Renal Blood Flow Dynamics and Arterial Pressure Lability in the Conscious Rat

Silene L.S. Pires, Christian Barrès, Jean Sassard, Claude Julien

Abstract—It is not known whether renal blood flow (RBF) is still autoregulated when the kidney is exposed to large transient blood pressure (BP) fluctuations such as those occurring spontaneously in conscious sinoaortic baroreceptor–denervated (SAD) rats. In this study, BP and RBF were simultaneously recorded in 8 SAD rats (2 weeks before study) and 8 baroreceptor-intact rats during ~3 hours of spontaneous activity. The kidney used for RBF recordings was denervated to prevent the interference of changes in renal sympathetic tone with autoregulatory mechanisms. In intact rats, RBF variability (coefficient of variation 9.1 ± 0.8%) was larger (P < 0.02) than BP variability (5.9 ± 0.2%). This was mainly because of slow changes in RBF that were unrelated to BP and also to a prominent oscillation of RBF of ~0.25-Hz frequency. Autoregulatory patterns were identified at frequencies <0.1 Hz and provided a modest attenuation of BP fluctuations. In SAD rats, RBF variability (12.4 ± 1.6%) was lower (P < 0.02) than BP variability (18.2 ± 1.1%). Autoregulation powerfully attenuated BP changes <0.1 Hz (normalized transfer gain 0.21 ± 0.02 in the 0.0015- to 0.01-Hz frequency range) but at the expense of an oscillation located at ~0.05 Hz that possibly reflected the operation of the tubuloglomerular feedback. Large transient hypertensive episodes were not translated into RBF changes in SAD rats. We conclude that autoregulatory mechanisms have an ample capacity to protect the kidney against spontaneous BP fluctuations in the conscious rat. This capacity is not fully used under normal conditions of low BP variability. (Hypertension. 2001;38:147-152.)

Key Words: autoregulation ■ blood pressure ■ rats ■ denervation

Although hemodynamic factors are likely to play a role in the progression of renal failure,1,2 the possible deleterious effects of an exaggerated blood pressure (BP) variability on the kidney have not yet been established. It is often proposed that autoregulatory mechanisms prevent acute changes in systemic BP from being transmitted to the glomerular capillary circulation.3 Most studies on renal blood flow (RBF) autoregulation have been performed in anesthetized rats, with the use of either induced4 or spontaneous5 changes in BP. In the conscious rat, RBF autoregulation has been described by measuring RBF responses to steady-state changes in BP variability (coefficient of variation 9.1 ± 0.8%) was larger (P < 0.02) than BP variability (5.9 ± 0.2%). This was mainly because of slow changes in RBF that were unrelated to BP and also to a prominent oscillation of RBF of ~0.25-Hz frequency. Autoregulatory patterns were identified at frequencies <0.1 Hz and provided a modest attenuation of BP fluctuations. In SAD rats, RBF variability (12.4 ± 1.6%) was lower (P < 0.02) than BP variability (18.2 ± 1.1%). Autoregulation powerfully attenuated BP changes <0.1 Hz (normalized transfer gain 0.21 ± 0.02 in the 0.0015- to 0.01-Hz frequency range) but at the expense of an oscillation located at ~0.05 Hz that possibly reflected the operation of the tubuloglomerular feedback. Large transient hypertensive episodes were not translated into RBF changes in SAD rats. We conclude that autoregulatory mechanisms have an ample capacity to protect the kidney against spontaneous BP fluctuations in the conscious rat. This capacity is not fully used under normal conditions of low BP variability.

Methods

Animals

Experiments were conducted on 16 male Sprague-Dawley rats (Iffa-Credo, L’Albresie, France) age 10 to 12 weeks at the time of the study. In 8 of these rats, surgical denervation of aortic and carotid sinus baroreceptors was performed 14 days before study according to a previously described technique.12,14 One week later, the rats were

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From the Département de Physiologie et Pharmacologie Clinique, CNRS UMR 5014, Institut Fédératif de Recherche Cardio-Vasculaire No. 39, Université Claude Bernard, Lyon, France.
Correspondence to C. Julien, CNRS UMR 5014, Faculté de Pharmacie, 8 av. Rockefeller, 69373 Lyon, France. E-mail julien@univ-lyon1.fr
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instrumented for RBF recording. Five days after probe placement, i.e., 2 days before the study, rats were instrumented with arterial and venous catheters. For long-lasting surgical procedures, animals were anesthetized with a mixture of acepromazine maleate (12 mg/kg IP) and ketamine hydrochloride (120 mg/kg IP). Catheterization was performed under halothane (1.5% to 2% in oxygen) anesthesia. After each intervention, rats received a single injection of penicillin G (40 000 IU IP). All experiments were performed in accordance with the guidelines of the French Ministry of Agriculture for animal experimentation.

**Chronic Instrumentation**

For RBF measurement, the left renal artery was exposed via a flank incision and carefully dissected to remove sympathetic fibers. An ultrasonic transit-flow probe (1RB, Transonic Systems) was placed around the artery and packed in polyester mesh to ensure proper alignment of the probe and vessel. The probe cable was secured to back muscles, routed subcutaneously, and exteriorized at the back of the neck. For BP measurement and drug administration, polyethylene catheters were inserted into the femoral artery and vein and led subcutaneously to exit at the back of the neck.

**Recording Protocol**

Instantaneous RBF was measured with a transit-time flowmeter (T106, Transonic Systems). BP was measured with a pressure transducer (TNP-R, Ohmeda) coupled to an amplifier (No. 13-4615-52, Gould). BP and RBF were fed simultaneously to a chart recorder (No. 8802, Gould) and to a computer via an analog-to-digital converter board (model AT-MIO-16E-10, National Instruments). By use of LabVIEW 5.0 software (National Instruments), both signals were sampled at 500 Hz. BP and RBF were continuously recorded for 4 to 5 hours. Then, phenylephrine hydrochloride (3 μg/kg IV) and sodium nitroprusside (10 μg/kg IV) were injected to assess baroreflex sensitivity. On completion of the experiments, rats were killed with an intravenous overdose of pentobarbital sodium. Kidneys were quickly weighed and frozen. To evaluate the effectiveness of renal denervation, the norepinephrine (NE) concentrations in the intact right kidney and in the denervated left kidney were measured separately by use of high-performance liquid chromatography with electrochemical detection.

**Data Analysis and Statistics**

BP and RBF 500-Hz data files were replayed, visually inspected to remove artifacts, and resampled at 50 Hz by computing mean values over consecutive 20-ms periods. The period used for analysis (3 hours 17 minutes) was split into segments of 65 536 points (≈21.8 minutes), which allowed us to analyze fluctuations down to 0.0015 Hz (≈10-minute periods). By using the fast Fourier transform analysis, as previously described,11 to allow for the comparison of BP and RBF variabilities, all data were normalized by their respective means before spectral analysis. The squared coherence function was calculated to quantify the strength of linear coupling between BP and RBF. With a 50% overlapping procedure, 17 periods could be analyzed, which yielded a significance threshold (P<0.05) for coherence of 0.22.17 Transfer function analysis, with BP as the input signal and RBF as the output signal, was also performed11 to calculate the normalized transfer gain and phase. The normalized gain is the ratio between the fractional variation in RBF and BP. When less than unity and when associated with a significant coherence, it allows us to locate frequencies at which hemodynamic patterns can reflect autoregulatory processes.8

Time-domain analysis consisted in the resampling of 500-Hz time series at 1 Hz (1-s moving averages). The overall variability of BP and RBF was estimated by calculating the coefficients of variation (CVs) over the entire 197-minute period. To evaluate the relative contribution of slow changes and of faster events to overall variability, the mean of CVs obtained over the 9 consecutive 21.8-minute periods was calculated (within-period CV, referred to as short-term variability), as well as the mean of the mean levels obtained over the same periods (between-period CV, referred to as long-term variability). The global trends of BP-RBF relations were evaluated by constructing the contour plots of the 3D frequency distributions of BP and RBF pairs.

Data are presented as mean±SEM. Comparisons between intact and SAD rats were made by the Mann-Whitney test for unpaired data. Within each group of rats, comparisons between indices of variability for BP and RBF were made by the Wilcoxon signed rank test.

**Results**

As indicated in Table 1, intact and SAD rats had similar body and kidney weights, and the NE content of the left denervated kidney was reduced to <10% of control values measured in the right kidney. The effectiveness of the SAD procedure was confirmed by the marked attenuation of heart rate reflex responses to drug-induced changes in BP.

**Time-Domain Analysis of Hemodynamic Variability**

SAD had no effect on the mean BP level but strongly increased all indices of BP variability (Table 2). In both intact and SAD rats, short-term BP fluctuations contributed more than did long-term changes to the overall BP variability (within-period CV versus between-period CV, P<0.05).

The mean level and overall variability of RBF did not significantly differ between intact and SAD rats (Table 2). The short-term RBF variability was increased in SAD rats compared with intact rats, whereas the long-term RBF variability was unaffected. In both intact and SAD rats, short- and long-term variabilities contributed almost equally to overall RBF variability.

<table>
<thead>
<tr>
<th>TABLE 1. Characteristics of Baroreceptor-Intact and SAD Rats</th>
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<tbody>
<tr>
<td><strong>Measurement</strong></td>
</tr>
<tr>
<td>Body weight, g</td>
</tr>
<tr>
<td>Kidney weight, g</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Kidney NE content, ng/g</td>
</tr>
<tr>
<td></td>
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<tr>
<td></td>
</tr>
<tr>
<td>Baroreflex sensitivity, bpm/mm Hg</td>
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<td></td>
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</tbody>
</table>

Values are mean±SEM. Baroreflex sensitivity was estimated as the ratio of peak changes in heart rate (in bpm) and mean BP during phenylephrine and sodium nitroprusside injections. P values refer to comparisons between SAD and intact rats.
TABLE 2. Hemodynamic Variability in Baroreceptor-Intact and SAD Rats

<table>
<thead>
<tr>
<th>Variable</th>
<th>Intact (n=8)</th>
<th>SAD (n=8)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean, mm Hg</td>
<td>111±1</td>
<td>113±6</td>
<td>0.401</td>
</tr>
<tr>
<td>CVs, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Global</td>
<td>5.9±0.2</td>
<td>18.2±1.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Within period</td>
<td>5.1±0.3</td>
<td>14.6±0.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Between period</td>
<td>2.8±0.3</td>
<td>10.0±1.7</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>RBF</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean, mL/min</td>
<td>11.3±1.3</td>
<td>11.1±0.9</td>
<td>0.674</td>
</tr>
<tr>
<td>Coefficients of variation, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Global</td>
<td>9.1±0.8*</td>
<td>12.4±1.6*</td>
<td>0.115</td>
</tr>
<tr>
<td>Within period</td>
<td>6.4±0.4*</td>
<td>8.8±0.7*</td>
<td>0.006</td>
</tr>
<tr>
<td>Between period</td>
<td>6.5±1.0*</td>
<td>8.6±2.0</td>
<td>0.529</td>
</tr>
</tbody>
</table>

*P<0.05 for indices of RBF variability vs corresponding indices of BP variability within each group of rats.

Values are mean±SEM. P values refer to comparisons between SAD and intact rats.

(r^2=0.14±0.04 and 0.19±0.08 in intact and SAD rats, respectively).

Figure 1 exemplifies the lack of an apparent relationship between BP and RBF variabilities in intact rats. In SAD rats, the 3D frequency distributions usually showed a horizontal stretching, suggesting that autoregulatory processes were operating. In particular, high BP values were not accompanied by renal hyperemia. As a systematic evaluation of these individual observations, the mean of 1% of highest BP values was calculated in each SAD rat, together with the mean of corresponding RBF values. The top 1% of BP values averaged 174±10 mm Hg, and the corresponding average RBF was 10.5±1.0 mL/min, which does not significantly differ from the mean value calculated over the whole data sets. Moreover, linear regression analysis did not disclose any trend of RBF changes during these hypertensive episodes (r^2=0.03±0.01 for RBF versus BP, n=118 data pairs in each SAD rat).

Frequency-Domain Analysis of Hemodynamic Variability

Spectral analysis revealed that in SAD rats, exaggerated BP variability was confined to frequencies <0.1 Hz (Figure 2 and Table 3), whereas increased short-term RBF variability was mainly secondary to larger fluctuations in the 0.01- to 0.1-Hz frequency band, including an oscillation centered at 0.047±0.002 Hz.

The comparison of BP and RBF variabilities showed that in intact rats, most of the extra RBF variability was concentrated in the 0.1- to 1-Hz range (Table 3); this occurrence was especially due to the presence of a prominent oscillation centered at 0.24±0.01 Hz (Figure 2). In SAD rats, BP variability exceeded RBF variability up to 0.1 Hz. At higher frequencies, RBF fluctuations were larger than those of BP; this occurrence was especially due to an oscillation centered at 0.23±0.01 Hz.

At low frequencies, coherence between RBF and BP was low, especially in intact rats, in which it fell below the significance threshold at ~0.01 Hz. Then, coherence rose progressively as a function of frequency but showed a clear peak at 0.050±0.005 Hz in SAD rats that was not observed in intact rats (Figure 2).

In both groups of rats, the normalized transfer gain was <1 in the very-low-frequency range (Figure 3). Transfer gain was lower in SAD than in intact rats (0.21±0.02 versus 0.40±0.05, P<0.01) in the 0.0015- to 0.01-Hz frequency band, suggesting more efficient autoregulation of RBF. In the 0.01- to 0.1-Hz frequency band, both gain functions showed a peak at ~0.05 Hz. At higher frequencies, gain increased steadily, became >1 beyond 0.15 Hz, and showed a peak at ~0.25 Hz in both intact and SAD rats. When expressed in decibels [20 log(gain)], both gain functions showed a linear portion between 0.1 and 0.2 Hz, the slope of which did not differ between intact (32.8±3.7 dB/decade) and SAD (29.0±3.4 dB/decade) rats.

Because of low coherence, the phase function could not be reliably estimated at low frequencies (<0.05 Hz) in intact rats. In SAD rats, phase angles were consistently positive starting from 0.005 Hz, indicating that RBF fluctuations led BP fluctuations. Above 0.05 Hz, both phase functions were almost identical and showed a maximum at ~0.15 Hz (Figure 3).

Discussion

The main finding of the present study is that SAD strongly increased spontaneous BP variability while minimally affecting RBF variability in conscious rats. In particular, acute hypertensive episodes that frequently occurred in SAD rats were not accompanied by parallel RBF variations. Such a constancy of RBF at high BP levels points to a strict...
proportionality between fractional changes in renal vascular resistance and BP. Theoretically, this proportionality can result either from autoregulatory mechanisms or from vasoconstrictor influences acting on both the renal and systemic circulations. In SAD rats, the pressor component of BP lability is primarily mediated by the sympathetic nervous system. Because the kidney used for RBF recordings was denervated, it sounds logical to propose that autoregulatory mechanisms were mainly responsible for the maintenance of RBF during BP surges in SAD rats. However, it cannot be excluded that an increase in circulating catecholamines contributed to the renal vasoconstriction during hypertensive episodes.

With the aid of spectral analysis, it was possible to discern the types of hemodynamic patterns that could reflect the operation of autoregulatory mechanisms. At high frequencies, fluctuations of RBF, including those linked to the cardiac and respiratory cycles, constantly exceeded fluctuations of BP. This classic observation is usually referred to as a compliance effect involving the passive elastic behavior of large arteries. At 0.2 to 0.3 Hz, the RBF spectra exhibited a prominent peak whose central frequency did not differ between intact and SAD rats. However, the peak was larger in intact than in SAD rats, probably as a consequence of entrainment by the BP Mayer waves (≈0.4 Hz) that were absent in SAD rats. The 0.25-Hz oscillation of RBF is often regarded as the signature of the fast component of RBF autoregulation, ie, the myogenic response of afferent glomerular arterioles. This would imply that myogenic autoregulation shows the properties of an underdamped, second-order, high-pass filter with a resonance frequency located at ≈0.25 Hz. Such filters are characterized by a linear increase in gain (when expressed in decibels) below their resonance frequency and a phase lead around this frequency. Although the features of the transfer functions between BP and RBF were indeed consistent with the presence of a high-pass filter, it is impossible to conclude as to whether this filter generates a resonant oscillation at 0.25 Hz. The linear portion of the gain function (0.1 to 1 Hz) had a slope of ≈30 dB/decade, which is consistent neither with a first-order (20 dB/decade) nor a second-order

<table>
<thead>
<tr>
<th>Spectral Power</th>
<th>Intact (n=8)</th>
<th>SAD (n=8)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP spectral powers, nu^2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.0015–3 Hz</td>
<td>32±5</td>
<td>227±27</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>0.0015–0.01 Hz</td>
<td>15±2</td>
<td>169±23</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>0.01–0.1 Hz</td>
<td>6±1</td>
<td>47±5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>0.1–1 Hz</td>
<td>11±2</td>
<td>9±1</td>
<td>0.623</td>
</tr>
<tr>
<td>1–3 Hz</td>
<td>0.8±0.1</td>
<td>1.1±0.2</td>
<td>0.172</td>
</tr>
<tr>
<td>RBF spectral powers, nu^2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.0015–3 Hz</td>
<td>51±6*</td>
<td>81±10*</td>
<td>0.027</td>
</tr>
<tr>
<td>0.0015–0.01 Hz</td>
<td>16±2</td>
<td>29±7*</td>
<td>0.074</td>
</tr>
<tr>
<td>0.01–0.1 Hz</td>
<td>4±1</td>
<td>21±4*</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>0.1–1 Hz</td>
<td>28±5*</td>
<td>27±3*</td>
<td>0.674</td>
</tr>
<tr>
<td>1–3 Hz</td>
<td>3±1*</td>
<td>4±1*</td>
<td>0.462</td>
</tr>
</tbody>
</table>

Values are mean±SEM. nu indicates normalized units. Spectral powers were calculated by integration over selected frequency bands as indicated by their boundaries. The upper frequency limit was set at 3 Hz to exclude from calculations the very large oscillations related to the cardiac cycle (~5–8 Hz; see Figure 2). P values refer to comparisons between SAD and intact rats. *P<0.05 for indices of RBF variability vs corresponding indices of BP variability within each group of rats.
time delays associated with these dynamic components.\textsuperscript{8} inv
olved but also because of the presence of various fixed filter, probably because several dynamic components were 
from that expected from the operation of a single high-pass filter. This is probably because of 
back system.\textsuperscript{4} Similarly, the phase function largely deviated 
and possibly the more slowly acting tubuloglomerular feed-

tuations may occur secondarily to the interference of noise sources, ie, factors affecting RBF independently of BP. Based on our arbitrarily defined time windows (21.8 minutes), both time-
frequency-domain analyses indicated that factors acting with slow time constants powerfully affected RBF independently of BP in both intact and SAD rats. These factors might include fluctuations in the metabolic demand,\textsuperscript{19} intrarenal release of vasoactive factors, or release in the circulation of hormones acting specifically on the renal vasculature.\textsuperscript{20} In SAD rats (compared with intact rats), despite robust autoregulatory responses, large low-frequency BP fluctuations were likely to induce coherent RBF fluctuations that were less easily superseded by other factors of variability. This was especially evident in the 0.01- to 0.1-Hz frequency band, at which coherence remained significant. Even slower changes in BP appeared to be actively buffered by autoregulatory mechanisms in the kidney of SAD rats, inasmuch as long-term variability of RBF was unaltered despite a 3- to 4-fold increase in long-term BP variability. The fact remains that in SAD rats, RBF variability was increased in a frequency range relevant to renal excretory function and renin secretion.\textsuperscript{21} Whether these increased RBF fluctuations have long-term renal and/or systemic effects remains to be determined.

We conclude that in the normotensive SAD rat, autoregulatory processes powerfully attenuate the impact of BP fluctuations on renal hemodynamics. This observation helps to explain why SAD rats do not develop glomerular injury, at least until 6 weeks after denervation.\textsuperscript{22} Taken together, these findings indicate that BP variability per se is probably not detrimental to the kidney, as long as autoregulatory mechanisms are normally functioning.

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References


(40 dB/decade) high-pass filter. This is probably because of the impingement of several regulatory mechanisms in this frequency band, ie, compliance effects, myogenic responses, and possibly the more slowly acting tubuloglomerular feedback system.\textsuperscript{4} Similarly, the phase function largely deviated from that expected from the operation of a single high-pass filter, probably because several dynamic components were involved but also because of the presence of various fixed time delays associated with these dynamic components.\textsuperscript{8} Alternatively, spontaneous rhythmic activity of the renal microcirculation (vasomotion) might explain the presence of an oscillation of RBF at $\omega_{0.25}$ Hz. Cyclic changes in the diameter of the afferent arteriole have indeed been observed at $\omega_{0.2}$ Hz in the in vitro blood-perfused rat juxtamedullary nephron preparation.\textsuperscript{18}

From the transfer functions, it can be concluded that in both intact and SAD rats, myogenic autoregulation effectively stabilizes RBF at $<0.1$ Hz, which is in accordance with previous studies in anesthetized rats.\textsuperscript{5,8} Both transfer functions showed a local maximum at $\omega_{0.05}$ Hz. It has been suggested that this peak in gain results from instability in the tubuloglomerular feedback control loop that is due to the presence of significant time delays in the loop.\textsuperscript{8} In SAD but not in intact rats, this increase in gain was associated with a peak in the coherence and RBF spectra that was due to larger BP oscillations at this frequency. At lower frequencies, the transfer gain was significantly lower in SAD than in intact rats. This finding must be interpreted with caution because coherence was not significant at these frequencies in most intact animals. Low coherence indicates nonlinear coupling and/or a lack of coupling. Nonlinear coupling can occur when the input power is large enough to drive the system outside its range of linear operation. This was certainly not the case in this experiment, because both input power (amplitude of BP fluctuations) and coherence were higher in SAD than in intact rats at low frequencies. Uncoupling of RBF from BP fluctuations may occur secondarily to the interference of noise sources, ie, factors affecting RBF independently of BP. Based on our arbitrarily defined time windows (21.8 minutes), both time- and frequency-domain analyses indicated that factors acting with slow time constants powerfully affected RBF independently of BP in both intact and SAD rats. These factors might include fluctuations in the metabolic demand,\textsuperscript{19} intrarenal release of vasoactive factors, or release in the circulation of hormones acting specifically on the renal vasculature.\textsuperscript{20} In SAD rats (compared with intact rats), despite robust autoregulatory responses, large low-frequency BP fluctuations were likely to induce coherent RBF fluctuations that were less easily superseded by other factors of variability. This was especially evident in the 0.01- to 0.1-Hz frequency band, at which coherence remained significant. Even slower changes in BP appeared to be actively buffered by autoregulatory mechanisms in the kidney of SAD rats, inasmuch as long-term variability of RBF was unaltered despite a 3- to 4-fold increase in long-term BP variability. The fact remains that in SAD rats, RBF variability was increased in a frequency range relevant to renal excretory function and renin secretion.\textsuperscript{21} Whether these increased RBF fluctuations have long-term renal and/or systemic effects remains to be determined.

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In the article “Renal Blood Flow Dynamics and Arterial Pressure Liability in the Conscious Rat” by Pires et al (Hypertension. 2001;38:147–152), a color version of Figure 3 was incorrectly used. Below is the correct black and white version.

**Figure 3.** Group-average transfer functions from BP to RBF in 8 baroreceptor-intact rats (gray traces) and 8 SAD rats (black traces). The frequency scale was limited to 1 Hz to emphasize the frequency band at which autoregulatory processes are expected to operate. Standard error lines have been omitted for legibility. The horizontal dotted line in the gain spectra shows the limit above which fractional variations of RBF exceed those of BP.

(Hypertension. 2001;38:e12.)

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