Autonomic Nervous System and Benign Essential Hypertension in Man

II. CIRCULATORY AND HORMONAL RESPONSES TO UPRIGHT POSTURE

By Jean-Louis Cuche, Otto Kuchel, André Barbeau, Yves Langlois, Roger Boucher, and Jacques Genest

ABSTRACT

The effect of upright posture as a physiological stimulus of the adrenergic nervous system was studied in 56 subjects with benign essential hypertension. The subjects received a controlled-sodium diet. Blood pressure, heart rate, catecholamines, plasma renin activity, and urinary creatinine, sodium, and potassium excretion were measured in the recumbent and upright positions. We found an alteration in the blood pressure response in subjects with benign essential hypertension; the postural increase in the mean blood pressure in normotensive subjects (3.18 ± 1.35 mm Hg) progressively disappeared and was replaced by a postural decrease in subjects with more severe stable hypertension (-6.71 ± 2.42 mm Hg). The hypertensive subjects also lacked the usual increase in urinary excretion of norepinephrine. A significant increase in plasma renin activity associated with a significant decrease in plasma norepinephrine occurred in subjects with labile hypertension with postural tachycardia. Finally, we found a highly significant correlation between the excretion of sodium and potassium in the recumbent position and the retention of both ions in the upright position.

KEY WORDS

postural adaptation  plasma renin activity urinary dopamine, norepinephrine, and epinephrine renal clearance of norepinephrine

orthostatic antinatriuresis

In a previous paper (1), we described the rationale for and the method in which limits between normotension and labile and stable hypertension were determined in recumbent subjects. In the present investigation, these same subjects were exposed to the stimulus of upright posture.

This approach was of interest because the reproducible, physiological stimulus of upright posture normally induces activation of hemodynamic and closely related hormonal mechanisms. Hemodynamic studies of subjects with hypertension have shown that the postural stimulus differentiates between subjects who react by an excessive response and those who have an insufficient postural response (2). Hormonal studies have demonstrated a differentiation between subjects who show a normal renin response and those who show a low renin response (3).

The autonomic nervous system is a key link between hemodynamic and hormonal mechanisms of blood pressure adaptation to upright posture. In normotensive subjects, orthostasis is associated with an increase in plasma (4,5) and urinary (6,7) norepinephrine and a decrease in urinary dopamine (7). In addition, an increase in dopamine-β-hydroxylase activity has recently been reported in normotensive subjects in response to upright posture (8,9).

Our present approach of exposing subjects with benign essential hypertension to a physiological postural challenge was designed to contribute to a better characterization of the heterogeneous entity of benign essential hypertension (1).

Methods

The classification and clinical presentation of the hypertensive subjects, as well as the methodology, have been previously described (1). Briefly, a total of 70 subjects—a control group of 14 healthy volunteers (group R) and a group of 56 subjects with benign essential hypertension—were studied. The hypertensive group was further divided into four subgroups: group Ia included subjects with labile hypertension and low orthostatic heart rate acceleration, group Ib included subjects with labile hypertension and an excessive orthostatic heart
rate acceleration, and group IIa and group IIb included subjects with less severe and more severe, respectively, stable hypertension.

**PROTOCOL OF INVESTIGATION**

For 3 days prior to the experiment, the subjects were maintained on a diet containing 135 mEq sodium/day and 90 mEq potassium/day. The fourth and fifth days of the diet were the experimental days.

On the fourth day at 8:30 AM, the subjects emptied their bladders after being confined to bed from the previous evening. A 4-hour urinary collection was then started in the recumbent subjects for the subsequent determination of catecholamines (10), creatinine (11), sodium, and potassium. At 12:30 PM, a blood sample was drawn for the determination of plasma renin activity (12) and in 50% of the subjects for the determination of plasma norepinephrine (13). These measurements in recumbent subjects were considered as base-line values.

On the fifth morning, the subjects remained recumbent until they had emptied their bladders at 8:30 AM. They were then kept either standing or sitting, but never recumbent, until 12:30 PM; urine was collected during the 4-hour period. At 12:30 PM, a blood sample was drawn for plasma renin activity and plasma norepinephrine determinations.

**STATISTICAL ANALYSIS**

The statistical analysis of the effects of blood pressure variation on each parameter has been described previously (1). To test the effect of upright posture, we used an analysis of variance for blocks with two treatments: recumbent and upright posture. Because the treatments within blocks were not randomized, the effects of treatment and time were mixed.

**Results**

**EFFECTS OF UPRIGHT POSTURE ON CARDIOVASCULAR FUNCTIONS**

On assuming upright posture, group R subjects showed a statistically significant \( P < 0.01 \) increase in their usual blood pressure indexes from 82.5 ± 2.54 mm Hg to 85.8 ± 2.16 mm Hg (\( \Delta = +3.18 \pm 1.35 \) mm Hg) and group IIb subjects showed a significant \( P < 0.05 \) decrease from 134.2 ± 2.64 mm Hg to 127.5 ± 3.05 mm Hg (\( \Delta = -6.71 \pm 2.42 \) mm Hg) (Table 1). This progressive, qualitative change can be explained mainly by an accentuation of the expected systolic blood pressure decrease and the disappearance of the expected diastolic increase induced by upright posture (Fig. 1). The net effect (recumbent-upright difference in pulse pressure) of the increase in gravitational pressure induced by upright posture on the decrease in pulse pressure was comparable in all five groups in spite of the higher pulse pressure in group IIb (75.8 ± 5.26 mm Hg) than in group R (47.6 ± 3.14 mm Hg) during recumbency.

We observed a significant increase in the heart rate of all subjects when they were in the upright posture. However, in spite of the same usual blood pressure indexes and systolic and diastolic pressures in both the recumbent and upright positions, there was a heart rate acceleration of \( 8 \pm 1 \) beats/min in group IA and \( 22 \pm 0.9 \) beats/min in group IIA.

**EFFECTS OF UPRIGHT POSTURE ON RENAL FUNCTIONS**

The well-known antiuretic, antinatriuretic, and antikaliuretic effects induced by upright posture were observed in all subjects (Table 1). Assuming that the recumbent-upright difference is an index of the orthostatic antinatriuresis, the magnitude of the mean orthostatic antinatriuresis in group IA \(( -44.82 \pm 7.74 \text{ mEq/4 hours} )\) was significantly higher than that in group IIb \(( -27.03 \pm 4.62 \text{ mEq/4 hours} )\). However, the degree of sodium excretion in recumbent subjects appeared to be a determinant of the degree of postural antinatriuresis. We computed an evident linear, negative correlation (Fig. 2) between recumbent natriuresis and orthostatic antinatriuresis, and we did not observe any appreciable difference in the percent of the orthostatic decrease in sodium excretion compared with the recumbent natriuresis. This percent was 76% in group R, 76% in group IA, 71% in group IIb, 66% in group IIa, and 79% in group IIb.

Using the same orthostatic difference that was determined for sodium, we computed a significant linear, negative correlation between recumbent kaliuresis and orthostatic antikaliuresis \(( r = -0.779, P < 0.001, y = 5.582 - 0.702x )\) and between recumbent and upright excretion of creatinine \(( r = -0.726, P < 0.001, y = 132.1 - 1.394x )\).

In groups IA and IIa, there was a significant increase in the ratio of urinary sodium to urinary potassium in recumbent subjects. In group IA, at least, this increase was probably due to the increase in sodium excretion \((60.5 \pm 9.44 \text{ mEq/4 hours})\) because no difference was observed in potassium excretion. On assuming the upright position, a significant decrease in the ratio of sodium to potassium was observed in all five groups. In the upright position, the ratio of sodium to potassium was only slightly higher in group IA than it was in group R \(( P < 0.10 )\), but it was significantly higher than that in group IIb \(( P < 0.05 )\).

**EFFECTS OF UPRIGHT POSTURE ON URINARY EXCRETION OF CATECHOLAMINES**

When the subjects assumed the upright position, a significant decrease in the urinary excretion of dopamine was observed in groups R, IA, and IIa, but not in groups Ib and IIb. The expected increase in norepinephrine excretion was observed in group R.
### TABLE 1

**Effects of Stimulation by Upright Posture**

<table>
<thead>
<tr>
<th></th>
<th>R</th>
<th>I₂₂</th>
<th>I₃₂</th>
<th>II₂₂</th>
<th>II₃₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>Usual blood pressure index (mm Hg)</td>
<td>+3.18 ± 1.35*</td>
<td>-0.86 ± 2.01</td>
<td>+0.14 ± 1.75</td>
<td>-2.96 ± 1.99</td>
<td>-6.71 ± 2.42</td>
</tr>
<tr>
<td>Systolic pressure (mm Hg)</td>
<td>-3.2 ± 1.52*</td>
<td>-8.90 ± 2.88*</td>
<td>-8.85 ± 2.81*</td>
<td>-13.0 ± 2.1*</td>
<td>-14.0 ± 3.7*</td>
</tr>
<tr>
<td>Diastolic pressure (mm Hg)</td>
<td>+7.1 ± 1.34†</td>
<td>+1.07 ± 2.57</td>
<td>+3.0 ± 1.60</td>
<td>+0.57 ± 0.9</td>
<td>-1.8 ± 2.47</td>
</tr>
<tr>
<td>Pulse pressure (mm Hg)</td>
<td>-10.4 ± 2.2†</td>
<td>-9.6 ± 2.1†</td>
<td>-10.5 ± 2.9†</td>
<td>-12.8 ± 1.9†</td>
<td>-11.0 ± 2.6†</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>+12.9 ± 1.1†</td>
<td>+8.0 ± 1.1†</td>
<td>+22.5 ± 0.97†</td>
<td>+12.8 ± 2.1†</td>
<td>+10.3 ± 1.3†</td>
</tr>
<tr>
<td>Urinary volume (ml/min)</td>
<td>-2.21 ± 0.35†</td>
<td>-2.30 ± 0.35†</td>
<td>-2.52 ± 0.31†</td>
<td>-1.37 ± 0.32†</td>
<td>-1.34 ± 0.22†</td>
</tr>
<tr>
<td>Urinary creatinine (mg/4 hours)</td>
<td>-62.0 ± 27.0</td>
<td>-88.0 ± 49.0</td>
<td>-80.0 ± 52.0</td>
<td>-46.0 ± 24.0</td>
<td>-28.0 ± 19.0</td>
</tr>
<tr>
<td>Urinary sodium (mEq/4 hours)</td>
<td>-33.1 ± 3.4†</td>
<td>-44.8 ± 7.7†</td>
<td>-33.4 ± 4.4†</td>
<td>-26.7 ± 3.5†</td>
<td>-27.1 ± 4.6†</td>
</tr>
<tr>
<td>Urinary potassium (mEq/4 hours)</td>
<td>7.63 ± 2.1†</td>
<td>-7.65 ± 1.86†</td>
<td>-8.80 ± 1.99†</td>
<td>-2.23 ± 1.72†</td>
<td>-5.61 ± 1.42†</td>
</tr>
<tr>
<td>Ratio of sodium to potassium</td>
<td>1.21 ± 0.45†</td>
<td>-1.67 ± 0.15†</td>
<td>-1.31 ± 0.28†</td>
<td>-1.13 ± 0.35†</td>
<td>-1.50 ± 0.35†</td>
</tr>
<tr>
<td>Urinary dopamine (µg/4 hours)</td>
<td>22.0 ± 12.0*</td>
<td>-42.0 ± 17.0*</td>
<td>-30.0 ± 15.0</td>
<td>-34.0 ± 10.0†</td>
<td>-27.0 ± 17.0</td>
</tr>
<tr>
<td>Urinary norepinephrine (µg/4 hours)</td>
<td>+4.18 ± 1.07†</td>
<td>-6.14 ± 8.53</td>
<td>-0.82 ± 3.07</td>
<td>-5.12 ± 3.65</td>
<td>+0.80 ± 2.14</td>
</tr>
<tr>
<td>Urinary epinephrine (µg/4 hours)</td>
<td>+1.94 ± 1.16</td>
<td>-2.81 ± 2.30</td>
<td>-0.71 ± 1.06</td>
<td>-2.20 ± 1.41</td>
<td>-0.68 ± 0.84</td>
</tr>
<tr>
<td>Ratio of dopamine to norepinephrine</td>
<td>6.3 ± 2.03†</td>
<td>2.19 ± 0.95†</td>
<td>-1.48 ± 0.82</td>
<td>-1.57 ± 0.89</td>
<td>-1.84 ± 1.07</td>
</tr>
</tbody>
</table>

Values are means ± SE.

* P < 0.05.
† P < 0.01.
ORTHOSTATIC DYSREGULATION IN HYPERTENSION

Effect of upright posture on systolic, diastolic, and mean blood pressure (B.P.) (diastolic + \( \frac{1}{3} \) the systolic-diastolic pressure difference) illustrated by the differences in recumbent (R) and upright (U) measurements are compared with recumbent data. Arrows indicate significant differences. Square limits on the middle section represent limits of variation (mean ± 1 SEM) of mean recumbent usual blood pressure indexes and mean recumbent-upright difference of such usual blood pressure indexes: mean recumbent usual blood pressure indexes in group R represent the mean ± 3 SEM.

FIGURE 1

Correlation between urinary sodium excretion during recumbency and urinary sodium excretion during 4 hours of upright posture (mEq sodium/4 hours).

FIGURE 2

but not in any of the hypertensive groups. A significant decrease in the ratio of dopamine to norepinephrine was observed in groups R and I, but not in groups I, II, and III. We did not find any significant difference in the ratio of dopamine to norepinephrine between groups in the upright position in spite of a significant decrease between group R and all hypertensive groups during recumbency.

EFFECTS OF UPRIGHT POSTURE ON PLASMA RENIN ACTIVITY, PLASMA NOREPINEPHRINE, AND RENAL CLEARANCE OF NOREPINEPHRINE

The expected increase in the means of plasma renin activity was observed in all groups (Table 2); when individual values were considered, it increased in 56 of 70 subjects, remained unchanged in 10 subjects, and decreased in 4 subjects. The increase in plasma renin activity was, however, significant only in groups R, I, and II.

In group I, the plasma norepinephrine concentration decreased significantly from 0.43 ± 0.09 ng/ml in recumbent subjects to 0.15 ± 0.06 ng/ml in subjects in the upright position.

Renal clearance of norepinephrine \( (C_{\text{NE}}) \) (ml/min) was calculated according to the formula \( C_{\text{NE}} = \frac{U}{V} \), where \( U \) is urinary norepinephrine (ng/ml), \( V \) is urinary volume (ml/min), and \( P \) is plasma norepinephrine (ng/ml). Only paired observations with detectable plasma norepinephrine are reported. The mean value for group I was approximately 3.7 times higher in the upright posture than it was during recumbency. In the four other groups, upright posture did not induce very much variation.

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TABLE 2
Effects of Upright Posture on Plasma Renin Activity, Plasma Norepinephrine, and Renal Clearance of Norepinephrine

<table>
<thead>
<tr>
<th>Group</th>
<th>R</th>
<th>I_A</th>
<th>I_B</th>
<th>II_A</th>
<th>II_B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma renin activity (ng/ml min⁻¹) (N = 14)</td>
<td></td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>Recumbent</td>
<td>0.29 ± 0.077</td>
<td>0.29 ± 0.069</td>
<td>0.58 ± 0.078*</td>
<td>0.43 ± 0.103</td>
<td>0.35 ± 0.098</td>
</tr>
<tr>
<td>Upright</td>
<td>1.01 ± 0.282†</td>
<td>0.85 ± 0.327</td>
<td>1.36 ± 0.241†</td>
<td>1.03 ± 0.145‡</td>
<td>0.92 ± 0.343</td>
</tr>
<tr>
<td>Plasma norepinephrine (ng/ml) (N = 7)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recumbent</td>
<td>0.25 ± 0.06</td>
<td>0.20 ± 0.08</td>
<td>0.43 ± 0.09</td>
<td>0.68 ± 0.22*</td>
<td>0.32 ± 0.11</td>
</tr>
<tr>
<td>Upright</td>
<td>0.39 ± 0.09</td>
<td>0.39 ± 0.19</td>
<td>0.15 ± 0.06†</td>
<td>0.53 ± 0.16</td>
<td>0.26 ± 0.07</td>
</tr>
<tr>
<td>Renal clearance of norepinephrine (ml/min)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recumbent</td>
<td>125 ± 18</td>
<td>303 ± 90</td>
<td>154 ± 37</td>
<td>272 ± 123‡</td>
<td>117 ± 29</td>
</tr>
<tr>
<td>Upright</td>
<td>146 ± 31</td>
<td>320 ± 102</td>
<td>501 ± 207</td>
<td>158 ± 50</td>
<td>165 ± 48</td>
</tr>
</tbody>
</table>

(N = 6) (N = 5) (N = 5) (N = 6) (N = 5)

Values are means ± SE. No statistical calculations were performed on the clearance of norepinephrine since it is a product of calculation in itself. N = number of subjects tested.
*P < 0.05 compared with group R.
†P < 0.01 compared with recumbent subjects in the same group.
‡If we eliminate the one subject with a very high norepinephrine clearance (956 ml/min), the mean value for the remaining 6 subjects is 158 ± 53.5 ml/min.

Discussion

In the present study, the effect of the physiological stimulus of upright posture was investigated in 56 subjects with benign essential hypertension, as previously classified (1). Assuming the upright posture induces a sympathetic discharge which plays a major role in orthostatic blood pressure regulation through hemodynamic and hormonal effects (14). Blood pressure in subjects in the upright position is maintained within a physiological range by an initial increase in diastolic pressure and heart rate (15, 16). This hemodynamic phase is followed by a more sluggish hormonal response; an increase in plasma renin activity (17, 18) can be demonstrated within 5 minutes of the assumption of upright posture (19, 20), but aldosterone requires a relatively longer time to stimulate the tubular reabsorption of sodium (22, 23).

Four major findings were observed in subjects with benign essential hypertension in the upright position: (1) an alteration in the blood pressure response, (2) an absence of the usual increase in urinary excretion of norepinephrine, (3) a significant increase in plasma renin activity associated with a significant decrease in plasma norepinephrine in subjects with labile hypertension with a postural tachycardia, and (4) a highly significant correlation between the excretion of sodium and potassium in recumbency and the retention of both ions during upright posture.

In control subjects, there was an increase in mean blood pressure in the upright position due to an increase in diastolic pressure (Fig. 1). With increasing blood pressure indexes in recumbent subjects with benign essential hypertension, the postural increase progressively disappeared and was replaced by a postural decrease in upright blood pressure indexes in subjects with more severe stable hypertension (group IIB). This finding suggests a progressive deterioration of the postural adaptive mechanisms for blood pressure regulation in essential hypertension. In this altered regulation, aging cannot be excluded as an important factor, although no significant correlation was calculated between the age of the subjects and their blood pressure response to upright posture. In subjects with essential hypertension, the excessive decrease in upright systolic pressure could be related to an exaggerated decrease in blood volume (24); this possibility could be predominant in subjects with a more severe stable hypertension (group IIB), because a contraction of plasma volume occurs with diastolic pressures over 105 mm Hg (25). In subjects with essential hypertension, the absence of an increase in upright diastolic pressure could be related to a defect in sympathetic response. However, an altered input to the baroreceptors is probably not the starting factor in the defect, if any, in sympathetic response. In the present study, the differences in pulse pressure
between recumbent subjects and subjects in the upright position were comparable in all five groups. Since a decrease in baroreceptor responsiveness occurs with increasing blood pressure (26), such a mechanism could be partly responsible for the absence of an increase in diastolic pressure in subjects in the upright position. Therefore, it is important to study the response of catecholamines as an indication of the integrated output of the sympathetic system.

In subjects with benign essential hypertension, urinary excretion of both dopamine and norepinephrine showed the same general trend during recumbency and upright posture (Table 1); both were increased in subjects with labile hypertension (groups IA and IB) and decreased in subjects with stable hypertension (groups Ia and IIa). In response to upright posture, urinary excretion of dopamine decreased in each group; however, statistical significance was achieved only in groups R, Ia, and IIa. Urinary excretion of norepinephrine was significantly increased in normotensive subjects but not in any hypertensive subjects; in fact, there was a mean decrease in groups Ia, Ib, and IIa. This absence of an increase in urinary excretion of norepinephrine could result from a deficiency in dopamine-norepinephrine conversion, a possible intrarenal defect, or both. In addition, its relation to the absence of an increase in diastolic pressure cannot be excluded.

In response to upright posture, the urinary ratio of dopamine to norepinephrine decreased in all groups (Table 1), but the decrease was significant only in groups R and Ia. This observation suggests a normal response in subjects with labile hypertension without postural tachycardia (group Ia) only. Assuming that these ratios could be related to either a systemic or an intrarenal activity of dopamine-β-hydroxylase, the absence of an increase in urinary norepinephrine could be explained, at least in subjects with stable hypertension, by a blunted dopamine-β-hydroxylase response. However, a role for a systemic blunted dopamine-β-hydroxylase response is unlikely, since the increase induced by upright posture (9), cold pressure test, and exercise (27) disappeared 1 hour after the beginning of stimulation. In the present study, subjects were standing for 4 hours.

In subjects with labile hypertension, the absence of an increase in urinary norepinephrine could be related to changes in plasma norepinephrine, renal clearance of norepinephrine, or both. Plasma norepinephrine increases in response to upright posture in normotensive subjects (4, 5), and the response is blunted in subjects with essential hypertension (4) after a short period of upright stimulation. In the present study, there was no significant increase in plasma norepinephrine in control subjects after 4 hours of upright posture. In the subjects with labile hypertension and postural tachycardia (group Ib), there was a significant decrease in plasma norepinephrine after 4 hours of upright posture; the decrease in plasma norepinephrine could be related to the absence of an increase in urinary norepinephrine. In the study of renal clearance of norepinephrine, we determined that a negative renal defect in norepinephrine excretion probably was not a major factor in the altered response of urinary norepinephrine in subjects in the upright position; mean renal clearance of norepinephrine was two to three times higher in groups IA and IB, respectively, than it was in group R.

Adrenergic innervation and circulating catecholamines are two important factors involved in the regulation of renin release. Our findings suggest, at least in subjects with labile hypertension, a possible dissociation between both mechanisms in response to upright posture. It should be stressed that for the same systolic, diastolic, mean and pulse pressures in groups Ia and Ib in both the recumbent and upright positions there was a higher orthostatic tachycardia in group Ib (Table 1). In group Ib there was a significant increase in plasma renin activity associated with a significant decrease in plasma norepinephrine. Therefore, the increase in plasma renin activity during upright posture in group Ib probably did not depend on circulating norepinephrine levels. A nerve-dependent mechanism could be involved because a significant correlation was calculated between the increase in heart rate and the increase in plasma renin activity in response to upright posture (Fig. 3) and because the increase in plasma renin activity in group Ib was associated with the highest heart rate acceleration. A receptor abnormality, however, cannot be excluded, because in subjects with labile hypertension there is an increase in urinary excretion of cyclic adenosine monophosphate (cyclic-AMP) during upright position, although no such change has been observed in normotensive subjects (28). The same group of hypertensive subjects has been shown to have renin hyperresponsiveness to posture and isoproterenol infusion (29), and propranolol has been shown to normalize heart rate, plasma renin activity, and urinary excretion of cyclic-AMP (30).

The well-known antidiuresis, antinatriuresis,
and antikaliuresis were observed in response to upright posture stimulation. In addition, a highly significant correlation between the excretion of sodium and potassium in recumbency and the retention of both ions in the upright position was observed (Fig. 2); this observation suggests that the magnitude of antinatriuresis and antikaliuresis in subjects in the upright position is directly related to the magnitude of natriuresis and kaliuresis in recumbent subjects. The mechanism involved is difficult to assess; homeostatic regulation of glomerular filtration rate, the so-called glomerulotubular balance, is probably a major factor because the same type of correlation was observed between the excretion of creatinine by recumbent and upright subjects.

Our findings suggest that there is a progressive deterioration of the blood pressure adaptive mechanism to upright posture in subjects with benign essential hypertension. This finding raises an important practical question. Orthostatic hypotension occurs easily after potent sympatholytic treatment; therefore, this kind of treatment could be questionable if a preexisting disturbance is present. We also found an absence of an increase in diastolic pressure in subjects in the upright position; this lack could be related to a relative deficiency in the sympathetic response to upright posture. The present study suggests a major role for a nerve-dependent mechanism or for receptor responsiveness in renin release in subjects with labile hypertension and postural tachycardia in the upright position. Finally, we found that the orthostatic regulation of sodium directly depends on the excretion of sodium during recumbency. Whether such a response is part of a larger homeostatic mechanism remains a fascinating question.

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References

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