The Influence of Physical Activity on the Variability of Ambulatory Blood Pressure
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The aim of this study was to assess the contribution of physical activity levels to blood pressure (BP) variability, and to assess the effect of age, gender, body mass index, and use of antihypertensive medications on this relationship. We simultaneously monitored 24-h ambulatory BP by automated recorder and activity by actigraphy in 431 patients. Mean activity scores for the 5, 10, 15, and 20 min preceding each BP measurement were calculated, and BP and heart rate were related to these variables using linear mixed model regression. Various patient characteristics were added to the mixed model as covariates. Patients were heterogeneous in age (48 ± 13 years), sex (49% men), and average 24-h BP (132/81 ± 15/10 mm Hg). Mean daytime activity level was 44 ± 15 U. During the daytime, systolic BP (r = 0.33), diastolic BP (r = 0.29), and heart rate (r = 0.42) correlated best with the average activity for the 15 min preceding each measurement (P < .001).

Variance was very high, with activity explaining from 0% to 62% of BP variability for different individuals. Men and the obese had a greater reactivity of systolic BP to activity; older patients and those on antihypertensive therapy had a lower reactivity of heart rate. Blood pressure level is significantly associated with physical activity, but the percentage of variance of BP explained by physical activity varies greatly between individuals. Correlation is strongest between BP and average activity integrated over the previous 15 min. Much of the variance in blood pressure remains unexplained. Am J Hypertens 2000;13:1067–1073 © 2000 American Journal of Hypertension, Ltd.

KEY WORDS: Blood pressure, ambulatory monitoring, actigraphy, blood pressure variability, physical activity.

It has been known since the earliest ambulatory studies that physical activity is an important determinant of blood pressure (BP) level.1,2 During ambulatory monitoring, higher blood pressures are recorded during physical exertion, and lower BP levels are seen during relaxation and during sleep.3–5 However, less is known of the contribution of physical activity to the variability of BP over the monitoring period. Studies aimed at exploring this question have traditionally used patient diaries to differentiate activity levels during ambulatory BP monitoring (ABPM), or measured BP response to standardized laboratory-based exercise tests.2,6,7 Four recent studies8–13 in healthy volunteers and two in hypertensive patients have used actigraphy as an objective means of assessing physical activity levels during ABPM. The results of these studies have been contradictory, suggesting that from 0% to 74% of the variability in BP during ambulatory monitoring may be explained by physical activity.

The previous studies using objective monitoring of physical activity levels have been limited in size and in the spectrum of BP and activity levels. The primary objective of this study was to determine the extent to
which variation in physical activity is responsible for the variability of BP in a large, heterogeneous group of patients. In addition, we aimed to examine the effect of possible confounding variables such as age, gender, body mass index (BMI), smoking status, and the use of antihypertensive medications on this relationship.

**METHODS**

We simultaneously monitored ambulatory BP and physical activity in 431 patients referred to our clinic for evaluation of hypertension. Patients underwent 24-h ambulatory BP monitoring with the Quiet-Trak automated recorder (Tyco-Welch-Allyn Inc., Arden, NC), according to British Hypertension Society guidelines.14 The Quiet-Trak has satisfied the accuracy standards proposed by the Association for the Advancement of Medical Instrumentation (AAMI) and British Hypertension Society (BHS) guidelines for the assessment of ambulatory BP monitors.14–17 Blood pressure and heart rate were measured every 20 min during the monitoring period; all BP readings rejected by the Quiet-Trak software as being inaccurate or having failed were excluded from the analyses. In addition, each patient was fitted with an electronic actigraph (Gaehwiler Electronics, Hombrechtikon, Switzerland). This device, which is slightly larger than a wristwatch, contains a monaxial piezoelectric accelerometer with a threshold of 0.1 G, and is designed to integrate motor activity over a defined time period and convert it to an activity score on an arbitrary scale ranging from 0 to 253 U. The actigraph was programmed to record an activity score every 10 sec, and was mounted on the dominant wrist, as previous work has shown that motor activity at this site correlates best with physical activity level.9 All patients were given a standardized activity diary to complete during monitoring in which they noted time of going to bed at night and time of waking in the morning.

One of the authors (ACL) examined each of the activity data files individually. Time of going to sleep and time of waking were determined for each patient by comparing activity scores and diary records. For each patient, the point of falling asleep was considered to be the time closest to diary sleeptime where there was onset of a series of at least 60 activity scores (\(\approx\) 10 min), of which at least 90% were zero. Waking time was defined as the time closest to diary wake time at which there was onset of regular activity scores of more than zero. These definitions compare closely to those used in other studies.12,18,19 Quiet-Trak and actigraph data files were downloaded into an IBM-compatible PC, formatted as text files, and then combined into a single ASCII data set. This dataset was then loaded into the statistical analysis system (SAS) software package, version 6.12 (SAS Institute Inc., Cary, NC). All subsequent statistical analyses were carried out using SAS. Summaries of patient demographics, medication details, mean sleep and wake times, mean daytime and nighttime activity levels, and mean clinic and ambulatory BP values were determined using the Microsoft Excel spreadsheet software package.

For each patient, BP and heart rate were regressed on physical activity. The summary activity measures used in the regression were mean activity scores in the 5, 10, 15, and 20 min before each BP measurement. Mean activity data were log transformed as the distributions of mean activity were positively skewed. Systolic BP, diastolic BP, and heart rate were analyzed as outcome variables in the linear regressions. Daytime and nighttime values were analyzed separately, as it was expected that the low levels of both activity and BP generally seen during sleep would spuriously elevate the correlation coefficients. The correlation coefficient \((r)\) between activity and BP was calculated, and the variability of each outcome variable explained by the regression on activity was represented by the R\(^2\) value expressed as a percentage.

To determine the influence of various patient characteristics on the relationship between BP and activity, the analyses were repeated for different subsets of the total sample in multiple linear regressions. Patient variables added to the regression included gender, age, BMI, smoking status, and whether the patient was receiving antihypertensive medication. Mean 24-h BP and mean nighttime BP were added to the regression as measures of underlying BP level. As before, systolic BP, diastolic BP, and heart rate were the continuous outcomes under consideration. For the latter patient variables under consideration, the initial regression on physical activity was adjusted for age and gender. These analyses were carried out using repeated measures models. The correlation between BP and physical activity was modeled using an autoregressive covariance matrix, an appropriate model for time series measurements. This model takes into account the correlated nature of the variables considered, and allows for both inter- and intra-individual variability. A test of interaction between each patient variable and physical activity was entered into the model to assess whether the reactivity of systolic BP, diastolic BP, or heart rate changed with different levels of the variable under consideration. Continuous variables were tested as such in the interaction and were split into categories to illustrate any differences found. The results were expressed as the mean regression coefficient \((\beta)\). The size of the regression coefficient is a measure of how reactive BP was to activity in the period of 5 to 20 min before a BP reading was taken. All analyses were carried out using the procedure PROC MIXED of SAS (version 6.12), which fits mixed models for continuous outcomes.
TABLE 1. CHARACTERISTICS (MEAN ± SD) OF STUDY POPULATION (n = 431)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>48 ± 13 (15–81)</td>
</tr>
<tr>
<td>Gender (n)</td>
<td>213 males (49%)</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>26.7 ± 4</td>
</tr>
<tr>
<td>Smokers (n)</td>
<td>61</td>
</tr>
<tr>
<td>Antihypertensive medication (n)</td>
<td>202 on treatment (47%)</td>
</tr>
<tr>
<td>Daytime activity (U)</td>
<td>44 ± 15</td>
</tr>
<tr>
<td>Nighttime activity (U)</td>
<td>2 ± 1.4</td>
</tr>
<tr>
<td>Clinic BP (mm Hg)</td>
<td>144/89 ± 19/11</td>
</tr>
<tr>
<td>Daytime BP (mm Hg)</td>
<td>140/86 ± 15/11</td>
</tr>
<tr>
<td>Nighttime BP (mm Hg)</td>
<td>118/71 ± 17/11</td>
</tr>
</tbody>
</table>

BMI = body mass index; BP = blood pressure; SD = standard deviation.

RESULTS

Patient demographics, mean BP, and activity levels are detailed in Table 1. Just over half (52%) of this heterogeneous patient group was referred to our clinic to confirm or refute a diagnosis of hypertension, having had at least one elevated BP reading in a doctor’s office. Of these patients, 159 were hypertensive on ABPM (mean daytime systolic BP ≥135 mm Hg or diastolic BP ≥85 mm Hg). A further 47% had previously been diagnosed as hypertensive, and had been referred to determine BP control on therapy; only 54 of these were controlled (mean daytime BP <135/85 mm Hg). Patients had, on average, 60 valid BP readings of a possible maximum of 72 readings over the monitoring period (83%). Mean 24-h BP was 132/81 ± 15/10 mm Hg. On average, clinic BP was 4/2 mm Hg higher than mean daytime ambulatory BP. Variability between individuals was high. Mean daytime activity levels varied widely between patients, with 95% confidence intervals of 14 to 74 U. Mean nighttime activity was generally very low, with some patients scoring 0 U for several hours at a time. By actigraphy, average time to sleep was 11:42 PM, and average time of waking was 8:08 AM, giving a mean sleep duration of 8 h 26 min. Variation between patients was high, with 95% confidence intervals for sleep duration ranging from 5 h 45 min to 11 h 5 min. These times were similar to diary entries; patients recorded earlier sleep and waking times by an average of 7 and 25 min, respectively.

Blood pressure and heart rate were significantly correlated to mean physical activity in the 5 to 20 min preceding BP measurement (P < .001). The percentage variance (R²) in daytime systolic BP explained by mean (log transformed) activity before BP measurement was low, ranging from 8.8% to 10.7% (r = 0.30 to 0.33). The highest percentage variation of daytime systolic BP was explained by the mean activity for the 15 min before each BP measurement (Table 2). The range of values of R² was wide, varying from 0% to 62% for different patients. Therefore, some individuals had up to 62% of the variability of their BP explained by physical activity, whereas for others, variation in activity appeared to have little or no effect on BP. The mean percentage variance of daytime diastolic BP explained by activity tended to be lower than for systolic BP (Table 2). The greatest mean R² was 8.7%, seen for the mean activity in the 20 min preceding BP measurement. Once again, the range of individual values for R² was extremely wide (0% to 60%). A higher correlation was seen between daytime heart rate and activity (r = 0.42), with mean preceding activity explaining 14.1% to 17.8% of variation in heart rate. As expected, the percentage variation of BP explained by activity was higher for nighttime, with a maximum R² of 18.3% (r = 0.43) for the mean activity score for the 20 min preceding systolic BP measurement.

For all activity measures, the regression coefficients between activity and systolic BP were significantly larger for men (β = 3.1 to 4.1) than for women (β = 2.1 to 3.0), indicating that the unit increase in systolic BP for a unit increase in (log transformed) activity was greater for men (Figure 1). A similar trend was seen for diastolic BP, but these gender differences failed to reach statistical significance. When heart rate was considered as outcome, there were no differences in regression coefficients between men and women. In general there was no clear pattern in the reactivity of systolic BP and diastolic BP to activity in relation to age. When the sample was split into the two categories...
of those less than or equal to 50 years and those older than 50 years, there were no differences in regression coefficients for systolic and diastolic BP. However, for those aged 50 years and less, the reactivity of heart rate to activity before each measurement was greater than for those aged more than 50 years. These differences in regression coefficients were significant for the mean activity in the 10, 15, and 20 min before measurement ($P = .02, .06, \text{ and } .01, \text{ respectively}$).

Regression coefficients for the reactivity of heart rate to activity were significantly higher for patients on no antihypertensive medications compared to those taking antihypertensives (Figure 1). There were no differences between regression coefficients for systolic and diastolic BP between those who smoked and those who were not current smokers. It should be noted that the number of current smokers in this study was relatively small. There was no indication of any difference in reactivity of ambulatory systolic BP to activity for different levels of mean 24-h systolic BP. Similarly, there was no relationship between the regression coefficients and nighttime systolic BP.

**FIG. 1.** Mean increase in (a) systolic blood pressure and (b) heart rate ($\pm SE$) per unit increase in (log transformed) physical activity in the 15 min preceding BP measurement for different patient groups. SE = standard error; BP = blood pressure; Meds = on antihypertensive medication; No meds = on no antihypertensive medication.

There were several secondary findings in this heterogeneous patient population. First, correlation coefficients were consistently higher for nocturnal measurements, probably because of generally low levels of both physical activity and BP, with low variability of both variables. Second, men had higher regression coefficients for the regression of systolic BP on physical activity than women, indicating a higher unit increase in systolic BP per unit increase in activity for men. Third, reactivity of heart rate to activity was greater for those patients aged less than 50 years compared to those more than 50 years, and for those patients on no antihypertensive medication compared to those taking antihypertensives. Subgroup analysis of those taking antihypertensive medications revealed that the lowest regression coefficients were seen for patients taking $\beta$-blocking drugs. As 57 of the 190 patients more than 50 years (30%) were on $\beta$-blockers, compared to 35 of 241 patients (15%) in the younger group, the lower reactivity of heart rate to activity

**DISCUSSION**

We have simultaneously monitored 24-h ambulatory BP and physical activity in a large group of patients referred to a hospital hypertension clinic. The principal finding of this study was that there was a significant correlation between mean physical activity in the 5 to 20 min before ambulatory measurement and systolic BP, diastolic BP, and heart rate. Mean correlation between physical activity and hemodynamic variables ranged from 0.27 for diastolic BP to 0.42 for heart rate. The percentage variance in BP explained by physical activity was low, however, with a mean value ranging from 7.1% to 10.7%. Physical activity was responsible for more of the variability in heart rate, with mean values ranging from 14.1% to 17.8%. Interindividual variability was extremely high. Physical activity explained up to 62% of the variability in systolic BP for some patients and 0% of the variability for others. Similar interindividual variability was found for diastolic BP and heart rate.

Obese patients (BMI $\geqslant 25$ kg/m$^2$) had consistently higher regression coefficients as a measure of reactivity of systolic BP to activity compared to normal patients (BMI $\leqslant 25$ kg/m$^2$). These differences reached statistical significance at the 10% level for mean activity in the 5 min before BP measurement ($P = .07$).

There were no differences in the regression coefficients for systolic and diastolic BP or heart rate between those who smoked and those who were not current smokers. It should be noted that the number of current smokers in this study was relatively small. There was no indication of any difference in reactivity of ambulatory systolic BP to activity for different levels of mean 24-h systolic BP. Similarly, there was no relationship between the regression coefficients and nighttime systolic BP.

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seen in those more than 50 years may have been confounded by medication use. Fourth, our data show that obese patients had greater reactivity of systolic BP to activity than did patients with a normal BMI. Last, smoking status and underlying BP level did not appear to influence the regression of hemodynamic variables on physical activity.

Four recent studies in normal volunteers used similar methodology. Gretler et al, in a study performed in 10 healthy subjects, found mean correlation coefficients of 0.61, 0.59, and 0.64 between physical activity and systolic BP, diastolic BP, and heart rate, respectively. Activity was adjudged to be responsible for 18% to 69% of systolic BP variation. Van Egeren reported a similar study in 82 subjects, whereas Kohno et al monitored BP and physical activity in 17 college students. Both studies found average correlation coefficients between physical activity and BP of just over 0.5. In a finding similar to our own, Van Egeren reported that heavier subjects had greater regression coefficients for diastolic BP per unit increase in mean activity than did thin subjects, but this difference did not reach statistical significance. In contrast to the above studies, Shapiro and Goldstein showed no evidence of significant covariation between mean and variability of physical activity and mean and variability of BP during either waking or sleeping in 119 healthy elderly volunteers.

Stewart et al used actigraphy and ABPM to examine the relationship between BP and activity in a sample of 30 hypertensive patients. In this study, the average correlation between physical activity and BP was 0.25 for systolic BP and 0.34 for diastolic BP, values that are very similar to our findings. As in our study, the strength of the relationship varied widely between individual patients. On average, physical activity was judged to be responsible for 20% and 26% of the variability of systolic and diastolic BP, respectively. In a recently published study, Kario et al used methodology similar to our own, and found a weak but significant correlation between awake systolic BP variability and mean awake activity (r = 0.16, P < .05) in 160 normotensive and mildly hypertensive adults.

The studies of Gretler et al and Kohno et al used small numbers of healthy subjects with a mean age considerably younger than that in our study. Although Van Egeren used larger numbers and older subjects (20 to 64 years), these were healthy volunteers. Furthermore, in all these studies these researchers included nighttime data in the analyses, thus possibly spuriously elevating the correlation coefficients. It is thus difficult to draw conclusions from these studies about the relationship between BP and activity in middle-aged, hypertensive patients. The lack of a significant relationship between BP and activity seen in the study of Shapiro and Goldstein may be due to the age of the subjects involved (55 to 79 years), which may have limited the amount of activity they engaged in. Also, subjects were instructed not to undergo strenuous activity during the monitoring period. Although Stewart et al specifically examined hypertensive patients, the number of subjects was small. Kario et al report the largest series apart from our own, but their subjects had mean 24-h BP levels considerably lower than that for our patients; subjects with elevated clinic BP levels had been excluded at initial recruitment.

The principle strength of our series is its large size and hence power to detect associations. Second, the heterogeneous nature of the population studied has allowed us to examine the influence of different confounding variables on the relationship between activity and BP. Third, ambulatory actigraphy by piezoelectric accelerometry is becoming increasingly recognized as the method of choice for objective measurement of physical activity levels in clinical research. This easy to use, validated tool has been shown in previous studies to be a reliable and reproducible means of monitoring activity. Actigraphy correlates well with diary reports of activity and with diary reports of times of going to sleep and of rising. At least two studies have shown actigraphy to perform almost as well as electroencephalograms for determination of sleep duration. In our study, actigraphy scores correlated well with diary records of sleep and waking times. Patients diarized earlier waking times (25 min), perhaps because they awoke some minutes before resuming activity. Actigraphy has also been used to show changes in daily activity levels in response to successful therapy for depression.

The comparatively low mean variability of hemodynamic variables explained by physical activity seen in our study may be due, in part, to various methodologic weaknesses. The heterogeneity of this group of patients means that a variety of confounders, both known and unknown, may have served to either increase or decrease the correlation between activity and BP. Furthermore, previous studies suggest significant differences in correlations for an individual for repeat monitorings, whereas in this cross-sectional study each individual was monitored only once. Diary entries suggest that the mean daytime activity score of 44 ± 15 arbitrary activity units seen in this group of patients indicated generally low levels of activity. Physical activity may influence BP chiefly when large increases in activity are observed, such as the difference between complete relaxation and walking. If so, generally low levels of activity during ABPM may have led to a spuriously low correlation between the two variables. On the other hand, it may be argued...
that generally low levels of physical activity may spuriously elevate the correlation between activity and BP. Some evidence for this was seen in our study, with nighttime correlation coefficients being higher than those for the daytime.

In spite of its strengths, the actigraph remains at best a crude tool for measuring everyday physical activity. Actigraphy is unable to discriminate the nature and intensity of physical activity. The device has been shown to record high scores during driving and eating, pursuits that are not generally considered to be physically taxing.\textsuperscript{18} Wrist movement during writing, typing, or eating may produce spuriously high activity scores. On the other hand, scores may be low during activities that use substantial isometric exertion, a practice known to elevate BP and heart rate.\textsuperscript{6,11} In addition, we cannot rule out the possibility that the actigraph was removed during certain activities such as ablutions or sexual intercourse, thus affecting mean activity scores. Actigraphy is unable to provide information on posture, emotional state, social setting, or neurohormonal rhythms, all of which are known to influence BP. We had little information on the presence or absence of such stressors, and no hormonal data. Last, the low correlations seen in this study may have been due in part to the intermittent nature of BP recording seen with ABPM, and the need for the patient to keep still during BP measurement. Physical activity scores were averaged over a period of 5 to 20 min leading up to a BP measurement, whereas each BP measurement represents pressure over a period of a few seconds, once every 20 min.

In conclusion, we have examined the influence of physical activity on BP variability during ABPM, and have assessed the influence of some of the confounders in this relationship. Our findings, which compare to previous work, suggest that although there is a significant association between physical activity and BP variability, this relationship is weak. The average correlation between BP and activity was approximately 0.3, with high interindividual variability, so that activity explained up to 62% of the variability of BP in some individuals, and none of the variability in BP for others. For these patients, other factors including mental stress, emotional state, posture, isometric muscular contraction, and neurohormonal activity may be responsible for much of the variability of BP. Overall, it appears that the majority of the variance in BP during both daytime and nighttime remains to be explained.

REFERENCES


