Norepinephrine in Urine and Plasma Following Provocative Maneuvers in Normal and Hypertensive Subjects

DAVID P. HENRY, M.D., FRIEDRICH C. LUFT, M.D., MYRON H. WEINBERGER, M.D., NAOMI S. FINEBERG, PH.D., AND CLARENCE E. GRIM, M.D.

SUMMARY Urinary norepinephrine (UNE) excretion rate and venous plasma norepinephrine (PNE) concentrations were studied in 266 normotensive and 107 essential hypertensive men and women under conditions of volume expansion with 2 liters of intravenous normal saline over 4 hours, and volume contraction with a 10 mEq sodium diet and 120 mg oral furosemide. The UNE excretion rate was correlated with age in normal women only. In men, and in hypertensives of both sexes, the relationship appeared to be biphasic. The PNE concentration was not correlated with age in the hypertensive subjects. Insufficient numbers of older subjects were available to exclude absolutely such a relationship among normals. The UNE and PNE were influenced by volume expansion and contraction in both normals and hypertensives; however, normals exhibited a correlation between UNE and blood pressure as well as consistent correlations between UNE and PNE, neither of which were observed in the hypertensives. Hypertensive women generally had greater UNE and PNE values than normal women or hypertensive men. Hypertensive women may have altered sympathetic activity.

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KEY WORDS • essential hypertension • norepinephrine • urine • plasma • sympathetic nervous system

The sympathetic nervous system plays a major role in regulating the blood pressure of normal persons1 and has also been implicated in the development of experimental hypertension,2 and of certain forms of human essential hypertension.3-6 Norepinephrine concentrations in plasma (PNE) and urine (UNE) have been proposed as parameters of generalized sympathetic nervous system activity.6 In the present report, we describe the effects of volume expansion and contraction on the rate of UNE excretion and venous PNE concentration in two large groups of men and women, one normal, the other with essential hypertension. Our results extend previous observations that age and sex exert important influences on UNE excretion.7,8 The state of body fluid volume and posture influence both UNE and PNE.

Also, the relationships between UNE and PNE differ between normal and hypertensive subjects. Hypertensive women may exhibit altered sympathetic nervous activity compared to normal women or hypertensive men.

Methods

Since January 1974, hypertensive patients referred to Indiana University Medical Center for evaluation have been studied according to a protocol designed for the efficient diagnosis of secondary forms of hypertension and establishment of a renin profile if they are found to have essential hypertension.8 Normal values for the humoral studies and for the natriuretic and kaliuretic responses engendered by the protocol were derived from large numbers of black and white normal men and women who answered our advertisement.10 The hypertensive patients were admitted to either the Clinical Research Center or a subspecialty ward of the Indiana University Hospital designated for endocrinologic or hypertensive patients. The normal volunteers were studied in the Clinical Research Center. The protocol was approved by the Indiana University Medical Center Human Use Committee, and informed consent was obtained after detailed explanation of the procedures to be performed.
Our study includes 266 normal subjects and 107 patients with categorized essential hypertension. In the hypertensives, secondary causes for blood pressure elevation were ruled out by the protocol, which is described in detail elsewhere.11 The hypertensive subjects were classified into "low," "normal," and "high" renin categories depending upon their renin responses to volume expansion and contraction. The renin classification system was devised so as to take into account the patient's age and race, both of which have been found to influence renin and aldosterone responses.12-15 Neither normotensive nor hypertensive subjects were taking medications for at least 2 weeks prior to the study. Estrogens and spironolactone were discontinued for at least 1 month prior to the study because of their known prolonged effects on components of the renin-angiotensin-aldosterone system.16, 17

Dietary sodium and potassium were unrestricted prior to admission. In the hospital, dietary sodium intake was 150 mEq/day and potassium 70 mEq/day unless otherwise indicated. Each subject was instructed to collect a 24-hour urine specimen on the day prior to admission to provide an estimate of glomerular filtration rate, previous dietary sodium, and potassium intake. Urinary sodium (UNa) excretion, urinary potassium (UK) excretion, and creatinine clearance (CCr) were determined for each subject.

On the day of admission, each subject was carefully examined, and baseline hemogram and plasma chemistries were obtained. Blood pressure measurements made on this and subsequent days were obtained four times daily by specially trained nurses using mercury manometers (Baum, Inc., New York, NY), with the subjects in the supine and upright positions. The fifth Korotkoff component, the point of sound disappearance, was accepted as the diastolic pressure. Mean arterial blood pressure (MAP) was measured using radioenzymatic assay.17 Acetic acid was added to the urine collection containers as a preservative. The NE was converted to radio-labeled epinephrine using partially purified bovine adrenal phenylethanolamine N methyl transferase and tritiated S-adenosylmethionine. The epinephrine formed was isolated by batch alumina adsorption chromatography, and quantified using liquid scintillation spectrophotometry.

Concentrations of Na and K in plasma and urine were measured by flame photometry (Instrumentation Laboratories, Boston, MA), the Cr by an automated technique (Technicon, Chauncey, NY), and the PRA and PA by radioimmunoassay methods previously reported.18 Surface area corrections were estimated by Dubois' equation. The data were analyzed statistically by analysis of variance, t test, and multiple regression analysis, where appropriate.19 The 95% limits of probability were accepted as significant. Data are expressed as mean ± SEM.

Results

Table 1 outlines the general characteristics of the normal subjects and patients with essential hypertension, and their significant differences on the day of admission. The hypertensive subjects were older, heavier, and had higher mean arterial blood pressures (p < 0.05); the men weighed more than the women (p < 0.05), and normal men had higher blood pressures than normal women (p < 0.05). Hypertensive men had lower PNa values than normal men (p < 0.05). Both hypertensive men and women had lower PK values than normal subjects (p < 0.05);
women, regardless of blood pressure, had lower PK values than men (p < 0.05). The PCr values were higher in men than in women (p < 0.05). This suggests only minor differences in dietary Na intake among the groups. The UCr excretion of men exceeded that of women (p < 0.05).

Table 2 shows the UNE concentrations during each of the collection periods, expressed as micrograms per hour and also as micrograms per hour per square meter. In the 110 normal women, there was no correlation between UNE excretion rate and body surface area during any of the eight time periods studied. In the 156 normal men, UNE was weakly associated with body surface area; however, the correlation coefficient never exceeded r = 0.09. The correlation between UNE excretion rate and body surface area during any of the eight time periods studied. In the 156 normal men, UNE was weakly associated with body surface area; however, the correlation coefficient never exceeded r = 0.09. This

<table>
<thead>
<tr>
<th>Variable</th>
<th>Normal men</th>
<th>Normal women</th>
<th>Hypertensive men</th>
<th>Hypertensive women</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of subjects</td>
<td>156</td>
<td>110</td>
<td>54</td>
<td>53</td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>25.5 ± 0.9</td>
<td>30.1 ± 1.3</td>
<td>37.7 ± 1.5†</td>
<td>44.4 ± 1.5‡</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>80.7 ± 1.1</td>
<td>62.9 ± 1.3†</td>
<td>85.0 ± 1.6‡</td>
<td>72.6 ± 1.8‡</td>
</tr>
<tr>
<td>MABP (mm Hg)</td>
<td>89.7 ± 0.6</td>
<td>85.5 ± 0.6†</td>
<td>120.4 ± 1.4†</td>
<td>118.3 ± 1.4‡</td>
</tr>
<tr>
<td>PNa (mEq/l)</td>
<td>14.0 ± 0.4</td>
<td>140.3 ± 0.4</td>
<td>139.8 ± 0.2*</td>
<td>139.5 ± 0.2</td>
</tr>
<tr>
<td>PK (mEq/l)</td>
<td>4.3 ± 0.04</td>
<td>4.2 ± 0.04†</td>
<td>4.1 ± 0.03†</td>
<td>4.0 ± 0.03*</td>
</tr>
<tr>
<td>PCr (mg/dl)</td>
<td>1.10 ± 0.03</td>
<td>0.89 ± 0.02†</td>
<td>1.19 ± 0.04‡</td>
<td>0.94 ± 0.03†</td>
</tr>
<tr>
<td>UNa excretion (mEq/24 hr)</td>
<td>150 ± 5</td>
<td>133 ± 6†</td>
<td>142 ± 10</td>
<td>137 ± 10</td>
</tr>
<tr>
<td>UK excretion (mEq/24 hr)</td>
<td>47 ± 2</td>
<td>44 ± 2</td>
<td>49 ± 4</td>
<td>44 ± 3</td>
</tr>
<tr>
<td>UCr (g/24 hr)</td>
<td>1.55 ± 0.04</td>
<td>1.25 ± 0.04†</td>
<td>1.65 ± 0.08</td>
<td>1.19 ± 0.05†</td>
</tr>
<tr>
<td>CCr (ml/min)</td>
<td>130 ± 7</td>
<td>100 ± 3†</td>
<td>126 ± 8</td>
<td>97 ± 4‡</td>
</tr>
</tbody>
</table>

Abbreviations: MABP = mean arterial blood pressure; PNa = plasma sodium; PK = plasma potassium; PCr = plasma creatinine; UNa = urine sodium; UK = urine potassium; UCr = urine creatinine; CCr = creatinine clearance.

*Normals differ from hypertensives within sex (p < 0.05).
†Males differ from females within blood pressure group (p < 0.05).
‡Hypertensives differ from normals within sex (p < 0.05).

Table 2. Urinary Norepinephrine Excretion (μg/hr) under Conditions of Volume Expansion and Contraction (Mean ± SEM)

<table>
<thead>
<tr>
<th>Urine Collections</th>
<th>Normal men</th>
<th>Normal women</th>
<th>Hypertensive men</th>
<th>Hypertensive women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal “night”</td>
<td>1.04 ± 0.04</td>
<td>0.96 ± 0.05*</td>
<td>1.10 ± 0.07</td>
<td>1.25 ± 0.11†‡</td>
</tr>
<tr>
<td>4-hr saline infusion</td>
<td>1.53 ± 0.06</td>
<td>1.28 ± 0.05*</td>
<td>1.78 ± 0.24</td>
<td>1.63 ± 0.13†</td>
</tr>
<tr>
<td>Saline “day”</td>
<td>1.74 ± 0.05</td>
<td>1.60 ± 0.06*</td>
<td>1.68 ± 0.11</td>
<td>1.85 ± 0.11†</td>
</tr>
<tr>
<td>Saline “night”</td>
<td>0.93 ± 0.04</td>
<td>0.82 ± 0.03*</td>
<td>1.06 ± 0.08</td>
<td>0.98 ± 0.07†</td>
</tr>
<tr>
<td>Saline 24 hr</td>
<td>1.77 ± 0.05</td>
<td>1.59 ± 0.06*</td>
<td>1.77 ± 0.13</td>
<td>1.92 ± 0.13†</td>
</tr>
<tr>
<td>Furosemide “day”</td>
<td>2.27 ± 0.08</td>
<td>1.95 ± 0.07*</td>
<td>2.38 ± 0.13</td>
<td>2.19 ± 0.13†</td>
</tr>
<tr>
<td>Furosemide “night”</td>
<td>1.48 ± 0.05</td>
<td>1.39 ± 0.06</td>
<td>1.62 ± 0.11</td>
<td>1.88 ± 0.13‡</td>
</tr>
<tr>
<td>Furosemide 24 hr</td>
<td>2.05 ± 0.06</td>
<td>1.79 ± 0.06*</td>
<td>2.16 ± 0.12</td>
<td>2.11 ± 0.12‡</td>
</tr>
</tbody>
</table>

Abbreviations: UNe = urine norepinephrine; UNe corrected for surface area (μg/hr/m²).

Values in parentheses denote UNe corrected for surface area (μg/hr/m²).

*Normal men differ from normal women (p < 0.05).
†Hypertensives differ from normals within sex (p < 0.05).
‡Hypertensive women differ from hypertensive men (p < 0.05).
relationship explained less than 4% of the observed variance. Among hypertensives, the opposite situation was observed. No significant correlations between body surface area and UNE were identified in the hypertensive men, whereas a significant correlation ($r = 0.30, p < 0.05$) was observed on the saline day in the hypertensive women.

Normal men generally excreted UNE at a greater rate than normal women regardless of the volume state. Hypertensive women generally excreted UNE at a greater rate than normotensive women, a feature that was not observed when comparing hypertensive to normotensive men. In both normal subjects and hypertensives, daytime UNE excretion rates exceeded those observed during the night collection periods ($p < 0.05$). Moreover, volume contraction with furosemide resulted in greater UNE excretion rates than those observed with saline-induced volume expansion when the respective day and night collection periods were compared ($p < 0.05$).

When corrected for body surface area, the UNE of normal men and normal women was not different; however, the values of hypertensive women generally exceeded those of hypertensive men. The UNE values of hypertensive women were greater than those of normal women with or without surface area correction.

Only normal women showed a significant correlation between age and UNE (fig. 1) in each collection period; for the total UNE on the volume expansion day, the correlation was $r = 0.34$ ($p < 0.001$). The data in figure 1 are from women in the second to sixth decades of life; insufficient numbers of older normal women were available to give an accurate assessment of UNE excretion past the sixth decade. Similarly,
there were few hypertensive women in the second decade. No linear relationship between age and UNE was identified in hypertensive women (fig. 1), normotensive men, or hypertensive men (fig. 2). Rather, the values increased until middle life and then decreased.

In normal men and women, UNE was directly correlated with mean blood pressure on the volume expansion day \((r = 0.24, p < 0.001; r = 0.28, p < 0.001)\) and on the volume contraction day \((r = 0.36, p < 0.001; r = 0.27, p < 0.01)\). A multivariate analysis was performed removing the effects of age and body surface area in the normal subjects. Nevertheless, the significant correlation between UNE and mean blood pressure persisted \((r = 0.21, p < 0.05)\). In hypertensive men and women, no correlations between UNE and mean blood pressure were identified in either the volume expanded or volume contracted state. When systolic and diastolic blood pressures were examined separately in the normal and hypertensive subjects, the results were not different from those identified using the mean blood pressure values. No consistent correlations between PNE and systolic, diastolic, or mean blood pressure were observed in normotensive or hypertensive men and women.

The normal and hypertensive subjects were subdivided into low, normal, and high PRA subgroups according to their age and race-adjusted PRA responses following volume expansion and contraction. No
differences in the absolute values of PNE or UNE, under conditions of either volume expansion or contraction, were observed in normal or hypertensive subgroups regardless of sex.

The PNE values (table 3) were uniformly increased by upright posture \((p < 0.05)\), and by volume depletion with furosemide in both the supine and upright states \((p < 0.05);\) these increased values were not suppressed by acute volume expansion with intravenous saline. Hypertensive women generally had higher PNE values than normotensive women or hypertensive men.

Relationships between age and PNE values in our normal subjects could not be established, as, unfortunately, only eight of our normal subjects were over 40 years of age. Among hypertensive subjects, however, the age distribution was adequate, with at least seven subjects in each decade through the sixth decade. No relationship between age and PNE values was identified in these hypertensive subjects.

Recumbent PNE (at 6 a.m.) and UNE of the previous night were correlated in normal men and women respectively as follows: basal day, \(r = 0.48, p < 0.001, r = 0.55, p < 0.05;\) after volume expansion, \(r = 0.24, p < 0.01, r = 0.82, p < 0.001;\) and after volume contraction, \(r = 0.19, p < 0.05, r = 0.63, p < 0.01.\) In hypertensive men, no correlations between recumbent PNE and the previous night's UNE values were identified. In hypertensive women, such a relationship was found only after volume expansion \((r = 0.82, p < 0.001).\)

Discussion

Normal men excreted UNE at a greater rate than normal women under conditions of both volume expansion and contraction. Correction of UNE for surface area removed this difference; however, the inconsistencies and low degrees of association in the UNE surface-area relationships within the sex groups suggest that corrections for body surface area may not be justified. Our findings are at variance with those reported by Cuche et al.,\(^2\) who observed that adult women excreted significantly more UNE than age-matched men during recumbency; during upright posture, however, the men excreted a greater degree of UNE than women. Cuche et al. utilized 4-hour urine collections under standardized conditions and expressed their data in terms of body surface area, although they did not specifically state that UNE was correlated with body surface area in their subjects. Contrary to previous observations,\(^3\) the PNE values of normal men and women were not different under any of the conditions tested in our study.

In our study, UNE excretion increased with age in normal women, but not in normal men or in hypertensive men and women. Increases in UNE excretion with age have been described previously by Weidmann and colleagues;\(^2\) in their study, however, the effect of sex was not examined. Cuche et al.\(^2\) were unable to correlate UNE excretion with age in a study of 70 normotensive and hypertensive subjects. It is likely that the relationships between age and UNE are influenced by a number of variables. The effect of age on plasma catecholamines remains conjectural. Lake et al.,\(^2\) Pedersen and Christensen,\(^2\) and Sever et al.\(^2\) observed that PNE increased with age; however, DeChamplain and Cousineau\(^2\) and DeQuattro and Chan\(^2\) found no such relationship. Unfortunately, our data cannot resolve the issue since our study did not include sufficient samples from older normal persons; however, among hypertensives who were evenly dis-

### Table 3. Plasma Norepinephrine Concentration (pg/ml) under Conditions of Volume Expansion and Contraction (Mean ± SEM)

<table>
<thead>
<tr>
<th>Day tested</th>
<th>Normal men</th>
<th>Normal women</th>
<th>Hypertensive men</th>
<th>Hypertensive women</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Saline day</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 a.m.</td>
<td>143 ± 11 (78)</td>
<td>151 ± 38 (15)</td>
<td>126 ± 20 (20)</td>
<td>192 ± 30 (12)</td>
</tr>
<tr>
<td>8 a.m.</td>
<td>247 ± 18 (82)</td>
<td>241 ± 41 (18)</td>
<td>216 ± 24 (42)</td>
<td>327 ± 43 (31)*†</td>
</tr>
<tr>
<td>12 noon</td>
<td>134 ± 10 (82)</td>
<td>133 ± 19 (18)</td>
<td>128 ± 15 (41)</td>
<td>176 ± 24 (31)*†</td>
</tr>
<tr>
<td><strong>Furosemide day</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Prior to drug)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 a.m.</td>
<td>143 ± 17 (81)</td>
<td>153 ± 38 (16)</td>
<td>100 ± 16 (21)</td>
<td>233 ± 39 (12)†</td>
</tr>
<tr>
<td>8 a.m.</td>
<td>259 ± 16 (81)</td>
<td>281 ± 47 (18)</td>
<td>256 ± 23 (40)</td>
<td>345 ± 42 (29)*†</td>
</tr>
<tr>
<td><strong>Post-furosemide day</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 a.m.</td>
<td>240 ± 15 (79)</td>
<td>292 ± 65 (16)</td>
<td>227 ± 30 (20)</td>
<td>326 ± 47 (12)†</td>
</tr>
<tr>
<td>8 a.m.</td>
<td>470 ± 32 (79)</td>
<td>572 ± 121 (18)</td>
<td>450 ± 41 (39)</td>
<td>550 ± 71 (26)</td>
</tr>
</tbody>
</table>

Numbers in parentheses denote number of subjects.

*Hypertensives differ from normals within sex \((p < 0.05)\).
†Hypertensives females differ from hypertensive males \((p < 0.05)\).
tributed between the second and sixth decade, we found no such relationship.

We found that UNE excretion during the day exceeded that during the night in both normotensive and hypertensive subjects. Similar responses were observed by Berglund et al.\(^2\) These findings were not unexpected since UNE is influenced by upright posture, activity, work, and stress.\(^2\) Similarly, the volume-contracted state was characterized by a greater excretion of NE than the volume-expanded state in both normotensive and hypertensive men and women. These findings confirm previous reports from this laboratory and elsewhere indicating that both UNE excretion and PNE values are inversely correlated with Na intake.\(^9\)-\(^13\) The PNE responded to provocative maneuvers in a fashion similar to UNE excretion. Supine posture decreased PNE, while upright posture and volume contraction increased it. These observations suggest an important role of the sympathetic nervous system in maintaining blood pressure homeostasis during Na and volume perturbations in normal humans.

The suggestion that the sympathetic nervous system is involved in the pathogenesis of hypertension stems from the observations of Goldenberg and colleagues\(^2\) who noted that injections of NE could mimic the hemodynamic changes observed in essential hypertension. Since then, attempts to investigate the sympathetic nervous system directly by studying UNE excretion in hypertensive patients or the plasma catecholamine concentration have failed to reveal consistent findings except in pheochromocytoma.\(^23\) In previous studies, urinary catecholamines have been found to be increased,\(^34\)-\(^41\) normal,\(^42\)-\(^45\) or decreased\(^46\)-\(^49\) in patients with essential hypertension. These discrepancies may stem from differences in technique since many of the older studies relied on the less sensitive or specific bioassay and fluorometric methods. Not all of the patients in previous studies were in comparable states of sodium balance, had comparable degrees of renal function, or had been deprived of antihypertensive medications for similar periods of time. Elevated plasma catecholamine values in hypertensives have been observed in several previous studies.\(^50\)-\(^57\) However, Lake et al.\(^58\) found no difference when their data were subjected to age correction. In addition to age matching, the selection of control subjects and differences in methodology may account for the discrepancies in studies reporting plasma catecholamines.\(^59\)

In our present study, we observed a number of consistent differences between normal and hypertensive subjects. Normal subjects exhibited a correlation between UNE and blood pressure while hypertensive subjects did not. Hypertensive women excreted more NE than normal women. In addition, their plasma values were generally greater than those of normal women. Furthermore, hypertensive women had greater PNE and UNE/m² values than hypertensive men. These findings raise the possibility that hypertensive women may differ from hypertensive men by virtue of altered sympathetic activity. Sever\(^60\) has suggested that a subgroup of young hypertensives has an altered sympathetic activity that may contribute to their hypertension; however, the hypertensive women in our study were older than the normal women, not different in age from hypertensive men, and failed to exhibit age-related correlations with either PNE or UNE.

Although hypertensive subjects exhibited similar responses in UNE and PNE values to provocative maneuvers, hypertensives differed from normal subjects in that supine PNE was not correlated with the previous night's UNE. The NE appearing in the urine is only a very small fraction of that produced and utilized in the body.\(^61\) In addition, there is evidence that the NE appearing in the urine may be derived from the kidneys themselves rather than reflecting PNE.\(^62\) Since the kidneys are influenced by a variety of complex physical and humoral factors,\(^63\) it is possible that these may modulate NE excretion. Such influences may differ between women, men, normotensives, and those with fixed hypertension.\(^64\),\(^65\)

We also compared the UNE and PNE values of subjects with low, normal, and high renin forms of essential hypertension. No differences in either measurement could be discerned among these hypertensive subgroups in men and women. Although Weidmann et al.\(^66\) were unable to correlate PRA levels with UNE excretion in hypertensive subjects, they nevertheless found that patients with low PRA values showed the lowest, and those with high PRA values showed the highest, mean UNE excretion rates. In addition, Esler et al.\(^67\) have suggested that high renin hypertension in young males may be a form of neurogenic hypertension. They based their conclusion on levels of PNE and the results of responses to autonomic blockade. Young males as a group were not specifically studied in this investigation. The reason for the discrepancy between the observations of Weidmann et al.,\(^68\) Esler et al.,\(^69\) and our own findings may stem in part from differences in renin profiling. Our technique relied on volume expansion and contraction maneuvers rather than on a renin-sodium index,\(^70\) and also included specific adjustments for age and race.\(^71\)

In summary, our study defined the responses of UNE and PNE to provocative maneuvers. Normal subjects exhibited relationships between recumbent venous PNE and the previous night's UNE, as well as between UNE and blood pressure, that were not observed in hypertensives. Normal women differed from normal men in that they exhibited a correlation between UNE and age, and excreted UNE at a reduced rate under conditions of volume expansion and contraction. Hypertensive women generally had greater UNE and PNE values than normal women or hypertensive men. Hypertensive women may have altered sympathetic activity.

Acknowledgments

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