

# 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.

**INTRODUCTION** Active cigarette smoking is known to increase the risk of cardiovascular diseases.<sup>1,2</sup> Numerous multicenter cohort and randomized clinical studies have shown that active cigarette smoking represents one of the main risk factors for the development of cardiovascular diseases, particularly, atherosclerosis and its complications.<sup>3-5</sup>

The problem of smoking cannot be reduced to active smoking only. Environmental exposure to cigarette smoke, known as passive smoking, seems to be equally important.<sup>3,6</sup> The spread of passive smoking epidemic has been confirmed

by the results of the Third Health and Nutrition Examination Survey (NHANES III) conducted in the USA in the years 1988–1991. According to the survey, passive exposure to cigarette smoke, estimated on the basis of serum cotinine concentrations, may affect 87.9% of the American population.<sup>7</sup>

Effects of active cigarette smoking on blood pressure (BP) and essential hypertension has been broadly discussed. Higher BP in active smokers compared with nonsmoking individuals was reported. However, there are studies that did not confirm this association or even showed lower BP

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Received: May 30, 2014.

Revision accepted: July 21, 2014.

Published online: July 21, 2014.

Conflict of interest: none declared.

Pol Arch Med Wewn. 2014;

124 (9): 436-442

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**TABLE 1** Clinical characteristics of the study groups

	Group 1 (n = 39)	Group 2 (n = 39)
age, y	53.3 ± 9.4	54.2 ± 9.1
height, cm	164.1 ± 6.1	166.4 ± 7.3
body mass, kg	71.7 ± 7.9	72.1 ± 8.1
BMI, kg/m <sup>2</sup>	26.4 ± 2.7	26.1 ± 2.8
sex, %		
men	41.0	41.0
women	59.0	59.0
essential hypertension, %	100.0	100.0
diabetes, %	7.7	7.7
coronary artery disease, %	15.4	15.4
atherosclerosis, %	12.8	10.2
hypercholesterolemia, %	20.5	23.1
hypertriglyceridemia, %	25.7	23.1
environmental exposure to cigarette smoke, %	100.0 <sup>a</sup>	0.0
daily exposure to cigarette smoke, h	5.7 ± 2.2 <sup>a</sup>	0.0 ± 0.0

**a**  $P < 0.05$

Abbreviations: BMI – body mass index

**TABLE 2** Characteristics of hypertension and hypotensive treatment in the study groups

	Group 1 (n = 39)	Group 2 (n = 39)
essential hypertension, y	10.4 ± 5.2	9.9 ± 4.7
grades of essential hypertension, %		
mild	33.3	33.3
moderate	53.8	53.8
severe	12.8	12.8
hypotensive treatment, %		
monotherapy	28.2	28.2
combination therapy	71.8	71.8
hypotensive drugs, %		
diuretics	30.8	28.2
β-blockers	38.5	35.9
ACEIs	56.4	61.5
angiotensin receptor blockers	10.3	10.3
calcium channel blockers	33.3	30.8

Abbreviations: ACEIs – angiotensin-converting enzyme inhibitors

values in active smokers.<sup>8</sup> A relationship between environmental exposure to cigarette smoke and BP has not been well-established and requires further research.

BP measured by 24-hour ambulatory BP monitoring (ABPM) shows higher reproducibility than that obtained during individual outpatient visits or by patients themselves.<sup>9,10</sup> Moreover, 24-hour ABPM allows to define BP variability and pulse pressure (PP).<sup>11</sup> In multicenter studies, both abnormal variability in BP and abnormal PP have been recognized as prognostically unfavorable consequence of arterial hypertension.

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/m<sup>2</sup>), 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 10 mg/dl), nonsmoking history, and no passive exposure to cigarette smoke determined on the basis of the questionnaire. A total of 39 patients who fulfilled those criteria were included in group 2. The main clinical characteristics of both groups are presented in **TABLE 1**, while details on essential hypertension and hypotensive treatment, in **TABLE 2**.

Group 1 was divided into 2 subgroups using the threshold value of median daily exposure to cigarette smoke (expressed in hours; median, 5 h). Subgroup A consisted of patients suffering from arterial hypertension who were pharmacologically treated and environmentally exposed to cigarette smoke with the mean time of daily exposure of 5 hours or longer, and subgroup B, of patients suffering from arterial hypertension who were pharmacologically treated and environmentally exposed to cigarette smoke with the mean time of daily exposure of less than 5 hours.

All individuals included in the study completed the questionnaire regarding the current and past health status (with emphasis on the treatment of essential hypertension) as well as family history. There were also questions related to alimentary habits, current diet, physical activity, cigarette smoking, and alcohol and coffee drinking. Passive exposure to cigarette smoke was defined as smoking no cigarettes (now and in the past); being exposed to cigarette smoke in a closed space for at least an hour a day; staying

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

	Group 1 (n = 39)	Group 2 (n = 39)
24-hour monitoring (6:00–6:00)		
SBP, mmHg	136.6 ± 17.4 <sup>a</sup>	126.1 ± 16.3
DBP, mmHg	84.9 ± 9.2 <sup>a</sup>	78.1 ± 8.3
MAP, mmHg	99.6 ± 10.2 <sup>a</sup>	91.0 ± 8.5
PP, mmHg	56.9 ± 7.1 <sup>a</sup>	48.9 ± 7.3
variability in SBP, mmHg	15.5 ± 5.1	13.4 ± 4.8
variability in DBP, mmHg	10.4 ± 3.4	10.4 ± 2.2
dipper, %	66.7	74.3
day monitoring (6:00 AM – 10:00 PM)		
SBP, mmHg	139.1 ± 18.1 <sup>a</sup>	128.4 ± 17.0
DBP, mmHg	86.7 ± 10.3 <sup>a</sup>	80.4 ± 9.1
MAP, mmHg	99.8 ± 9.4 <sup>a</sup>	90.2 ± 8.1
PP, mmHg	57.4 ± 7.3 <sup>a</sup>	50.2 ± 7.6
night monitoring (10:00 PM – 6:00 AM)		
SBP, mmHg	115.3 ± 11.7 <sup>a</sup>	109.7 ± 10.9
DBP, mmHg	70.7 ± 7.6	67.9 ± 6.8
MAP, mmHg	84.3 ± 8.8 <sup>a</sup>	78.7 ± 7.9
PP, mmHg	51.1 ± 6.4 <sup>a</sup>	45.1 ± 6.9

**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

	Subgroup A (n = 20)	Subgroup B (n = 19)
24-hour monitoring (6:00–6:00)		
SBP, mmHg	144.8 ± 16.0 <sup>a</sup>	133.1 ± 17.6
DBP, mmHg	87.3 ± 8.6	85.0 ± 9.6
MAP, mmHg	102.8 ± 10.1 <sup>a</sup>	96.1 ± 9.5
PP, mmHg	61.0 ± 6.1 <sup>a</sup>	52.6 ± 7.5
variability in SBP, mmHg	14.9 ± 6.4	15.7 ± 5.7
variability in DBP, mmHg	10.0 ± 3.4	9.7 ± 3.1
dipper, %	60.0	73.7
day monitoring (6:00 AM – 10:00 PM)		
SBP, mmHg	147.1 ± 14.8 <sup>a</sup>	134.3 ± 17.6
DBP, mmHg	86.5 ± 13.4	87.3 ± 10.8
MAP, mmHg	103.4 ± 11.6	98.0 ± 9.0
PP, mmHg	60.3 ± 7.0 <sup>a</sup>	53.9 ± 6.5
night monitoring (10:00 PM – 6:00 AM)		
SBP, mmHg	116.1 ± 11.7	113.6 ± 11.1
DBP, mmHg	71.4 ± 7.7	69.9 ± 6.7
MAP, mmHg	86.8 ± 7.2	82.0 ± 9.4
PP, mmHg	52.1 ± 6.1	49.8 ± 5.4

**a**  $P < 0.05$

Abbreviations: see **TABLE 3**

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 Abbots, 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 BP 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.<sup>12</sup>

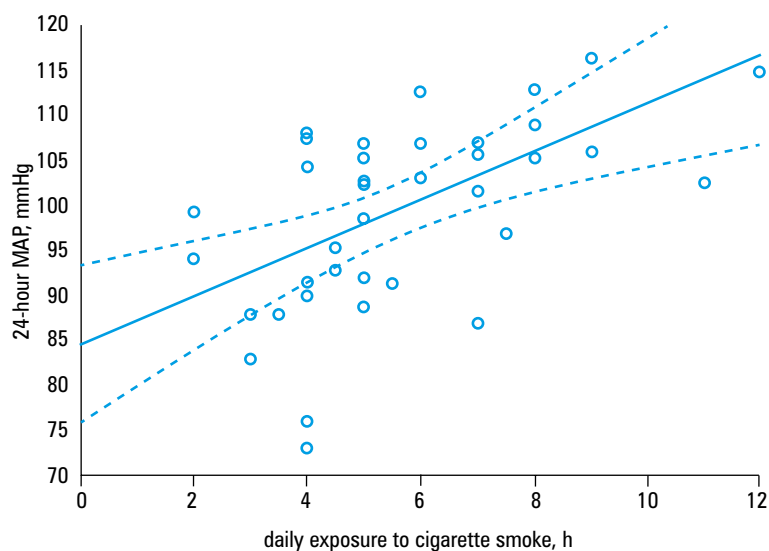
A statistical analysis was conducted using the STATISTICA 10 software (StatSoft Poland). For quantitative variables, arithmetical means and SDs of the estimated parameters were calculated in the study groups. The distribution of the variables was examined using the Lilliefors and Shapiro–Wilk tests. 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.

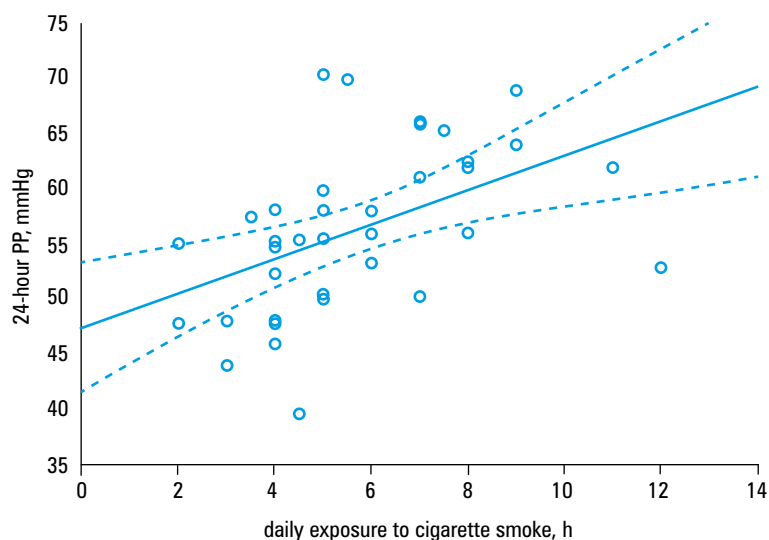
**RESULTS** Compared with group 2, group 1 had significantly higher mean values of 24-hour SBP, DBP, MAP, and PP; daytime SBP, DBP, MAP, PP; and nighttime SBP, MAP, and PP (**TABLE 3**).

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



**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 ( $r = 0.48$ ,  $P < 0.05$ )

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  $\pm$  4.21 (TABLE 5).

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.<sup>11</sup> In recent years, BP variability has been recognized in multicenter studies as a prognostically significant cardiovascular complication of hypertension.<sup>13</sup> In the Ohasama Study,<sup>14</sup> 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.<sup>15</sup> 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.<sup>13</sup> The Hypertension Detection and Follow-up Program study<sup>16</sup> 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,<sup>17</sup> Gropelli et al.,<sup>18</sup> Minami et al.,<sup>19</sup> Pannarale et al.,<sup>20</sup> and Verdecchia et al.<sup>21</sup> demonstrated that the mean values of BP in ABPM were significantly higher in active smokers compared with nonsmokers. Moreover, according to Pannarale et al.,<sup>20</sup> active smokers had an increased variability of BP in ABPM. Ekstrand et al.<sup>22</sup> 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.,<sup>23</sup> active smokers and nonsmokers had similar values of BP both in office measurements and ABPM. A study by Okubo et al.<sup>24</sup> 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.<sup>25-27</sup>

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

**TABLE 5** Estimation for the final model obtained in multivariate regression analysis

	Model for 24-hour PP, mmHg		
	regression coefficient	SEM of Rc	P value
intercept	7.612	1.369	0.001
age, y	0.749	0.022	0.001
LDL cholesterol, mg/dl	0.012	0.008	0.043
glucose, mg/dl	0.011	0.016	0.029
environmental exposure to cigarette smoke	2.991	0.461	0.001

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

Seki et al.<sup>28</sup>; on the other hand, studies in which arterial BP was estimated by ABPM did not unequivocally determine the type of relationships between environmental exposure to tobacco smoke and arterial BP. Leone et al.<sup>29</sup> showed that single acute exposure to tobacco smoke failed to significantly affect the values of arterial BP evaluated using ABPM.<sup>29</sup> Pijanowska and Zajączkowska<sup>30</sup> reported no differences in arterial BP values in ABPM studies in the group of healthy children exposed and not exposed to tobacco smoke. On the other hand, Makris et al.<sup>31</sup> documented a relationship between passive smoking and arterial BP values evaluated using ABPM. Studies conducted in healthy normotensive nonsmoking adults exposed to tobacco smoke manifested significantly higher values of arterial BP in 24-hour measurements and higher probability of being burdened with masked hypertension as compared with nonsmokers not exposed to tobacco smoke.<sup>31</sup> Considering the above data, it seemed interesting to study the relationship between environmental exposure to cigarette smoke and BP in patients with essential hypertension using 24-hour ABPM.

Our study confirmed a positive relationship between environmental exposure to cigarette smoke and BP values evaluated using 24-hour ABPM in patients with essential hypertension. Our results seem to indicate that the mean time of daily exposure to cigarette smoke (expressed in hours) may be a useful parameter characterizing the extent of environmental exposure to cigarette smoke. Moreover, our study allowed to answer the questions related to the relationship between the environmental exposure to cigarette smoke and complications of essential hypertension, namely, abnormal variability of BP and increased PP. The environmental exposure to cigarette smoke has been found to induce no alterations in the variability of BP. On the other hand, a relationship has been documented between environmental exposure to cigarette smoke and an increase in PP.

Our study has several limitations: small study groups from the epidemiological point of view,

lack of clinical homogeneity within the studied group, effects of hypotensive treatment on BP values, and assessing environmental exposure to cigarette smoke using a questionnaire. In our study, we used a case-to-case analysis. Such methodology was appropriate in that it allowed us to make comparisons in terms of the variables other than the variable that differentiated the groups. However, its main limitation was a small size of the study groups because of the time-consuming process of recruiting controls who met the inclusion criteria. However, it should be noted that the size of the group was sufficient to meet the quantitative criteria for all statistical analysis. The effects of hypotensive treatment on BP values could not be avoided, considering the risk of serious health complications linked to discontinuation of hypotensive drug treatment, including the risk of life-threatening conditions.

In summary, our results show that modern integrated educational systems, implemented for several years and aimed at fighting active smoking, should be supplemented with the activities aimed at preventing environmental exposure to cigarette smoke, which is equally harmful to health. In line with such expectations, in recent years, the World Health Organization created a pack of 6 antinicotine programs, called MPOWER.<sup>32</sup> The pack contains activities involving monitoring of spread manifested by exposure to cigarette smoke, counteracting exposure to cigarette smoke by smoking bans in public spaces, offering help in abandoning smoking habit, warning against the effects of smoking, enforcing prohibition of advertising, promotion and sponsoring of tobacco products, and regular increases in tobacco tax.<sup>33</sup>

**Acknowledgments** The study was funded by local grants from the Wrocław Medical University.

## REFERENCES

- 1 Szpak D, Grochowalski A, Chrzyszcz R, et al. Tobacco smoke exposure and endothelial dysfunction in patients with advanced coronary artery disease. *Pol Arch Med Wewn.* 2013; 123: 474-481.
- 2 Wilk G, Osmenda G, Matusik P, et al. Endothelial function assessment in atherosclerosis: comparison of brachial artery flow-mediated vasodilation and peripheral arterial tonometry. *Pol Arch Med Wewn.* 2013; 123: 443-452.
- 3 Flouris AD, Vardavasm CI, Metsios GS, et al. Biological evidence for the acute health effects of secondhand smoke exposure. *Am J Physiol Lung Cell Mol Physiol.* 2010; 298: L3-L12.
- 4 Ambrose JA, Barua RS. The pathophysiology of cigarette smoking and cardiovascular disease: an update. *J Am Coll Cardiol.* 2004; 43: 1731-1737.
- 5 Hausberg M, Kosch M, Barenbrock M. Smoking, blood pressure and cardiovascular risk. *Hypertension.* 2002; 20: 1699-1701.
- 6 Zawadzki M, Gać P, Poreba R, et al. Levels of hemoglobin and lipid peroxidation metabolites in blood, catalase activity in erythrocytes and peak expiratory flow rate in subjects with passive exposure to tobacco smoke. *Pol Arch Med Wewn.* 2008; 118: 705-712.
- 7 Pirkle JL, Flegal KM, Bernert JT, et al. Exposure of the US population to environmental tobacco smoke: the Third National Health and Nutrition Examination Survey, 1988 to 1991. *JAMA.* 1996; 275: 1233-1240.
- 8 Katsiki N, Papadopoulou SK, Fachantidou AI, et al. Smoking and vascular risk: are all forms of smoking harmful to all types of vascular disease? *Public Health.* 2013; 127: 435-441.
- 9 Grassi G, Bombelli M, Seravalle G, et al. Role of ambulatory blood pressure monitoring in resistant hypertension. *Curr Hypertens Rep.* 2013; 15: 232-237.

- 10 Mancia G, Parati G. The role of blood pressure variability in end-organ damage. *J Hypertens Suppl.* 2003; 21: S17-S23.
- 11 Czaja-Mitura I, Bortkiewicz A. Ambulatory blood pressure monitoring (ABPM) and its usefulness in occupational medicine. *Med Pr.* 2012; 63: 701-709.
- 12 Hermida RC, Smolensky MH, Ayala DE, et al. 2013 ambulatory blood pressure monitoring recommendations for the diagnosis of adult hypertension, assessment of cardiovascular and other hypertension-associated risk, and attainment of therapeutic goals. *Chronobiol Int.* 2013; 30: 355-410.
- 13 Conen D, Bamberg F. Noninvasive 24-h ambulatory blood pressure and cardiovascular disease: a systematic review and meta-analysis. *J Hypertens.* 2008; 26: 1290-1299.
- 14 Kikuya M, Hozawa A, Ohkubo T, et al. Prognostic significance of blood pressure and heart rate variabilities: the Ohasama study. *Hypertension.* 2000; 36: 901-906.
- 15 Sander D, Kukla C, Klingelhöfer J, et al. Relationship between circadian blood pressure patterns and progression of early carotid atherosclerosis: a 3-year follow-up study. *Circulation.* 2000; 102: 1536-1541.
- 16 Benetos A, Zureik M, Morcet J, et al. A decrease in diastolic blood pressure combined with an increase in systolic blood pressure is associated with a higher cardiovascular mortality in men. *J Am Coll Cardiol.* 2000; 35: 673-680.
- 17 Bolinder G, de Faire U. Ambulatory 24-h blood pressure monitoring in healthy, middle-aged smokeless tobacco users, smokers, and nontobacco users. *Am J Hypertens.* 1998; 11: 1153-1163.
- 18 Groppelli A, Giorgi DM, Omboni S, et al. Persistent blood pressure increase induced by heavy smoking. *J Hypertens.* 1992; 10: 495-499.
- 19 Minami J, Ishimitsu T, Matsuoka H. Effects of smoking cessation on blood pressure and heart rate variability in habitual smokers. *Hypertension.* 1999; 33: 586-590.
- 20 Pannarale G, Acconcia MC, Gianturco L, et al. Cigarette smoking and ambulatory blood pressure: a case-control study in normotensives. *J Hum Hypertens.* 2008; 22: 129-131.
- 21 Verdecchia P, Schillaci G, Borgioni C, et al. Cigarette smoking, ambulatory blood pressure and cardiac hypertrophy in essential hypertension. *J Hypertens.* 1995; 13: 1209-1215.
- 22 Ekstrand K, Nilsson JA, Lijja B, et al. Markers for development of hypertension in commercial flight aviators. *Aviat Space Environ Med.* 1991; 62: 963-968.
- 23 Yano Y, Hoshida S, Shimada K, et al. The impact of cigarette smoking on 24-hour blood pressure, inflammatory and hemostatic activity, and cardiovascular risk in Japanese hypertensive patients. *J Clin Hypertens (Greenwich).* 2013; 15: 234-240.
- 24 Okubo Y, Suwazono Y, Kobayashi E, et al. An association between smoking habits and blood pressure in normotensive Japanese men: a 5-year follow-up study. *Drug Alcohol Depend.* 2004; 73: 167-174.
- 25 Mikkelsen KL, Winberg N, Hoegholm A, et al. Smoking related to 24-h ambulatory blood pressure and heart rate: a study in 352 normotensive Danish subjects. *Am J Hypertens.* 1997; 10: 483-491.
- 26 Demers M, Vézina M, Bernard PM, et al. Relation between blood pressure and smoking in a population of Quebec workers. *Arch Mal Coeur Vaiss.* 1988; 81: 775-780.
- 27 Gofin R, Kark JD, Friedlander Y. Cigarette smoking, blood pressure and pulse rate in the Jerusalem Lipid Research Clinic Prevalence Study. *Isr J Med Sci.* 1982; 18: 1217-1222.
- 28 Seki M, Inoue R, Ohkubo T, et al. Association of environmental tobacco smoke exposure with elevated home blood pressure in Japanese women: the Ohasama study. *J Hypertens.* 2010; 28: 1814-1820.
- 29 Leone A, Guidi A, Bertoincini G, et al. Ambulatory blood pressure monitoring in passive smoking and atropine – response in healthy volunteers. *Singapore Med J.* 2000; 41: 104-106.
- 30 Pijanowska M, Zajackowska M. Passive smoking and patterns of 24-hour ambulatory blood pressure in healthy children. *Pol Merkur Lekarski.* 2004; 16: 320-322.
- 31 Makris TK, Thomopoulos C, Papadopoulos DP, et al. Association of passive smoking with masked hypertension in clinically normotensive nonsmokers. *Am J Hypertens.* 2009; 22: 853-859.
- 32 World Health Organization. Report on the Global Tobacco Epidemic 2008. The MPOWER package. Geneva: WHO, 2008.
- 33 World Health Organization. Report on the Global Tobacco Epidemic 2009. Implementing smoke-free environments. Geneva: WHO, 2009.

# 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 włączono 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 znacząco 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.

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Praca wpłynęła: 30.05.2014.  
Przyjęta do druku: 21.07.2014.  
Publikacja online: 21.07.2014.  
Nie zgłoszono sprzeczności interesów.

Pol Arch Med Wewn. 2014; 124 (9): 436-442

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