Moment-to-Moment Characteristics of the Relationship Between Arterial Pressure and Renal Interstitial Hydrostatic Pressure

Marina Komolova, Michael A. Adams

Abstract—The kidney is a key controller of the long-term level of arterial pressure, in part through pressure-natriuresis. Although direct coupling of changes in renal arterial pressure to renal interstitial hydrostatic pressure (RIHP) and consequent sodium excretion is well established, few studies have characterized the moment-to-moment aspects of this process. These studies characterized the short-term hemodynamic component of pressure-natriuresis in vivo before and after autonomic nervous system and renin-angiotensin system inhibition. Changes in RIHP were determined over a range of renal arterial pressures in Wistar rats receiving no treatment, a ganglionic blocker (hexamethonium; 20 mg/kg per hour IV), or an angiotensin II type 1 receptor blocker (losartan; 10 mg/kg per hour IV). After a series of changes in renal arterial pressure, a delay of only ≈1 second was found for the onset of RIHP responses that was independent of the stimulus magnitude and neurohumoral manipulation; however, completion of the full RIHP response was within ≈15 seconds for renal arterial pressure changes of ≤30 mm Hg. The overall slope of the renal arterial pressure-RIHP relationship (0.09±0.01) was also not affected by autonomic nervous system and renin-angiotensin system inhibition despite decreasing renal arterial pressure (↓40% and ↓28%, respectively). Separate assessment of this relationship above and below the prevailing arterial pressure revealed that the pressor versus the depressor portion was blunted (P<0.001), a difference that was abolished after autonomic nervous system and renin-angiotensin system inhibition. The results suggest that spontaneous changes in arterial pressure are coupled to moment-to-moment changes in RIHP over a wide range of pressures, emphasizing a likely role for the dynamic component of the renal arterial pressure-RIHP relationship in the modulation of sodium excretion and, hence, arterial pressure. (Hypertension. 2010;56:650-657.)

Key Words: kidney ■ hydrostatic pressure ■ arterial pressure ■ natriuresis ■ renin-angiotensin system ■ autonomic nervous system

The kidney plays an important role in regulating blood volume and arterial pressure, in particular via a pressure-dependent regulation of sodium and water balance, a process known as pressure-natriuresis.1–4 Pressure-natriuresis acts through a final common pathway that directly couples renal arterial pressure (RAP) with renal interstitial hydrostatic pressure (RIHP), leading to changes in downstream sodium excretion (ie, increases in RIHP lead to enhanced sodium excretion).4–8 Given that pressure-natriuresis has been regarded as a “long-term” regulatory mechanism, it is not surprising that numerous studies have investigated the specific relationship between RAP and RIHP over a prolonged time course. Specifically, these studies induced graded step changes in RAP for ≥30 minutes and then recorded “steady-state” changes in RIHP and associated changes in sodium excretion.9–17 However, such assessments may not reflect true in vivo control of arterial pressure, because arterial pressure in conscious animals does not change from one steady-state to another but rather fluctuates spontaneously in varying degrees of magnitude around an operating point (baseline) and over a wide range of time frames (frequencies).8 For example, oscillations in arterial pressure assessed over an acute time frame (Figure 1A) tend to be more variable and of greater magnitude than fluctuations assessed over a prolonged time-frame (Figure 1B). This suggests that kidneys are exposed to greater arterial pressure variations over a shorter time frame than have been examined using conventional pressure-natriuresis methodologies. Interestingly, only one study to date has described a statistically significant moment-to-moment correlation between spontaneous arterial pressure oscillations and RIHP within frequency windows of 1.00 to 0.01 Hz.8 This same group also found that urine flow, a variable downstream from RIHP, responded to changes in arterial pressure within ≈6 seconds.18 Because changes in RIHP are an intermediate step in pressure-natriuresis, there must be a shorter delay between arterial pressure and RIHP; however, the exact time course is yet to be elucidated.

Received December 30, 2009; first decision January 18, 2010; revision accepted July 20, 2010.
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© 2010 American Heart Association, Inc.
Hypertension is available at http://hyper.ahajournals.org DOI: 10.1161/HYPERTENSIONAHA.109.149633
In addition, despite that previous studies using the conventional “graded step” methodology describe the overall relationship between components of pressure-natriuresis (ie, RIHP and sodium excretion) and arterial pressure as linear,9,20 a more physiological characterization of the RAP-RIHP relationship (ie, alternating acute pressor and depressor stimuli around the arterial pressure operating point) is warranted. In fact, Steele et al19 suggest that acute increases above the arterial pressure operating point are dealt with more effectively than decreases with respect to urine flow. Whether such a differential response to pressor and depressor stimuli exists with regard to RIHP has yet to be determined.

Lastly, because of the duration of the conventional graded step methodology, the influence of potential compensatory neurohumoral factors was typically controlled for by various methods (eg, hormonal mixtures).9–17 However, there has not been a systematic assessment of how various neurohumoral systems impact on the RAP-RIHP relationship.

Thus, given that in conscious, freely moving animals arterial pressure oscillations occur on a moment-to-moment basis, we sought to determine the renal hemodynamic responses (ie, RIHP) associated with such changes. More specifically, our objectives were as follows: (1) to examine the time course of changes in RIHP after acute changes in arterial pressure; (2) to determine whether RIHP responds differentially and linearly to acute changes in arterial pressure above and below its operating point; and (3) to assess whether neurohumoral systems, specifically the renin-angiotensin system (RAS) and autonomic nervous system (ANS), impact on the acute RAP-RIHP relationship.

**Methods**

**Animals**

Male Wistar rats (n = 40; 300 to 400 g; 11 to 12 weeks old) were obtained from Charles River. Rats were housed individually (21 ± 1°C; 12-hour light/dark cycle) and acclimatized for ≥96 hours before experimentation. All of the rats were provided with standard rat chow (Purina; 0.4% Na+) and water ad libitum. All of the procedures followed guidelines of the Canadian Council on Animal Care and were approved by the Queen’s University Animal Care Committee.

**Surgical Preparation**

Rats were anesthetized with rogarsetic (ketamine; 30 mg/kg body weight IP; Rogar/STB) and inactin (thiobarbital sodium; 100 mg/kg body weight IP; Sigma-Aldrich). Body temperature was monitored using a thermistor (model 402; Yellow Springs Instruments) and maintained at 37 ± 0.5°C using a temperature controller (model 73A; Yellow Springs Instruments) connected to a heating pad and lamp. Additional anesthetic was given, as necessary, throughout experiment. Rats were tracheostomized (PE-240), and 95% O2/5% CO2 was passively blown over the intake to assist respiration. A midline abdominal incision was made and the right kidney removed. A saline-filled catheter was introduced into the inferior vena cava (at the level of right iliofemoral vein) for continuous infusion of saline (0.9% NaCl) at 33 μL/min per 100 g of body weight via a syringe pump (KD Scientifica 220) to compensate for fluid loss during surgery. A second catheter was introduced just above the other for drug administration.

The modified in vivo assessment of renal vascular properties21,22 was based on the technique described by Roman and Cowley16 and the direct determination of RIHP according to Ott et al15,23; however, the neurohumoral control systems were left intact. The superior mesenteric artery was catheterized with heparinized saline-filled (50 IU/mL) phycoerythrin 50 tubing for continuous measurement of arterial pressure at the level of the renal artery (hereinafter referred to as RAP). Water-filled silastic balloon cuffs were placed between the celiac and superior mesenteric arteries and 10 mm distal from the left renal artery. The cuffs were connected to a syringe with a 3-way stopcock via a water-filled line, which allowed for manual manipulation and control of RAP over a wide range of pressures (ie, pressor and depressor).9 An electrosurgical unit (Elmed ESU30; Elmed Incorporated) was used to create a 3-mm hole into the longitudinal axis of the left kidney for insertion of a heparinized saline-filled (50 IU/mL) catheter for RIHP measurements (Tygon tubing; ID 0.64 mm; fitted with 2- to 3-mm-long polyethylene matrix with 15- to 45-μm pores; Forex).21–23 The catheter was held in place with cyanoacrylate glue. RAP and RIHP were continuously monitored via pressure transducers (CDX3, Cobe) connected to a PowerLab/8s...
not achieve the desired target within the attached syringe and using the stopcock to keep the inflated cuff desired RAP within the real-time RAP signal, an investigator manually generated a tudes around the operating point (baseline). Through visualization of sequential pressor and depressor changes in RAP of various magni-
tions of RAP (1 to 60 seconds). These manipulations consisted of RAP-RIHP relationship was determined via short-term manipula-
tions. The pressure difference between corresponding arrows (ie, black for RAP and gray for RIHP) represents values used to determine the slope of the RAP-RIHP relationship. The time delays between the initial RAP change and onset of RIHP response (TI) and that between achieving steady state for RAP and RIHP (Ts) were characterized as shown. C, The rate of change (DP/dt) in RAP and RIHP after a pressor manipulation of ~26 mm Hg, which peaked at 8 seconds and achieved steady state (ie, consecutive 0 values) at 15 and 23 seconds, respectively. D, The DP/dt in RAP and RIHP after a depressor manipulation of ~24 mm Hg, which peaked at 8 and 9 seconds, respectively, and achieved steady state at 11 and 20 seconds, respectively. Data displayed are means obtained every second. (ADInstruments) data acquisition system with Chart version 4.2.2 software.

Experimental Protocol
After surgery, an equilibration period of ~15 minutes was allowed before recording steady-state baselines of RAP and RIHP. The acute RAP-RIHP relationship was determined via short-term manipulations of RAP (1 to 60 seconds). These manipulations consisted of sequential pressor and depressor changes in RAP of various magnitudes around the operating point (baseline). Through visualization of the real-time RAP signal, an investigator manually generated a desired RAP within ~5 seconds by inflating the appropriate cuff via the attached syringe and using the stopcock to keep the inflated cuff static at the desired RAP target. RAP changes that overshot or did not achieve the desired target within ~5 seconds were not included in the analysis.

The groups were as follows: group 1 (n=19) received no treatment; group 2 (n=14) was given a ganglionic blocker (hexamethonium; 20 mg/kg per hour IV); and group 3 (n=7) was given an angiotensin II (Ang II) type 1 receptor blocker (losartan; 10 mg/kg per hour IV) after an Ang II (250 ng/kg bolus IV) pretreatment. Ang II was also administered after losartan treatment to ensure sufficient blockade of Ang II type 1 receptors. In addition, control values were also obtained in half of the hexamethonium- and losartan-treated animals, such that paired analysis could be conducted.

On completion of the experiment, hematocrit was determined and rats were euthanized via exsanguination. Lastly, the left kidney was removed, and the position of the RIHP catheter was verified at the cortical-outer medullary junction.

Data Analysis
Baseline RAP and RIHP were determined by averaging the 60-
second period before starting RAP manipulations. The slopes were determined from steady-state values of RAP and RIHP after manipulations of RAP (Figure 2A and 2B). The time delay between the initial RAP change and the onset of RIHP response was characterized as the difference between the first detectable change from the operating point after a manipulation (Figure 2A and 2B). The time delay between achieving steady-state RAP and RIHP was also characterized as the time difference when RAP and RIHP approach a plateau (Figure 2A and 2B). Although, not calculated for all of the data, we determined that when the variables reached a plateau, the rate of change at this point was not significantly different from the rate of change at steady state (Figure 2C and 2D). Recovery from stimulus data (ie, point at which cuff was deflated) was assessed in a similar fashion.

Statistical Analysis
All of the statistical calculations were performed and graphs constructed using GraphPad Prism 5 and/or Microsoft Excel. Linear regression analysis was used to calculate the overall, pressor, and depressor relationships between RAP and RIHP.24 Statistical significance of differences in values measured among untreated, hexamethonium-treated, and losartan-treated rats was determined using a 1- or 2-way ANOVA followed by a Newman-Keuls or Bonferroni post hoc test, where appropriate. All of the data are presented as mean±SEM. *P<0.05 was considered statistically significant.

Results
Baseline Characteristics and Intrarenal Hemodynamic Variables
A summary of body weights and hematocrit and intrarenal hemodynamic parameters assessed in control and pharmaco-
logically manipulated animals is presented in the Table. Body weights and hematocrit were not significantly different across groups. In the control group, there was no difference between the baseline mean RAP and RIHP at the beginning of the experiment (94.2±3.3 and 7.8±0.8 mm Hg, respectively) and the average RAP and RIHP throughout the experiment (94.1±3.1 and 7.8±0.8 mm Hg, respectively). The mean RAPs after

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<td>Treatment</td>
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<td>Body weight, g</td>
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<td>Hematocrit, %</td>
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<td>Percentage of change in RAP from baseline, %</td>
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*P<0.001 vs control group (1-way ANOVA followed by a Newman-Keuls post hoc test used to compare between groups).
†P<0.05 vs hexamethonium-treated group (1-way ANOVA followed by a Newman-Keuls post hoc test used to compare between groups).
hexamethonium and losartan treatments were ≈40% (54.4±3.3 mm Hg) and 28% (65.2±3.7 mm Hg) lower than the pretreatment baseline RAPs, respectively (P<0.001). In addition, there was a significant 36% decrease in mean RIHP after hexamethonium treatment (3.9±0.8 mm Hg; P<0.01), and a 15% decrease after losartan treatment (5.2±1.1 mm Hg).

As part of protocol, the losartan group was given a pressor dose of Ang II before and after losartan administration to confirm sufficient blockade of Ang II type 1 receptors. Thus, whereas brief pretreatment with Ang II produced a rapid but transient increase in RAP (23%) and a decrease in RIHP (~15%), after losartan, Ang II did not produce any significant changes (data not shown).

In Vivo Characterization of the Acute RAP-RIHP Relationship
As described, a modified in vivo protocol was used to assess the acute RAP-RIHP relationship with all of the neurohumoral systems left intact. In all of the animals, there was a clear, positive relationship between RAP and RIHP (Figures 2 and 3). Similar to previous studies showing a linear RAP-RIHP relationship over a prolonged time frame, the overall acute RAP-RIHP responses were also found to be correlated linearly (ie, response to stimulus slope was 0.09±0.005; R²=0.94; Figure 3A). In addition, the acute RAP-RIHP relationship during the recovery phase from each manipulation had a similar slope (ie, recovery from stimulus slope was 0.090±0.006; R²=0.95; Figure 3B). However, separate analysis of the pressor and depressor components of this relationship revealed that the pressor slope was substantially lower (~43%) than the depressor slope (P<0.001; Figure 4A). A similar blunted pattern was present in the recovery phase after pressor manipulations (~39%; P<0.001; Figure 4B).

In all cases, RIHP was found to respond rapidly to changes in RAP (Figure 2). That is, after a change in RAP, initial changes in RIHP occurred with an average delay of only 1.1±0.2 seconds. This response time was independent of the magnitude or type of change (pressor or depressor) in RAP (Figure 5A). Furthermore, the RIHP response time was the same for both the response to and recovery from RAP manipulations (Figure 5B). To characterize whether there was a lag in the overall RAP-RIHP time course, the time difference between achieving steady state in RAP and RIHP was assessed. There was a clear impact of the magnitude of RAP change on the time difference to achieve steady state between RAP and RIHP, because the time delay was found to be ~6 seconds for RAP changes of ±0 to 10 mm Hg, whereas for greater pressure changes (±20 to 30 mm Hg) the delay was >2-fold longer (Figure 6A).

Role of Neurohumoral Control Systems on the Acute RAP-RIHP Relationship
As for the controls, the overall RAP-RIHP relationship was found to be linearly correlated after ANS and RAS inhibition (Figure 3). In addition, there were no differences in the slopes for responses to stimulus (hexamethonium=0.09±0.005, R²=0.91; losartan=0.07±0.004, R²=0.95) and the recovery from stimulus (hexamethonium=0.08±0.004, R²=0.92; losartan=0.07±0.005, R²=0.94) for both between group comparisons and with respect to controls (Figure 3A and 3B). In addition, paired analysis of data from animals that were initially controls but then received one of the treatments revealed that there was no significant effect of the treatments on the overall slope of the response or recovery data (data not shown). However, the significant difference between the slopes of the pressor and depressor arms of RAP-RIHP relationship in controls was abolished after lowering the operating point with hexamethonium and losartan treatments (Figure 4A through 4C). In fact, this treatment-induced equalization of the pressor and depressor aspects of the RAP-RIHP relationship occurred for both the response and recovery slopes.

The time course for RIHP to respond to changes in RAP was assessed among the hexamethonium and losartan groups. Similar to controls, both treatments had a rapid RIHP onset time after a change in RAP; more specifically, in the hexamethonium group there was an initial delay of 0.9±0.2
seconds and in the losartan group an initial delay of 1.4±0.2 seconds (Figure 5C). Also, the difference in the time delays between achieving steady state in RAP and RIHP was not significantly different from controls in both treatment groups. Specifically, the time delay was 7.4±1.1 seconds in the hexamethonium group and 6.7±1.1 seconds in the losartan group for RAP changes of ±0 to 10 mm Hg, whereas for greater pressure changes (±20 to 30 mm Hg) the delay was ~2-fold longer (Figure 6B and 6C).

Discussion
Pressure-natriuresis is an important renal mechanism involved in the regulation of the long-term level of blood volume and arterial pressure. Investigations of pressure-natriuresis, specifically the RAP-RIHP relationship, have usually been conducted over time periods sufficient to induce measurable changes in urine production (e.g., ≥30 minutes). Although previous research has provided important foundational knowledge of renal mechanisms over this time frame, regulation on a moment-to-moment basis has not been as well studied. The key findings of the present study were that the initial RIHP response time to an acute change in RAP was ~1 second, regardless of the magnitude of RAP change and neurohumoral status of the animal, and that completion of the full RIHP response occurred within ~15 seconds for RAP changes of ~30 mm Hg. In addition, the acute RAP-RIHP relationship correlated linearly over a wide range of arterial pressures, and the slope was not affected markedly by pharmacological antagonism of the ANS and RAS. A novel finding from a specific analysis of the RAP-RIHP relationship above and below the arterial pressure operating point revealed that the pressor component was not as steep as the depressor. However, after pharmacological inhibition of the ANS or RAS, this difference between the pressor and depressor components was abolished. These results suggest that this intrarenal hemodynamic mechanism, which is linked to downstream mechanisms regulating sodium excretion, has short-term baroreflex-like properties that may facilitate the regulation of arterial pressure around an operating point.

The time course of RIHP responses after rapid changes in arterial pressure has not been characterized previously. The present study is the first to reveal that the delay between an acute change in RAP and the onset of a RIHP response is ~1 second, regardless of the magnitude or direction of arterial pressure stimulus. These results are consistent with a previous study assessing the onset of changes in urine flow, a down-
stream step from RIHP in the pressure-natriuresis mechanism, where a 6-second delay after various arterial pressure changes was documented.18 Furthermore, the time difference between achieving a steady-state RAP and RIHP was found to depend, in part, on the magnitude of arterial pressure change ranging from 6 to 12 seconds for pressure changes of ±5 to 30 mm Hg. These findings suggest that the consequences of the oscillations in RIHP (ie, on sodium excretion) after spontaneous changes in arterial pressure could occur at a frequency of 5 to 10 changes per minute. Given that arterial pressure fluctuates spontaneously and over a wide range of pressures, the observed baroreflex-like nature of RIHP suggests that this moment-to-moment aspect of pressure-natriuresis can act via progressive, cumulative effects on the level of arterial pressure. That is, moment-to-moment changes in RIHP may impact on urine formation, and the cumulative effect of these acute changes over time may account for the long-term aspect of pressure-natriuresis by inducing homeostatically appropriate changes in blood volume and thereby returning arterial pressure toward its operating point.8,18

Similar to previous studies using conventional pressure-natriuresis methodology,9–17 the overall acute RAP-RIHP relationship was found to correlate linearly and to have a similar slope over a wide range of changes in RAP. However, despite the numerous studies describing the RAP-RIHP relationship as linear, none assessed whether there are differences in slope over a wide range of changes in RAP. The present findings demonstrate that the acute RAP-RIHP relationship appears to also reflect the salt-sensitive phenotype of the animal but does not yet indicate that it is causal. Despite this, it would be expected to find strain- and species-specific differences in the renal mechanisms necessary to regulate sodium excretion, and thereby plasma volume and arterial pressure, under different physiological and pathophysiological conditions. Although it is speculation, the basis for the differences in pressor and depressor components may be because of mechanisms such as myogenic response,25 flow-mediated vasodilation,26 or alterations in the functioning of certain neurohormonal systems.27 Given the blunted RIHP response after pressor manipulations, the myogenic response seems the more likely explanation, because the resulting increase in vascular tone should, in theory, decrease medullary blood flow, capillary hydrostatic pressure, and thereby RIHP, although medullary blood flow responses to spontaneous changes in RAP have yet to be examined. For example, this hypothesis is consistent with the finding that medullary interstitial NO does not rise substantially at RAPs of 110 to 140 mm Hg.28 However, given that Lieb et al29 found that cGMP levels increase after pressor stimuli of 30 minutes, it may be that cGMP levels are increased after a sustained pressor response through the NO-soluble guanylyl cyclase-cGMP pathway. Therefore, the role of these and other vasoactive molecules (ie, arachidonic acid metabolites and endothelins) in the functioning of the acute RAP-RIHP relationship needs to be examined in future studies.27 Lastly, because the full RIHP response is achieved in ≈15 seconds, it is unlikely that slower-acting neurohormonal mechanisms play a role in altering the acute functioning of the RAP-RIHP relationship, although this also needs to be confirmed. Thus, although the basis for the disparity between the pressor and depressor slopes of RIHP and urine flow have not been fully resolved, one concept that appears certain is that the directionally opposing arms of the pressure-natriuresis relationship do not operate equivalently. Thus, future studies should assess these renal mechanisms in both directions from baseline to prevent masking the differences that may have occurred using conventional approaches.
Many neurohumoral factors are known to modulate sodium balance, yet the precise mechanisms by which these factors may affect pressure-natriuresis, particularly in the short term, have not been fully resolved. Nevertheless, most previous studies have tried to control for potential compensatory effects of neurohumoral factors. In the present study, inhibition of the ANS or RAS did not modify the overall slope of the moment-to-moment RAP-RIHP relationship or the time course of RIHP responses to changes in RAP despite the decrease in arterial pressure. However, unlike in untreated animals, the pressor and depressor slopes became similar after these treatments. Given that the treatments lowered the arterial pressure operating point, it appears that the moment-to-moment RAP-RIHP response has become a composite of the pressor and depressor arms in controls (ie, increased pressor and decreased depressor slopes). In fact, these data support the hypothesis that a myogenic response may be responsible for the blunting of the pressor response in control animals. That is, animals treated with the depressor agents may have a dampened intrinsic myogenic response because of lower levels of wall stress and thereby a heightened RIHP response. Future studies will have to determine whether lowering arterial pressure and/or inhibiting the ANS and RAS trigger compensatory responses of vasoactive molecules (ie, NO and arachidonic acid metabolites) that may impact on the functioning of the acute RAP-RIHP relationship.

Although the outcomes of these studies provide a better understanding of acute pressure-natriuresis mechanisms, there are some limitations that need to be addressed. Specifically, given the short-term and bidirectional design of the assessments, urine could not be collected. In addition, whether the occlusion cuff–induced reductions in pulse pressure affect the generation of RIHP independent of changes in mean RAP requires further investigation. However, the finding of a 1-second response lag makes single pulsation transmission unlikely and indicates that there is an inherent smoothing function linking pulsatile RAP with the relatively nonpulsatile RIHP. In addition, it is unknown whether anesthesia and surgery could affect the acute RAP-RIHP relationship; however, given that the overall slope of the present experiments is comparable with those performed previously in conscious animals, the influence of these does not appear to be significant.

In summary, short-term changes in arterial pressure were found to be coupled to moment-to-moment changes in RIHP over a wide range of pressures. That is, this intrarenal mechanism is rapid (≈1 second) and linear, but different, over a wide range of acute pressor and depressor changes. Taken together, these findings suggest that this intermediate component of the pressure-natriuresis mechanism can respond rapidly and repeatedly to transient and bidirectional changes in arterial pressure, thereby modulating sodium excretion in a cumulative manner.

**References**


**Sources of Funding**

This work was funded by the Canadian Institutes of Health Research. M.K. is funded by a Canadian Institutes of Health Research Canada Graduate Doctoral Scholarship.
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Hypertension. 2010;56:650-657; originally published online August 9, 2010;
doi: 10.1161/HYPERTENSIONAHA.109.149633

Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2010 American Heart Association, Inc. All rights reserved.
Print ISSN: 0194-911X. Online ISSN: 1524-4563

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