rat studies have pointed to a structural narrowing of the afferent arterioles, this was not supported in the present study on primates. In prehypertensive and hypertensive SHR, the afferent arteriole lumen diameter was consistently smaller compared to control rats, and it was not associated with growth of the surrounding media. In HBP, there was no difference in afferent arteriole lumen diameter and media area compared to controls. This could indicate that the findings in inbred hypertensive rats may not be comparable with hypertension in primates.

In the present study, kidney weight corrected for body weight was lower in HBP, but glomerular number and size were similar in the two groups. The estimates of glomerular number and mean glomerular volume were not different from those previously obtained in the normal monkey kidney using similar stereologic methods. Moreover, the reduced kidney weight-to-body weight ratio in the HBP corresponds to studies in recombinant inbred rat strains where kidney weight index correlated negatively to BP. However, although the reduced kidney weight index in the HBP could be taken in support of the Brenner et al. hypothesis, our finding that glomerular number was not decreased in HBP, whereas mean glomerular volume tended to be increased, suggests that glomerular number and size is not an important determinant of BP in these animals. This is supported by the large interindividual variation in glomerular number (here twofold) and mean glomerular volume (sixfold), which suggest that there could be a large extracapacity of nephrons in kidneys from birth; therefore, glomerular number and size are not tightly controlled parameters. Demonstrating a potential difference in glomerular number or total glomerular filtration area between hypertensive and normotensive primates would involve a larger number of subjects than used here.

In summary, this study provides the first evidence that narrowed renal afferent arterioles are correlated with increased left ventricle heart weight in primates, consistent with these being controlled by a common mechanism. However, as neither afferent arteriole diameter, nor glomerular number, nor heart weight correlated significantly with BP as measured under anesthesia, the results do not support a role for BP in this mechanism. The lack of measurements in conscious animals precludes conclusions as to whether the mechanism involves ambulatory BP.

Acknowledgments

We thank Dr. Roberta Palmour and Dr. Frank Ervin for providing animals, arranging for the blood pressure measurements, and for their help and encouragement. We are indebted to Mette Schandorff for excellent technical assistance.

References