Nighttime Blood Pressure and Nocturnal Dipping Are Associated With Daytime Urinary Sodium Excretion in African Subjects
Lise Bankir, Murielle Bochud, Marc Maillard, Pascal Bovet, Anne Gabriel and Michel Burnier

*Hypertension* published online Mar 3, 2008;
DOI: 10.1161/HYPERTENSIONAHA.107.105510
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Blood pressure (BP) is known to follow a circadian rhythm, with 10% to 15% lower values during nighttime than during daytime. The absence of a nocturnal BP decrease (dipping) is associated with target organ damage, but the determinants of dipping are poorly understood. We assessed whether the nighttime BP and the dipping are associated with the circadian pattern of sodium excretion. Ambulatory BP and daytime and nighttime urinary electrolyte excretion were measured simultaneously in 325 individuals of African descent from 73 families. When divided into sex-specific tertiles of day:night ratios of urinary sodium excretion rate, subjects in tertile 1 (with the lowest ratio) were 6.5 years older and had a 9.8-mm Hg higher nighttime systolic BP (SBP) and a 23% lower SBP dipping (expressed in percentage of day value) compared with subjects in tertile 3 (P for trend <0.01). After adjustment for age, the SBP difference across tertiles decreased to 5.4 mm Hg (P=0.002), and the SBP dipping difference decreased to 17% (P=0.05). A similar trend across tertiles was found with diastolic BP. In multivariate analyses, daytime urinary sodium and potassium concentrations were independently associated with nighttime SBP and SBP dipping (P<0.05 for each). These data, based on a large number of subjects, suggest that the capacity to excrete sodium during daytime is a significant determinant of nocturnal BP and dipping. This observation may help us to understand the pathophysiology and clinical consequences of nighttime BP and to develop therapeutic strategies to normalize the dipping profile in hypertensive patients. (Hypertension. 2008;51:1-8.)

Key Words: circadian rhythm ♦ glomerular filtration rate ♦ potassium ♦ humans ♦ families
centrations may reduce the capacity of the kidney to excrete NaCl. For this reason, special attention was given to the concentration of sodium in the urine, in addition to its excretion rate. Our results confirm that sodium excretion during daytime and even more so, sodium concentration in the urine, are significant determinants of nighttime BP and of the nocturnal dipping.

Materials and Methods

The study took place in the Seychelles islands, which are populated predominantly by individuals of East African descent. Participants were recruited between August 1999 and January 2002. The study was approved by the ethical committees of the Ministry of Health in the Seychelles and of the University of Lausanne Faculty of Medicine. All of the participants provided written informed consent. The selection process for families has been described previously.23 For the analyses, we used the average of 10 daytime and 10 nighttime randomly selected measures to have the same number of measures for each participant and each period. Sensitivity analyses conducted using all of the available daytime and nighttime BP measures led to very similar results and did not alter our conclusions. In a subgroup of subjects (n = 55), a second off-treatment ABPM was performed after 6 weeks to calculate the reproducibility of the dipping pattern in this population using the Pearson’s correlation coefficient.

Participants were investigated under their usual diet. Twenty-four–hour urine was collected on the same day as ABPM. Urine was collected separately for day and night. Additional methodological criteria have been described previously.24 For the analyses, we used the average of 10 daytime and 10 nighttime randomly selected measures to have the same number of measures for each participant and each period. Sensitivity analyses conducted using all of the available daytime and nighttime BP measures led to very similar results and did not alter our conclusions. In a subgroup of subjects (n = 55), a second off-treatment ABPM was performed after 6 weeks to calculate the reproducibility of the dipping pattern in this population using the Pearson’s correlation coefficient.

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Participants were investigated under their usual diet. Twenty-four–hour urine was collected on the same day as ABPM. Urine was collected separately for day and night. As for BP, day and night were defined according to each participant’s self-reported bedtime and wake-up time. The average±SD durations of the daytime and nighttime urine collections were, respectively, 14.1±1.9 and 9.2±1.6 hours. Blood was drawn under fasting conditions between 7:30 AM and 10:00 AM, just after completion of the ABPM recording and urine collection.

Plasma and urinary sodium and potassium concentrations (PNa, PK) were measured by flame photometry (IL-943, Instrumentation Laboratory). Creatinine concentration was measured by the picric acid method (Cobas-Mira, Richete) and creatinine clearance (Ccr), often used as an approximation of the glomerular filtration rate (GFR), was calculated for daytime and nighttime urine collections separately. The average±SD durations of the daytime and nighttime urine collections were, respectively, 14.1±1.9 and 9.2±1.6 hours. Blood was drawn under fasting conditions between 7:30 AM and 10:00 AM, just after completion of the ABPM recording and urine collection.

Ambulatory BP was measured for 24 hours using Diasys Integra devices (Novacor SA, Rueil-Malmaison) every 20 minutes during the day and every 30 minutes at night. Additional methodological criteria have been described previously.24 For the analyses, we used the average of 10 daytime and 10 nighttime randomly selected measures to have the same number of measures for each participant and each period. Sensitivity analyses conducted using all of the available daytime and nighttime BP measures led to very similar results and did not alter our conclusions. In a subgroup of subjects (n = 55), a second off-treatment ABPM was performed after 6 weeks to calculate the reproducibility of the dipping pattern in this population using the Pearson’s correlation coefficient.
ABPM. Urine osmolality was not measured, but the ratio of urine:plasma creatinine concentrations was used as an index of urine concentration, as validated previously.\textsuperscript{25} Urine flow rate (V) and sodium and potassium excretion rates (\(U_{\text{Na}}^x\)V and \(U_{\text{K}}^x\)V, respectively) were calculated for the whole 24-hour period and for daytime and nighttime separately. Fractional excretion of sodium (\(\text{FENa}\)) was calculated as \(U_{\text{Na}}^x/V/(P_{\text{Na}}^x\cdot C_{\text{Creat}}^x)\).

Reported smoking and alcohol consumption were obtained by trained health professionals using a standardized questionnaire. Measurements of body mass index (BMI) and fasting blood glucose have been described previously.\textsuperscript{26}

### Statistical Analyses

The data were divided into sex-specific tertiles (T1, T2, and T3) according to the day:night ratio of urinary sodium excretion. A nonparametric test was used to evaluate trends across tertiles. The Wilcoxon matched-pairs signed-rank test was used to compare day and night values within each tertile. The ASSOC program (5.2v) in the Statistical Analysis for Genetic Epidemiology package was used to conduct multiple linear regression models while accounting for familial correlations and also to estimate heritability to assess familial aggregation of nocturnal dipping. The following dependent variables were used: night values and differences between daytime and nighttime values (ie, nocturnal dipping) for systolic (SBP), diastolic (DBP) and pulse pressure (PP; ie, 6 models). All of the models were adjusted for age, sex, BMI, ascertainment, 24-hour sodium and potassium excretion, urine flow rate (milliliters per minute), and urinary creatinine concentration in day and night urine. Models with DBP or PP as the dependent variable were also adjusted for age\textsuperscript{2} to account for the nonlinear relation of DBP with age. We used the daytime and nighttime urinary \(U_{\text{Na}}\) and \(U_{\text{K}}\) (millimoles per liter) as the covariates of interest. Sensitivity analyses were conducted that included an additional adjustment for the following: (1) GFR measured using inulin clearance; (2) 24-hour \(\text{FENa}\); (3) reported tobacco consumption; (4) reported alcohol consumption; and (5) fasting blood glucose.

### Results

We stratified the sample using the day:night ratio of urinary sodium excretion rate to evaluate whether a disturbed circadian pattern of sodium excretion was associated with a difference in the level of BP and/or its nocturnal dipping. The characteristics of the 325 subjects, divided into sex-specific tertiles of day:night ratio of urinary sodium excretion rate, are presented in Table 1. Subjects in T3 can be qualified as “high daytime sodium excretors,” because they excrete sodium at a rate that is 46% higher during daytime than during nighttime, whereas subjects in T1 are “low daytime sodium excretors,” excreting 3 times less sodium during daytime than during nighttime. Despite this different circadian pattern of sodium excretion, the total 24-hour sodium excretion was similar across tertiles (Table 1). Subjects in T1 were significantly older than those in T2 and T3 by \(\approx 6\) and 4 years, respectively. In addition, subjects in T1 tended to have a higher BMI and fasting blood glucose than subjects in T3 and a lower creatinine clearance. GFR measured using inulin clearance was similar across tertiles (Table 1). Heritability estimates of SBP, DBP and PP nocturnal dipping were not significantly different from 0 (\(P>0.10\)).

Marked differences in BP were observed across tertiles of the day:night ratio of urinary sodium excretion rate (Figure). During both daytime and nighttime, SBP and DBP were highest in T1, lower in T2, and even lower in T3. The magnitude of the difference was larger for SBP than for DBP, resulting also in a progressive decline in PP values from T1 to T3. The nocturnal BP dipping was significantly lower in T1 than in other tertiles, especially for SBP. Adjustment for age (model M2 in Table 2) but not 24-hour creatinine clearance (model M3) substantially modified the results for nighttime BP, and the percentage dipping in nighttime BP (Table 2). Age explained \(\approx 40\%\) of the trend observed across tertiles. Fully adjusted models (M4), including BMI and 24-hour urinary sodium and potassium excretion showed similar trends than age-adjusted models. The reproducibility of the dipping pattern, estimated using correlation coefficients between BP dipping on 2 ABPMs 6 weeks apart in 55 subjects, was 0.53 for SBP and 0.37 for DBP (\(P<0.001\)).

Although 24-hour urinary sodium and potassium excretion rates were similar across tertiles of the day:night ratio of urinary sodium excretion rate (Table 1), strikingly different excretion rates were observed during day and night. Mean sodium excretion rate was 41 (SD: 24) and 120 (SD: 63) \(\mu\text{mol/min}\) during daytime and nighttime, respectively, in T1 and 82 (SD: 40) and 62 (SD: 36) \(\mu\text{mol/min}\), respectively, in T3. The excretion rate of any given solute is the product of its concentration in the urine by the urine flow rate. The lower sodium excretion rate observed in T1 during daytime was largely because of a low sodium concentration in the urine, whereas the urine flow rate was only marginally lower in T1 than in the 2 other tertiles (Table 3). This defect seemed to be selective for sodium, because other urinary variables, such as the index of overall urinary concentration, the \(U_{\text{K}}\) (Table 3), and that of creatinine (not shown) showed fairly similar values in all of the tertiles during daytime. In subjects in T2...
and T3, the UNa was significantly lower at night than during day, but not in subjects in T1. The fractional excretion of sodium in T1 was much lower during daytime and much higher during nighttime than in the 2 other tertiles: T1 to T3 were 0.33 (SD: 0.19), 0.42 (SD: 0.21), and 0.49 (SD: 0.24) during daytime and 0.74 (SD: 0.45), 0.52 (SD: 0.29), and 0.38 (SD: 0.21) during nighttime, respectively ($P$ for trend $<0.0001$ for both).

The online supplemental table (Table S1), available at http://hyper.ahajournals.org, presents the regression coefficients (and SEs) found in multiple linear regression models (all accounting for familial correlations), 1 for each dependent variable using either concentrations or excretion rates. Daytime sodium concentration was negatively and strongly associated with nighttime SBP, DBP, and PP and positively associated with the nighttime dip of SBP and PP. This suggests that, at constant urine flow rate, subjects who are less able to concentrate sodium in the urine during daytime fail to decrease their BP at night. Coefficients for daytime and nighttime UK were of similar magnitude but in opposite directions as those for UNa (Table S1). Sensitivity analyses that included an additional adjustment for GFR (measured using inulin clearance), 24-hour FENa, tobacco consumption, alcohol intake, or fasting blood glucose did not substantially change the results and led to the same conclusions (results not shown). Urinary sodium excretion (millimoles per 24 hours), a proxy of dietary sodium intake, was positively and independently associated with nighttime SBP, DBP, and PP but was not a significant determinant of nocturnal SBP, DBP, or PP dipping. By contrast, neither GFR, measured using inulin clearance, nor 24-hour FENa was a significant determinant of nocturnal SBP, DBP, or PP dipping.

**Discussion**

The main finding of our study is that, in a large group of subjects from African descent, individuals who are poor daytime sodium excretors have an increased nighttime BP and a blunted nocturnal BP dipping. The magnitude of this effect is highly clinically relevant, because it represents a 10-mm Hg difference for nighttime SBP between the first and third tertiles of the day:night ratio of urinary sodium excretion rate. More importantly, this study provides the first

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Model</th>
<th>All</th>
<th>Tertile 1</th>
<th>Tertile 2</th>
<th>Tertile 3</th>
<th>$P$ (Trend)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SBP, mm Hg</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>M1</td>
<td>118.2 (16.9)</td>
<td>123.3 (15.9)</td>
<td>117.6 (18.0)</td>
<td>113.5 (15.3)</td>
<td>&lt;0.001</td>
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<tr>
<td>M2</td>
<td>119.3 (15.2)</td>
<td>122.2 (14.7)</td>
<td>119.1 (16.1)</td>
<td>116.7 (14.3)</td>
<td>0.002</td>
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<tr>
<td>M3</td>
<td>119.4 (15.2)</td>
<td>122.4 (14.7)</td>
<td>118.9 (16.1)</td>
<td>116.7 (14.2)</td>
<td>0.002</td>
<td></td>
</tr>
<tr>
<td>M4</td>
<td>119.3 (14.9)</td>
<td>122.1 (14.4)</td>
<td>118.9 (16.1)</td>
<td>116.8 (13.9)</td>
<td>0.002</td>
<td></td>
</tr>
<tr>
<td><strong>DBP, mm Hg</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M1</td>
<td>75.9 (11.8)</td>
<td>78.3 (11.4)</td>
<td>75.5 (12.4)</td>
<td>73.7 (11.0)</td>
<td>0.004</td>
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</tr>
<tr>
<td>M2</td>
<td>76.8 (11.0)</td>
<td>78.1 (11.1)</td>
<td>76.3 (11.7)</td>
<td>75.9 (10.0)</td>
<td>0.13</td>
<td></td>
</tr>
<tr>
<td>M3</td>
<td>76.8 (11.0)</td>
<td>78.2 (11.1)</td>
<td>76.3 (11.6)</td>
<td>75.9 (10.1)</td>
<td>0.10</td>
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</tr>
<tr>
<td>M4</td>
<td>76.7 (10.9)</td>
<td>78.0 (10.9)</td>
<td>76.3 (11.6)</td>
<td>75.9 (9.9)</td>
<td>0.12</td>
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</tr>
<tr>
<td><strong>PP, mm Hg</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M1</td>
<td>42.3 (10.5)</td>
<td>45.0 (10.1)</td>
<td>42.1 (11.3)</td>
<td>39.8 (9.3)</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>M2</td>
<td>43.1 (9.5)</td>
<td>44.6 (9.5)</td>
<td>43.2 (9.8)</td>
<td>41.4 (9.2)</td>
<td>0.006</td>
<td></td>
</tr>
<tr>
<td>M3</td>
<td>43.1 (9.5)</td>
<td>44.7 (9.5)</td>
<td>43.2 (9.9)</td>
<td>41.4 (9.1)</td>
<td>0.006</td>
<td></td>
</tr>
<tr>
<td>M4</td>
<td>43.1 (9.5)</td>
<td>44.5 (9.5)</td>
<td>43.1 (9.8)</td>
<td>41.5 (9.0)</td>
<td>0.010</td>
<td></td>
</tr>
</tbody>
</table>

Results are means (SDs). Night BP dipping is the difference between night and day BPs, expressed in percentage of day BP. M1 indicates unadjusted model; M2, model adjusted for age; M3, model adjusted for age and 24-hour creatinine clearance; M4, model adjusted for age, BMI, 24-hour sodium, and potassium excretions (millimoles per 24 hours), and 24-hour creatinine clearance.

* M4 is the model adjusted for baseline BP in addition to covariates listed for M4.
evidence that the low sodium excretion during daytime is due essentially to an inability to concentrate sodium in the urine, because no significant difference in urinary volume was found across tertiles of the day:night ratio of urinary sodium excretion rate, and nighttime BP was negatively and highly significantly associated with daytime urine sodium concentration. This relationship was still significant after adjustment for total 24-hour sodium and potassium excretion and for the other possible considered confounding factors. Familial clustering had no major influence on our results, because heritability estimates for nocturnal BP dipping were not significantly different from 0. This is, to our knowledge, the largest study to date that has explored the relationships between the circadian pattern of fluid and sodium excretion and those of BP, either because ABPM was not performed or because urinary electrolyte excretion was not measured separately during daytime and nighttime.

Some authors have shown limited reproducibility of the nocturnal BP dipping in individual subjects. When ABPM was repeated, a 40% change in the dipping or nondipping pattern was observed in a large Italian clinical study. In the present study, the correlation coefficients between repeated ABPM were 0.53 for SBP dipping and 0.37 for DBP dipping, which show significant but moderate reproducibility. One potential limiting factor for an adequate determination of the dipping pattern is the difference in the number of BP measurements between daytime and nighttime. To avoid any imbalance in the number of measurements, 10 daytime and 10 nighttime BP values were randomly selected for each subject to calculate mean daytime and nighttime BPs. In each tertile, there was no significant difference in mean daytime or nighttime BP between 10 and all of the measures. Our finding of a significant association between the circadian variations in urinary electrolyte excretion and those in BP and the first such study in a population of African descent.

Several previous studies have suggested that a reduced nocturnal fall in BP is associated with a reduced excretion of sodium during daytime. This has led to the hypothesis that the relative increase in blood pressure observed during the night is a pressure-natriuresis mechanism favoring a compensatory rise in sodium excretion and the maintenance of sodium balance. In accordance with this hypothesis, small groups of subjects with salt-sensitive hypertension exhibiting a nondipping profile have been found to recover a normal dipping pattern after 1 week of sodium restriction or administration of thiazide diuretics. In contrast, salt-sensitive subjects show a normal nocturnal dipping that is not modified by the low-salt diet or the diuretic. A blunted circadian pattern of fluid and sodium excretion has recently been described in young normotensive African Americans, a population with high prevalence of sodium-sensitive hypertension and known to exhibit a lower dipping of night-time BP than whites. However, none of these latter studies could analyze the association between the circadian changes in sodium excretion and those of BP, either because ABPM was not performed or because urinary electrolyte excretion was not measured separately during daytime and nighttime.

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Our data reveal, for the first time in a large group of subjects, the wide range of interindividual variation in the circadian pattern of sodium excretion. The mean day:night ratio of sodium excretion rate varied over more than a 3-fold range between the 2 extreme tertiles. T1 subjects excreted 64.6% of their total daily sodium during nighttime versus

<table>
<thead>
<tr>
<th>Variable</th>
<th>All</th>
<th>Tertile 1</th>
<th>Tertile 2</th>
<th>Tertile 3</th>
<th>P (Trend)</th>
</tr>
</thead>
<tbody>
<tr>
<td>V, mL/min</td>
<td>1.15 (0.79)</td>
<td>1.06 (0.84)</td>
<td>1.22 (0.83)</td>
<td>1.17 (0.68)</td>
<td>0.05</td>
</tr>
<tr>
<td>U\text{ur} /P\text{creat}</td>
<td>141 (106)</td>
<td>140 (112)</td>
<td>143 (114)</td>
<td>140 (91)</td>
<td>0.40</td>
</tr>
<tr>
<td>U\text{Na}</td>
<td>72 (51)</td>
<td>56 (42)</td>
<td>70 (45)</td>
<td>90 (58)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>U\text{K}</td>
<td>42 (30)</td>
<td>42 (29)</td>
<td>42 (30)</td>
<td>42 (30)</td>
<td>0.88</td>
</tr>
<tr>
<td>V, mL/min</td>
<td>1.77 (0.97)</td>
<td>2.17 (1.09)</td>
<td>1.74 (0.94)</td>
<td>1.41 (0.68)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>U\text{ur} /P\text{creat}</td>
<td>94 (68)</td>
<td>74 (52)</td>
<td>103 (80)</td>
<td>107 (65)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>U\text{Na}</td>
<td>59 (37)</td>
<td>63 (36)</td>
<td>64 (43)</td>
<td>51 (32)</td>
<td>0.008</td>
</tr>
<tr>
<td>U\text{K}</td>
<td>19 (14)</td>
<td>16 (12)</td>
<td>20 (14)</td>
<td>20 (16)</td>
<td>0.08</td>
</tr>
<tr>
<td>D/N V</td>
<td>0.72 (0.45)</td>
<td>0.51 (0.32)</td>
<td>0.75 (0.40)</td>
<td>0.91 (0.50)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>D/N U\text{ur} /P\text{creat}</td>
<td>1.66 (1.02)</td>
<td>2.03 (1.26)</td>
<td>1.53 (0.93)</td>
<td>1.43 (0.68)</td>
<td>0.0001</td>
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<td>D/N U\text{Na}</td>
<td>0.85 (0.59)</td>
<td>0.36 (0.12)</td>
<td>0.73 (0.13)</td>
<td>1.46 (0.63)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>D/N U\text{K}</td>
<td>1.52 (0.85)</td>
<td>1.16 (0.69)</td>
<td>1.49 (0.69)</td>
<td>1.89 (0.99)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>D/N U\text{ur} /V</td>
<td>1.43 (0.52)</td>
<td>1.24 (0.47)</td>
<td>1.41 (0.49)</td>
<td>1.64 (0.63)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

$U_{\text{ur}} /P_{\text{creat}}$ indicates urine to plasma creatinine concentrations; $U_{\text{ur}}$, urine creatinine concentration; D/N, daytime/nighttime.
potassium, sodium is not known to undergo active secretion. Note that $U_{Na}$, even in the “good” daily excretors of T3, is only 90 mmol/L during daytime, a value distinctly lower than that of plasma and extracellular fluids. In T1 subjects, a too-intense neurohormonal activity and/or some genetic factors could enhance tubular reabsorption in the proximal tubule and/or in the distal nephron.

An increase in potassium intake has also been shown to reduce BP. Here, we find a positive association between the nocturnal BP dipping and urinary potassium excretion rate during daytime, a relationship that is independent of and inverse to that with sodium. Potassium usually exhibits variations in its excretion rate during daytime and nighttime that are of a much greater amplitude than the rates for other electrolytes and creatinine, and a large fraction of the total 24-hour potassium is excreted during daytime. Recent studies in rats suggest that the beneficial effect of potassium on BP may be because of the fact that its secretion induces an increase in urine flow rate selectively during the active period (nighttime for rats but daytime for humans), thus enhancing the day:night ratio of the sodium excretion rate.

This study has some advantages and some limitations. Daytime and nighttime durations were not arbitrarily fixed for all of the participants but corresponded with each subject’s own rhythm, and daytime in our sample ranged from 9 to 19 hours (interquartile range: 13 to 15 hours). The cross-sectional nature of this study cannot disentangle causes from consequences: it cannot differentiate whether an inability to excrete sodium during daytime increases BP at nighttime or whether an increased nighttime BP induces a greater proportion of sodium to be excreted during the night and a lesser proportion during the following day. However, the experimental evidence that diuretics or a low-salt diet are able to restore a normal BP dipping in nondippers is consistent with the first sequence of events. Although this does not compensate for a truly random design, the large number of subjects in our study allowed controlling analytically for several potential confounders. Also, we do not know the timing of meals and, hence, the participants’ sodium loads. We cannot exclude that T1 subjects ate more salt in the evening than during daytime and, in this respect, differ from T3 subjects. This is, however, unlikely, because large and systematic differences in eating habits across tertiles would be needed to explain our findings. Part of the increased nighttime BP could be due to having to get up at night because of higher urine volume. This may additionally contribute to poor sleep quantity and quality, possibly induced by cuff inflation for ABPM. Such sleep disturbances may reduce the prognostic significance of nighttime BP.

**Perspectives**

Because nighttime BP is associated with target organ damages and cardiovascular events, it is crucial to identify its clinical and physiological determinants. In this study performed on a large group of normotensive and hypertensive subjects, we demonstrate that sodium excretion during daytime is a significant determinant of nighttime BP. This observation has 2 important implications. First, it highlights the importance of considering sodium intake as a major...
confounding factor whenever investigating or trying to explain circadian variations of BP. Second, because a low-sodium diet and diuretics have been found to restore a normal diurnal pattern of BP in nondippers, our findings further support the recommendation of a low-sodium intake in hypertension not only to lower BP but also to restore a normal diurnal rhythm of BP and, hence, to further reduce the patients’ cardiovascular risk.

Acknowledgments
We thank the Ministry of Health of the Republic of Seychelles for its support of this epidemiological research and Air Seychelles and SkyChef for their logistic support in transporting equipment and samples.

Sources of Funding
The study benefited from a grant from the Swiss National Science Foundation (TANDEM No. 31-11151.97). M.B. is supported by a SkyChef for their logistic support in transporting equipment. We thank the Ministry of Health of the Republic of Seychelles for its support of this epidemiological research and the US Public Health Service resource grant (1 P41 RR03655) from the National Center for Research Resources.

Disclosures
None

References


