Effect of environmental exposure to cigarette smoke on blood pressure in 24-hour ambulatory blood pressure monitoring in patients with essential hypertension

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KEY WORDS
blood pressure, hypertension, passive smoking

ABSTRACT

INTRODUCTION A relationship between environmental exposure to cigarette smoke and blood pressure has not been well-established.

OBJECTIVES The aim of the study was to evaluate the effects of environmental exposure to cigarette smoke on blood pressure (BP) in patients with essential hypertension.

PATIENTS AND METHODS The study involved 39 nonsmoking patients with essential hypertension treated with hypotensive agents and environmentally exposed to cigarette smoke (group 1) and 39 nonsmoking patients with essential hypertension treated with hypotensive agents and not exposed to cigarette smoke (group 2). The following variables of 24-hour ambulatory BP monitoring (ABPM) were measured: systolic BP (SBP), diastolic BP (DBP), mean arterial pressure (MAP), and pulse pressure (PP) during 24-hour ABPM, and, separately, for the period of daily activity and night rest.

RESULTS In group 1, the mean values of 24-hour SBP, DBP, MAP, and PP, daytime SBP, DBP, MAP, and PP, and nighttime SBP, MAP, and PP were significantly higher than those in group 2. Statistically significant positive linear correlations were demonstrated between the mean time of daily exposure (expressed in hours) to cigarette smoke and 24-hour MAP and PP ($r = 0.52$ and $r = 0.48$, respectively, $P < 0.05$).

Advanced age, higher low-density lipoprotein cholesterol and glucose concentrations, and environmental exposure to cigarette smoke were independent factors of elevated 24-hour PP in the study group.

CONCLUSIONS In patients with essential hypertension, environmental exposure to cigarette smoke may result in elevated BP values in 24-hour ABPM.
The aim of the study was to evaluate the effects of environmental exposure to cigarette smoke on BP in 24-hour ABPM in patients with essential hypertension.

**Patients and Methods** The study involved 78 individuals. Group 1 consisted of 39 consecutive patients with essential hypertension from our outpatient cardiac clinic. They were nonsmokers with environmental exposure to smoke. The inclusion criteria were as follows: essential hypertension, diagnosed at least 5 years earlier and treated pharmacologically, nonsmoking history, and passive exposure to cigarette smoke determined on the basis of a questionnaire. Subsequently, using the case-to-case approach, every patient from group 1 was matched with a patient with essential hypertension who has never smoked and has never been exposed to environmental tobacco smoke. The inclusion criteria were as follows: essential hypertension diagnosed at least 5 years earlier and treated pharmacologically, the same grade of arterial hypertension according to the European Society of Cardiology / European Society of Hypertension, a similar duration of essential hypertension (a difference of no more than 2 years), similar protocol of essential hypertension treatment (hypotensive agents from the same drug group), the same sex, similar age (a difference of no more than 2 years), similar body mass index (a difference of no more than 0.5 kg/m²), similar total cholesterol concentration (a difference of no more than 20 mg/dl), similar triglyceride concentration (a difference of no more than 20 mg/dl), similar blood glucose concentration (a difference of no more than 20 mg/dl), similar blood glucose concentration (a difference of no more than 20 mg/dl), similar body mass index (a difference of no more than 0.5 kg/m²), similar height (a difference of no more than 2 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for at least 30 minutes a day next to persons who smoke cigarettes; and living with 1 or more smokers in a household.

In all participants, 24-hour ABPM was conducted using the Welch Allyn ABPM 6100 apparatus (Welch Allyn, Aston Abbotts, Buckinghamshire, United Kingdom). A proper cuff was selected according to the size of the patient’s arm and placed on the nondominant arm. Patients were asked to keep their arms still at the time of measurements. The estimated parameters included systolic BP (SBP), diastolic BP (DBP), mean arterial pressure (MAP), and PP during 24-hour monitoring, and, separately, for day and night monitoring. PP was calculated using the following formula: \( PP = SBP - DBP \).

Daytime monitoring was from 6:00 AM to 10:00 PM, measured automatically every 30 minutes, and during nighttime, from 10:00 PM to 6:00 AM, BP was measured once an hour. Moreover, variability in SBP and DBP was assessed. The variability in SBP was defined as the standard deviation (SD) of all measurements of SBP in 24 hours and variability in DBP, as the SD of all measurements of DBP in 24 hours. ABPM data series were considered invalid for the analysis if 30% or more of the scheduled measurements were unavailable, if data were lacking for more than 2 consecutive hourly intervals, if data were obtained while patients maintained an irregular rest-activity schedule during the 2 consecutive 24-hour periods of monitoring, or if the night sleep was shorter than 6 hours or longer than 12 hours.

A statistical analysis was conducted using the STATISTICA 10 software (StatSoft Poland). For quantitative variables, arithmetical means and SDs of the estimated parameters were calculated in the study groups. The distribution of the variables was examined using the Lilliefors and Shapiro–Wilk tests. For independent quantitative variables with normal distribution, a subsequent statistical analysis involved the \( t \) test for independent variables. In the case of variables with nonnormal distribution, the Mann–Whitney test was applied for independent quantitative variables. The results for qualitative (nominal) variables were expressed as percentages. In the case of independent qualitative variables, a subsequent statistical analysis involved the \( \chi^2 \) test of the highest likelihood. To define relationships between the studied variables, the analyses of correlation and regression were conducted. A \( P \) level of less than 0.05 was considered statistically significant.

The study was conducted according to the principles of Good Clinical Practice and the Declaration of Helsinki and was approved by the local bioethics committee.

**RESULTS** Compared with group 2, group 1 had significantly higher mean values of 24-hour SBP, DBP, MAP, and PP; daytime SBP, DBP, MAP, PP; and nighttime SBP, MAP, and PP (Table 3).

Within group 1, subgroup A had significantly higher mean values of 24-hour SBP, MAP, and PP, as well as daytime SBP and PP compared with subgroup B (Table 4).

We observed positive linear correlations between the mean time of daily exposure (expressed in hours) to cigarette smoke and 24-hour MAP and PP in patients with essential hypertension.

### TABLE 3 Parameters of ambulatory blood pressure monitoring in the study groups

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (n = 39)</th>
<th>Group 2 (n = 39)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SBP, mmHg</strong></td>
<td>136.6 ± 17.4</td>
<td>126.1 ± 16.3</td>
</tr>
<tr>
<td><strong>DBP, mmHg</strong></td>
<td>84.9 ± 9.2</td>
<td>78.1 ± 8.3</td>
</tr>
<tr>
<td><strong>MAP, mmHg</strong></td>
<td>99.6 ± 10.2</td>
<td>91.0 ± 8.5</td>
</tr>
<tr>
<td><strong>PP, mmHg</strong></td>
<td>56.9 ± 7.1</td>
<td>48.9 ± 7.3</td>
</tr>
<tr>
<td>Variability in SBP, mmHg</td>
<td>15.5 ± 5.1</td>
<td>13.4 ± 4.8</td>
</tr>
<tr>
<td>Variability in DBP, mmHg</td>
<td>10.4 ± 3.4</td>
<td>10.4 ± 2.2</td>
</tr>
</tbody>
</table>

Note: \( a \) \( P < 0.05 \)

Abbreviations: DBP – diastolic blood pressure, MAP – mean arterial pressure, PP – pulse pressure, SBP – systolic blood pressure

### TABLE 4 Parameters of ambulatory blood pressure monitoring in the study subgroups

<table>
<thead>
<tr>
<th></th>
<th>Subgroup A (n = 20)</th>
<th>Subgroup B (n = 19)</th>
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</thead>
<tbody>
<tr>
<td><strong>SBP, mmHg</strong></td>
<td>144.8 ± 16.0</td>
<td>133.1 ± 17.6</td>
</tr>
<tr>
<td><strong>DBP, mmHg</strong></td>
<td>87.3 ± 8.6</td>
<td>85.0 ± 9.6</td>
</tr>
<tr>
<td><strong>MAP, mmHg</strong></td>
<td>102.8 ± 10.1</td>
<td>96.1 ± 9.5</td>
</tr>
<tr>
<td><strong>PP, mmHg</strong></td>
<td>61.0 ± 6.4</td>
<td>52.6 ± 7.5</td>
</tr>
<tr>
<td>Variability in SBP, mmHg</td>
<td>14.3 ± 6.4</td>
<td>15.7 ± 5.7</td>
</tr>
<tr>
<td>Variability in DBP, mmHg</td>
<td>10.0 ± 3.4</td>
<td>9.7 ± 3.1</td>
</tr>
<tr>
<td><strong>dipper, %</strong></td>
<td>60.0</td>
<td>73.7</td>
</tr>
</tbody>
</table>

Note: \( a \) \( P < 0.05 \)

Abbreviations: see Table 3
The obtained model demonstrated that in the group of patients with diagnosed essential hypertension who received pharmacological treatment and were at a more advanced age, higher LDL cholesterol and glucose concentrations as well as environmental exposure to cigarette smoke were independent factors of elevated 24-hour PP.

**DISCUSSION**

In our study, BP was evaluated using 24-hour ABPM. Apart from providing a reliable and reproducible evaluation of BP, ABPM allowed to estimate additional parameters including BP variability and PP. In recent years, BP variability has been recognized in multicenter studies as a prognostically significant cardiovascular complication of hypertension. In the Ohasama Study, an increase in the value of SD of BP measurements above 18 mmHg was found to be associated with an increased risk of cardiovascular complications. Sander et al. demonstrated that daily variability of SBP exceeding 15 mmHg represented a prognostic factor pointing to an early progression of arteriosclerotic lesions in the carotid arteries. Increased values of PP also represent an independent risk factor of cardiovascular complications. The Hypertension Detection and Follow-up Program study demonstrated that PP represents a risk factor of cardiac and overall mortality but not of cerebrovascular mortality.

The effect of active cigarette smoking on BP values and on manifestation of arterial hypertension has been widely discussed in recent studies. However, a positive relationship between active smoking and increased values of BP continues to be disputable and studies conducted so far provided equivocal or even contradictory results. Bolinder and de Faire, Groppelli et al., Minami et al., Pannarale et al., and Verdecchia et al. demonstrated that the mean values of BP in ABPM were significantly higher in active smokers compared with nonsmokers. Moreover, according to Pannarale et al., active smokers had an increased variability of BP in ABPM. Ekstrand et al. observed more frequent manifestations of arterial hypertension among active smokers than in the general population. However, studies are also available that failed to confirm the relationship between cigarette smoking and arterial hypertension. In a study by Yano et al., active smokers and nonsmokers had similar values of BP both in office measurements and ABPM. A study by Okubo et al. showed no effects of cigarette smoking on the rate of arterial hypertension. A number of studies showed even significantly lower mean values of BP in smokers compared with nonsmokers; in such studies, BP was evaluated using office measurements and/or ABPM.

The relationship between environmental exposure to cigarette smoke and BP has been much less clearly recognized. On the one hand, a positive relationship between environmental exposure to tobacco smoke and arterial BP values in domestic measurements in healthy women was confirmed in an epidemiological study by

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**FIGURE 1** Correlation between daily exposure to cigarette smoke and 24-hour mean arterial pressure in group 1 (r = 0.52, P < 0.05)

**FIGURE 2** Correlation between daily exposure to cigarette smoke and 24-hour pulse pressure in group 1 (r = 0.48, P < 0.05)

*Panel 10* environmentally exposed to cigarette smoke (correlation coefficients, 0.52 and 0.48, respectively, P < 0.05) (FIGURES 1 and 2).

A multivariable stepwise progressive regression analysis in the whole study group, including basic anthropological parameters (age, sex, body mass index), parameters of essential hypertension (duration of hypertension, grades of essential hypertension, hypotensive treatment), risk factors for cardiovascular diseases (total cholesterol, low-density lipoprotein [LDL] cholesterol, triglyceride, and glucose levels) and environmental exposure to cigarette smoke (dichotomous variable: 1 – yes, 0 – no), resulted in the following model: 24-hour PP = 7.61 + 0.75 age + 0.01 LDL cholesterol + 0.01 glucose + 2.99 environmental exposure to cigarette smoke ±4.21 (TABLE 5).
Environmental exposure to cigarette smoke, dichotomous variable where 1: yes and 0: no. Conversion factors to SI units are as follows: for LDL cholesterol, 0.0259; for glucose, 0.0555.

Abbreviations: 24-hour PP – pulse pressure during 24-hour monitoring, LDL – low-density lipoprotein, SEM of Rc – standard error of the mean of regression coefficient

REFERENCES

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ARTYKUŁ ORYGINALNY

Wpływ środowiskowego narażenia na dym papierosowy na wartości ciśnienia krwi oceniane metodą 24-godzinnego ambulatoryjnego monitorowania u chorych z pierwotnym nadciśnieniem tętniczym

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SŁOWA KLUCZOWE
bierne palenie, ciśnienie krwi, nadciśnienie tętnicze

STRESZCZENIE

WProwadzenie Zależność między środowiskowym narażeniem na dym tytoniowy a ciśnieniem krwi jest niewystarczająco poznana.

CELE Celem pracy była ocena wpływu środowiskowego narażenia na dym papierosowy na wartości ciśnienia krwi u chorych z pierwotnym nadciśnieniem tętniczym.

PACJENCI I METODY Do badania włączone 39 pacjentów chorujących na nadciśnienie tętnicze leczonych preparatami hipotensyjnymi, niepalących papierosów, środowiskowo narażonych na dym papierosowy (grupa 1) oraz 39 pacjentów chorujących na nadciśnienie tętnicze leczonych preparatami hipotensyjnymi, niepalących papierosów, nienarażonych na dym papierosowy (grupa 2). Określono następujące parametry 24-godzinnego ambulatoryjnego monitorowania ciśnienia krwi (ambulatory blood pressure monitoring – ABPM): skurczowe ciśnienie krwi (systolic blood pressure – SBP), rozkurczowe ciśnienie krwi (diastolic blood pressure – DBP), średnie ciśnienie tętnicze (mean arterial pressure – MAP) i ciśnienie tętna (pulse pressure – PP) w obserwacji 24-godzinnej oraz osobno dla godzin dziennej aktywności i godzin odpoczynku nocnego.

WYNIKI W grupie 1 średnie wartości 24-godzinnego SBP, DBP, MAP i PP, dziennego SBP, DBP, MAP i PP oraz nocnego SBP, MAP i PP były znamienie wyższe w porównaniu do grupy 2. Wykazano istotne dodatnie zależności liniowe między dzienną liczbą godzin narażenia na dym tytoniowy a 24-godzinnym MAP i PP (odpowiednio r = 0,52 i 0,48; p <0,05). Starszy wiek, wyższe stężenie cholesterolu lipoprotein o małej gęstości i glukozy oraz środowiskowe narażenie na dym papierosowy stanowią niezależne czynniki ryzyka zwiększenia 24-godzinnego PP w badanej grupie osób.

WNIOSKI U chorych z nadciśnieniem tętniczym środowiskowe narażenie na dym papierosowy może skutkować wyższymi wartościami ciśnienia krwi w 24-godzinnym pomiarze metodą ABPM.