Role of Cardiovascular Receptors on the Neural Regulation of Renin Release in Normal Men

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SUMMARY Although factors influencing renin release have been studied extensively, one facet of renin release remains controversial, namely, neural regulation by arterial high-pressure receptors and cardiopulmonary low-pressure receptors. We therefore designed four studies to investigate systematically the separate and combined effects of unloading (decreased stretch) high- and low-pressure receptors on renin release in normal men. Selective unloading of cardiopulmonary receptors was induced by impeding the venous return with tourniquets around the thighs. A predominant unloading of arterial (carotid) baroreceptors was elicited with upright posture and simultaneously preventing the venous pooling in the legs. Unloading of both high- and low-pressure receptors was achieved by both upright standing and tilting. During postural experiments to predominantly unload arterial baroreceptors, the heart rate increased and the veins constricted, but renin failed to increase. The postural increase of renin occurred only if we allowed venous pooling in the legs. Selective unloading of cardiopulmonary receptors elicited substantial increases of renin. When both the cardiopulmonary and arterial baroreceptors were unloaded, renin increased more than with isolated unloading of cardiopulmonary receptors. We conclude that: 1) in intact humans it is possible to demonstrate an independent role of cardiopulmonary receptors in the control of renin release; 2) there is evidence for interaction between the two receptor systems in renin control; but 3) an independent role for arterial baroreceptors in the control of renin release could not be demonstrated under the conditions of this experiment. (Hypertension 5: 779-786, 1983)

KEY WORDS • renin release • high-pressure receptors • low-pressure receptors

FACTORS controlling renin release have been the object of intensive research for more than two decades.1 Despite this effort, the details of one facet, namely, neural regulation of renin release in humans, remains controversial. Much of the disagreement centers on the anatomic location of the afferent sensor. The question is whether an increase in renin release in normal humans can occur with unloading (decreased stretch) of either arterial high-pressure2 or cardiopulmonary low-pressure receptors,3 or if it is necessary that both receptor systems are simultaneously unloaded.4 There are at least three reasons for obtaining accurate information on this question: 1) understanding factors regulating an important hormone in volume and pressure homeostasis is of basic physiological significance; 2) our understanding of abnormal renin responses in many patients with hypertension would be improved and this would possibly provide clues regarding the pathophysiology of abnormal responses; and 3) knowing the normal hemodynamic determinants that regulate the function of high- and low-pressure receptors with renin release as an easily measured humoral marker may facilitate research in various disease states where abnormalities of these receptors are suspected. Therefore, we designed the following series of experiments to evaluate the effect on renin release of separate and combined unloading of arterial high-pressure and cardiopulmonary low-pressure receptors.

Methods

Subjects

We conducted three studies using 24 normal male volunteers between the ages of 18 and 49 years who had been on a standardized sodium diet for 4 days. Subjects were instructed on a 20 mEq/day sodium diet supplemented to 150 mEq sodium with salt tablets.
Twenty-four-hour urine sodium analysis just prior to study showed a value of 142.0 ± 7.9 mEq. In a fourth study, 16 normal men were investigated in a mildly sodium-depleted state induced by furosemide 40 mg orally for 3 days preceding the study. All studies were completed on outpatient, ambulatory subjects without restrictions except for dietary sodium. The studies were approved by the University Human Use Committee, and all volunteers signed an informed consent form.

Study 1

Study 1 was designed to evaluate selective unloading of carotid receptors and combined unloading of both high- and low-pressure receptors. To accomplish this, 22 subjects were studied in the supine position (baseline), sitting with lower extremities horizontal, and in standing positions. Sitting was intended to unload carotid receptors because of the hydrostatic difference between the heart and carotid baroreceptors that occurs with upright posture. Pooling of blood in dependent areas was minimized by maintaining the lower extremities in the horizontal position. Standing maintained the same hydrostatic unloading of carotid receptors as in sitting and additionally induced unloading of low-pressure receptors secondary to blood pooling in dependent areas.

Upon arrival at the Clinical Physiology Laboratory, subjects reclined and an 18-gauge short Teflon catheter for blood sampling was placed in one antecubital vein. After 1 hour in the supine position, subjects had measurements taken of blood pressure (BP, mm Hg), heart rate (HR, bpm), plasma renin activity (PRA, ng/ml/hr), plasma norepinephrine concentration (NE, pg/ml), and venous distensibility (V, 30 percent changes in forearm volume). Subjects were then randomly assigned to either sit with lower extremities horizontal and the back bent in the thoracolumbar region so that the pelvis and lower abdomen were horizontal (to minimize blood pooling), or stand with their arms resting on a table. Each of the maneuvers lasted for 30 minutes whereupon variables were remeasured. After the first postural stimulus, subjects were supine for 1 hour and a second set of baseline measurements obtained. Finally, subjects completed this portion of the study with sitting if standing were done earlier and vice versa.

Blood pressure was measured in triplicate by a mercury sphygmomanometer with the cuff applied to the right arm. Blood pressure measurements in all subjects were determined by one observer. The average of the three measurements was recorded as the blood pressure for each postural stimulus. Heart rate was determined from 60 seconds of an electrocardiographic (ECG) tracing obtained during the last 5 minutes of each period. Venous distensibility was assessed by venous occlusion plethysmography. The upper extremity was inclined at a 20° angle from the heart with the forearm segment suspended. A mercury-in-Silastic strain gauge, which encircled the forearm 5 cm distal to the olecranon, was connected to a Hokanson EC-3 plethysmograph, D.E. Hokanson, Inc., Issaquah, Washington. Minimal occluding pressure, which averaged 3–5 mm Hg, was added to the occluding cuff pressure of 30 mm Hg. Forearm volume at equilibrium for each occluding pressure was obtained during each period. Plasma renin activity was assessed by radioimmunoassay, and plasma norepinephrine concentration by a radioenzymatic assay.

Study 2

Seven subjects participated in Study 2, which was designed to assess by invasive methods the hemodynamic changes that occurred on change of posture from supine to sitting, as described in Study 1. In addition to data obtained with this predominantly carotid receptor stimulus, information was obtained during supine thigh cuff inflation, which unloads low-pressure receptors by decreasing right atrial pressure and central blood volume without significantly altering arterial pressures.

Subjects were placed in the supine position and an 18-gauge 2-inch Teflon catheter was introduced into the right basilic vein into the right atrium. Catheter position was verified by obtaining a characteristic right ventricular tracing and withdrawing the catheter to obtain a right atrial waveform. Statham strain gauges for measuring arterial and central venous pressures were placed in the left fourth intercostal space in the midaxillary line. Cardiac output (CO, liters/min) was determined by dye dilution described previously. Cardiopulmonary blood volume (CBV, ml) was calculated multiplying the cardiac output by the mean transit time. Limitations of this method have been discussed elsewhere. Lead II of the ECG was monitored continuously. After lines were placed and 30 additional minutes had elapsed, measurements of arterial and right atrial pressures, cardiac output, central blood volume, heart rate, plasma renin activity, and norepinephrine were obtained in the supine position. Pediatric cuffs in series of sufficient length to encircle the upper thighs were inflated to 40 mm Hg for 30 minutes and variables remeasured. Thigh cuffs were deflated and subjects remained supine for another 45 minutes whereupon variables were remeasured. This study was concluded with subjects sitting for 30 minutes as described in Study 1, and a final set of measurements obtained.

Study 3

Study 3 was designed to induce graded isolated unloading of low-pressure receptors and to study further the simultaneous unloading of both high- and low-pressure receptors. Unloading of low-pressure receptors was accomplished by two levels of thigh cuff inflation. Unloading of both high- and low-pressure receptors was induced by tilting to 35°, which is comparable, but lesser in magnitude, to the effects of standing described in Study 1. This invasive hemodynamic study used instrumentation and measurements...
Table 1. Results From Noninvasive Study in 22 Normal Men Comparing the Effects of Selective Unloading of High-Pressure Receptors (Sitting) vs Combined Unloading of Both High- and Low-Pressure Receptors (Standing)

<table>
<thead>
<tr>
<th></th>
<th>Supine</th>
<th>Sitting</th>
<th>p</th>
<th>Standing</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (bpm)</td>
<td>63.7 ± 3.1</td>
<td>66.7 ± 3.1</td>
<td>0.03</td>
<td>62.2 ± 3.2</td>
<td>82.3 ± 4.3</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>84.6 ± 2.0</td>
<td>87.0 ± 2.1</td>
<td>NS</td>
<td>85.1 ± 2.2</td>
<td>84.3 ± 2.5</td>
</tr>
<tr>
<td>PP (mm Hg)</td>
<td>52.4 ± 2.2</td>
<td>43.3 ± 2.0</td>
<td>0.01</td>
<td>50.5 ± 2.3</td>
<td>32.5 ± 2.2</td>
</tr>
<tr>
<td>Vv30 (%)</td>
<td>3.17 ± 0.22</td>
<td>2.64 ± 0.20</td>
<td>0.01</td>
<td>3.24 ± 0.17</td>
<td>2.64 ± 0.20</td>
</tr>
<tr>
<td>PRA (ng/ml/hr)</td>
<td>1.03 ± 0.11</td>
<td>1.07 ± 0.13</td>
<td>NS</td>
<td>1.09 ± 0.16</td>
<td>2.68 ± 0.30</td>
</tr>
<tr>
<td>NE (pg/ml)</td>
<td>206 ± 18</td>
<td>266 ± 27</td>
<td>0.02</td>
<td>183 ± 17</td>
<td>422 ± 42</td>
</tr>
</tbody>
</table>

Data are presented as means ± SEM. The p value was obtained from Student’s paired t test of differences. There were no differences for any of the variables between the two baselines as assessed by the paired t test.

HR = heart rate; MAP = mean blood pressure; PP = pulse pressure; Vv30 = venous distensibility at 30 mm Hg (% change in forearm volume); PRA = plasma renin activity; and NE = plasma norepinephrine.

Table 2. Results of Invasive Hemodynamic Data Comparing Sitting to Supine Baseline Values in Seven Normal Men

<table>
<thead>
<tr>
<th></th>
<th>Supine</th>
<th>Sitting</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (bpm)</td>
<td>49.9 ± 2.6</td>
<td>52.1 ± 2.2</td>
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<tr>
<td>MAP (mm Hg)</td>
<td>78.7 ± 3.5</td>
<td>86.3 ± 2.1</td>
<td>0.03</td>
</tr>
<tr>
<td>PP (mm Hg)</td>
<td>55.1 ± 2.5</td>
<td>59.7 ± 2.9</td>
<td>0.02</td>
</tr>
<tr>
<td>CO (liters/min)</td>
<td>4.8 ± 0.3</td>
<td>5.0 ± 0.3</td>
<td>0.01</td>
</tr>
<tr>
<td>CBV (ml)</td>
<td>1595 ± 96</td>
<td>1484 ± 94</td>
<td>0.01</td>
</tr>
<tr>
<td>RAP (mm Hg)</td>
<td>3.7 ± 1.3</td>
<td>1.4 ± 0.9</td>
<td>0.03</td>
</tr>
<tr>
<td>PRA (ng/ml/hr)</td>
<td>0.63 ± 0.14</td>
<td>1.05 ± 0.51</td>
<td>0.41</td>
</tr>
<tr>
<td>NE (pg/ml)</td>
<td>179 ± 38</td>
<td>232 ± 36</td>
<td>0.06</td>
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</table>

Data are means ± SEM. The p value was determined from paired Student’s t test of differences. MAP = mean arterial blood pressure; CO = cardiac output; CBV = cardiopulmonary blood volume; and RAP = right atrial pressure. Other abbreviations are as in table 1.

*The apparent increase of the mean PRA value with sitting is accounted for entirely by one individual whose PRA increased from 0.52 to 3.83 while the other six had minor and insignificant changes.

Study 1

In this noninvasive study, sitting, compared to lying down, did not increase plasma renin activity (table 1). Heart rate and norepinephrine, however, increased, and venous distensibility decreased. Mean blood pressure tended to increase but the change was not significant, while pulse pressure decreased. Standing increased plasma renin activity from the preceding recumbent baseline and caused larger increases in heart rate and norepinephrine than sitting, although the decrease of venous distensibility was not larger.

Study 2

In this invasive study, changing position from supine to sitting did not increase plasma renin activity, although it again increased norepinephrine levels (table 2). Heart rate tended to increase, but not significantly, and mean intraarterial pressure increased. Both right atrial pressure and cardiopulmonary blood volume decreased while cardiac output increased slightly but consistently in each subject. Results of supine to cuff 40 mm Hg thigh cuff inflation portion of the experiment have been combined with supine to cuff 30 mm Hg results in Study 3 (table 3).
Study 3

A 35° tilt, compared to the supine position, increased plasma renin activity. Heart rate increased while cardiac output and central blood volume decreased. Mean blood pressure was unchanged. A 30 and 40 mm Hg thigh cuff inflation, compared to the recumbent baseline, increased plasma renin activity and heart rate, while central blood volume decreased. Mean arterial pressure, pulse pressure, and cardiac output were unchanged. A 70 mm Hg thigh cuff inflation appeared to cause greater increases in heart rate and plasma renin activity, and greater decreases in central blood volume, compared with changes induced by a 30° to 40° cuff inflation. Mean arterial pressure, pulse pressure, and cardiac output were again unchanged. For hormonal measurements, however, the higher thigh cuff pressure was not preceded by a comparable baseline (only 5 minutes of decompression) for direct statistical comparison. Consequently, the data are presented only for overall comparison of trends in figure 1.

Study 4

Plasma renin activity with tilt in this study increased an average of 78% (fig. 2). This renin increase was largely reversed with the antigravity suit. Filling of the antigravity suit in the tilted position reversed orthosta-

<table>
<thead>
<tr>
<th>Study</th>
<th>HPR</th>
<th>LPR</th>
<th>HPR+LPR</th>
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<tbody>
<tr>
<td></td>
<td>n=16</td>
<td>n=22</td>
<td></td>
</tr>
<tr>
<td>MAP</td>
<td>15</td>
<td>10</td>
<td>15</td>
</tr>
<tr>
<td>%Δ</td>
<td>5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>HR</td>
<td>30</td>
<td>30</td>
<td>30</td>
</tr>
<tr>
<td>%Δ</td>
<td>10</td>
<td>10</td>
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</tr>
<tr>
<td>NE</td>
<td>125</td>
<td>125</td>
<td>125</td>
</tr>
<tr>
<td>%Δ</td>
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<td>75</td>
<td>75</td>
</tr>
<tr>
<td>PRA</td>
<td>150</td>
<td>150</td>
<td>150</td>
</tr>
<tr>
<td>%Δ</td>
<td>75</td>
<td>75</td>
<td>75</td>
</tr>
<tr>
<td>CBV</td>
<td>-10</td>
<td>-10</td>
<td>-10</td>
</tr>
<tr>
<td>%Δ</td>
<td>25</td>
<td>25</td>
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</tr>
<tr>
<td>PP</td>
<td>+30</td>
<td>+15</td>
<td>+15</td>
</tr>
<tr>
<td>%Δ</td>
<td>-15</td>
<td>-15</td>
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**Figure 1. Overview of studies selectively unloading either high-pressure receptors (HPR) or low-pressure receptors (LPR) and simultaneously unloading both high- and low-pressure receptors (HPR + LPR). Data are presented as means ± SEM of the percentage changes compared to the nearest supine baseline. The p values are obtained from either a student's t test (*) or Wilcoxon's ($) rank sum test of paired differences. * or $ p < 0.05; † p < 0.01; ‡ p < 0.001.**
Figure 2. Hemodynamic and humoral responses to combined unloading of high- and low-pressure receptors (tilt) vs supine baseline and selective unloading of high-pressure receptors (tilt + water suit). Asterisks denote the significance of changes from baseline to tilt and from tilt without a filled suit to tilt with the suit filled. *p < 0.05; †p < 0.01; ‡p < 0.001. The significance of difference was also tested for values of baseline and values 30 minutes after filling the suit using a student’s paired t test with Bonferroni’s correction for multiple comparison. The heart rate (p < 0.01), mean blood pressure (p < 0.001), and plasma renin activity (p < 0.05) were significantly different; the rest of the variables did not differ from baseline. Analysis of variance (ANOVA) for repeated measures produced comparable results.

An overview of the four studies is given in figure 1. All variables are expressed as percentage changes from the baseline. Procedures that selectively unload the carotid receptors are given in the first column. Plasma renin activity did not increase appreciably with these procedures, although PRA did not return completely to
baseline 30 minutes after filling the antigravity suit in Study 4. In the middle column, two grades of isolated low-pressure receptor unloading are shown. These procedures elicited a graded increase of PRA. In the right-hand column are the results of two procedures where both high- and low-pressure receptors were unloaded. Plasma renin increased further with this combined unloading of both receptor systems, and mirrored the decrease of cardiopulmonary blood volume that occurred progressively from the middle to right-hand columns under the study conditions. Plasma norepinephrine and heart rate changes were parallel with the changes in plasma renin activity. Contrary to central blood volume trends, figure 1 does not suggest a relationship between blood pressure or pulse pressure changes and the changes in renin or norepinephrine.

**Discussion**

The question under study is of practical importance; abnormalities of plasma renin in the upright posture have been described in patients with essential hypertension. In some patients these abnormalities are associated with alteration of sympathetic activity. To explain to what degree this association between sympathetic and renin abnormalities in hypertension could be caused by an abnormality of the reflex release of renin to upright posture, it is important to understand the nature of these reflexes in intact humans. Thus, our study sought to determine whether the predominant effect on reflex release of renin in the upright posture originates in the cardiopulmonary or arterial baroreceptors. It is clear from animal experiments that both receptor systems play a role in the renin release. The effect on renin of cardiopulmonary receptors and of the interaction of cardiopulmonary with arterial baroreceptors in animals has been relatively well demonstrated. Complex animal experiments, however, are required to show the role of arterial baroreceptors on renin release. The blood pressure increase elicited by the unloading of arterial baroreceptors tends to offset the influence that arterial baroreceptors may have on renin release.

Thus, Jarecki et al. had to maintain a constant renal perfusion pressure, cut the vagal afferents, and only then were able to show that alterations of carotid sinus pressure cause variations of renin release. This important study clarifies the potential influence that arterial baroreceptors have on reflex renin release, but does not explain the role of these receptors under physiologic circumstances. In our study, we chose maneuvers that elicit responses that occur naturally and belong to the usual “repertoire” of the human body. Our water suit experiments mimic standing up in shallow water, and the sitting experiments are akin to sitting in a bath tub. We avoided such nonphysiologic stimuli as neck chambers or lower body negative pressure. Consequently, the results of our study can be viewed only as an investigation into the relative importance of cardiopulmonary vs arterial baroreceptors in the overall reflex response to upright posture. They are not designed to unravel the intricacies of complex interactions in the human body.

In contradistinction to animal data, there has been some controversy in the literature whether cardiopulmonary mechanoreceptors play a role in the renin release in humans. Our previous study provided substantial evidence that these receptors do have an independent influence on the reflex release of renin.

In that study we used the thigh cuff inflation method to reduce right atrial pressure and were able to elicit increases of renin that mirrored the decreases of cardiopulmonary blood volume, which could be abolished by beta-adrenergic blockade and which did not occur in patients with denervated, transplanted kidneys. In the previous study volunteers were sodium-depleted and the thigh cuff pressure was high (70 mm Hg). In the present study in sodium release subjects the initial thigh cuff congesting pressure was lower (30 to 40 mm Hg) and the starting cardiopulmonary blood volumes were higher. Despite a higher starting cardiopulmonary blood volume and a lesser degree of leg congestion, the plasma renin increased on the average of 55% compared to supine baseline. The renin increased further when the cuffs were inflated to 70 mm Hg (fig. 2). Significant decreases in central blood volume confirm...
that thigh cuff inflation elicited the expected unloading of low-pressure receptors. Major determinants of high-pressure receptor activity, namely, mean arterial pressure and pulse pressure, did not change during thigh cuff inflation. Cardiac output also did not change.

In the present study, we also attempted to investigate the role of carotid baroreceptors in the control of renin release. Our approach was to utilize the heart-to-neck difference in the hydrostatic pressure to unload the carotid baroreceptors. When the upper part of the body is vertical, the calculated heart-to-neck difference is 17 mm Hg. Thus, by elevating the upper trunk we unloaded the carotid baroreceptors, and then hoped to independently vary the load on cardiopulmonary receptors by allowing or preventing the pooling of blood in the lower extremities. The prediction was that, if the cardiopulmonary mechanoreceptors are important, the renin levels will reflect the variation of the stretch on cardiopulmonary mechanoreceptors even when carotid baroreceptors remain unloaded by upright posture. This prediction was fulfilled but the interpretation of the results is not as straightforward as we had hoped. Sitting with the legs in the horizontal position elicited an increase of heart rate, norepinephrine, and venous tone, whereas plasma renin failed to follow suit. Thus, a reflex was elicited, but the renin release was not affected. Sitting, as the invasive data indicate, did not fully prevent the pooling of blood and there was a minor but significant decrease of right atrial pressure and cardiopulmonary blood volume. Nevertheless, a predominant unloading of carotid baroreceptors with sitting, even when combined with mild unloading of cardiopulmonary receptors, does not increase plasma renin.

The results with tilting and the water suit are also in line with the prediction and on surface support the notion of a predominant influence of cardiopulmonary receptors on renin release. In this series of experiments, tilting elicited the expected increase of heart rate, plasma renin, and norepinephrine when the right atrial pressure and cardiopulmonary blood volume were permitted to fall. When the fall of the cardiopulmonary blood volume was prevented by filling of the suit, but the neck remained elevated above the heart, the heart rate, norepinephrine, and renin levels returned toward the values observed in recumbency. In both experiments (sitting with legs horizontal and filling of the suit), when the carotid baroreceptor was above the level of the heart but the fall of the cardiopulmonary blood volume was largely prevented, there was a small but significant increase of the arterial pressure. This increased pressure may have had an effect on renal or other arterial baroreceptors and thereby could have counterbalanced the effect of carotid sinus unloading on renin release.

Apparently, as Jarecki et al. did, one has to create highly artificial experimental conditions and prevent the naturally occurring pressure rises to show the effect of arterial baroreceptors on renin release. The practical message from our study and those of Mancia et al. is that, because of pressure changes, such an influence on renin release could not be shown in intact human beings. Since renin abnormalities in essential hypertension are not associated with an abnormal blood pressure response to posture, it is not likely that arterial baroreceptors are responsible for the aberration of the reflex renin release to upright posture in hypertension.

Unloading both high- and low-pressure receptors with 35° tilt (Study 3) induced larger increases in PRA compared to unloading of low-pressure receptors alone. This larger renin response may reflect only a greater unloading of low-pressure receptors during tilt (21% decrease in central blood volume versus 12% with thigh cuff). It is also possible that high-pressure receptors, under these circumstances, interact with low-pressure receptors and contribute to renin control. The design of our studies does not allow differentiation between these two possibilities.

The overall results of our four studies are summarized in figure 1 and support the physiologic role of low-pressure receptors in the control of renin release. They show that: 1) predominant unloading of carotid receptors alone increases heart rate and mean arterial pressure without affecting plasma renin activity (sitting and tilting with the filled suit); 2) selectively unloading low-pressure receptors increases plasma renin activity and heart rate (thigh cuff experiments); and 3) loading of low-pressure receptors, while the load on carotid receptors remains diminished, reverses the increase of plasma renin activity (filling of the suit in tilted position).

When both arterial and cardiopulmonary receptors were simultaneously unloaded, the heart rate, norepinephrine, and renin increases were augmented. This speaks for an interaction of the two receptor systems. Under the conditions of our experiments, however, we could not show an independent role of the arterial baroreceptors in the control of renin release.

In our experiments, changes in central blood volume symbolize the stretch of low-pressure baroreceptors. Changes in mean blood pressure and pulse pressure, as well as changes in heart-to-neck hydrostatic pressure, relate to the function of high-pressure receptors. A glance at figure 1 shows a clear trend: plasma renin activity, plasma norepinephrine, and heart rate are proportional and inversely related to changes in central blood volume. There is no evidence for a consistent relationship between the high-pressure receptor variables (mean blood pressure and pulse pressure) and plasma renin or plasma norepinephrine responses.

We should reemphasize that these studies in intact humans are designed to analyze the overall effects under physiologic conditions. It is conceivable that arterial baroreceptors may play an important role in renin release under some complex pathophysiologic circumstances.
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