

Using Ambulatory Blood Pressure Monitoring to Assess Blood Pressure of Firefighters with Parental History of Hypertension

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Objective: To evaluate the influence of family history of systemic arterial hypertension (FSAH) on the effect of stress from work in Uniformed Firefighters (BMCs) through Ambulatory Blood Pressure Monitoring (ABPM).

Methods: A prospective case-control study. Sixty-six healthy BMC underwent ABPM during 12 hours of work at the Communication Center (CC). Thirty-four had hypertensive parents (group 1) and thirty-two had normotensive parents (group 2).

Results: Group 1 differed from group 2 in that it showed higher mean systolic (134.1 ± 9.9 mmHg X 120.8 ± 9.9 mmHg $p < 0.0001$) and diastolic (83.8 ± 8.3 mmHg X 72.9 ± 8.6 mmHg $p < 0.001$) blood pressure, in addition to greater systolic (31.4 ± 25.6 % X 9.4 ± 9.4 % $p = 0.0001$) and diastolic (28.3 ± 26.6 % X 6.1 ± 8.9 % $p = 0.0001$) loads. The prevalence of systemic arterial hypertension (SAH) in group 1 at the workplace was 32.3%. Monitored away from the job, these subjects showed normal blood pressure (functionally hypertensive). Group 2 revealed normal blood pressure (BP) at work.

Conclusion: Higher blood pressure in BMC with hypertensive parents is explained independently by the SAH. Subjects who developed SAH during their work at the CC may be considered functionally hypertensive, whereas those with normotensive parents and who underwent psychological stress are free of blood pressure changes.

Key words: Hypertension, ambulatory blood pressure monitoring (ABPM), psychological stress.

The Communication Center (CC) of the Rio de Janeiro State Firefighters (CBMERJ) – responsible for incoming calls, triage, and confirmation of events that occur in the state – receives countless calls for help every day from the population of the State of Rio de Janeiro. The Communication Center is manned by Uniformed Firefighters (BMCs) who receive SOS notifications, take note of the characteristics and consequences of all events, and pass the information on to the firefighters responsible for handling the actual rescue and/or providing emergency medical care. The BMCs' job is psychologically very demanding (too many SOS calls and mental overload) despite the low level of administrative autonomy (strict supervision on the part of a senior officer due to the vertical hierarchy), often leading to feelings of anger, fear of erring, or insecurity in the performance of their duties, which configures an occupational psychological stress generating job^{1,2}. Familial Systemic Arterial Hypertension (FSAH) is considered a risk factor for the development of high blood pressure and has aroused the scientific community's interest in carrying out various tasks aimed at detecting functional^{3,4}, structural^{4,6}, hemodynamic⁷⁻⁹, and/or metabolic¹⁰⁻¹¹ alterations that precede the diagnosis of Systemic Arterial Hypertension (SAH). Many authors have

suggested that a probable lower sympathetic nervous system activation threshold in normotensive individuals with FSAH may be responsible for exaggerated response to psychological stress¹²⁻¹⁴. The object of this paper was to evaluate the influence of Systemic Arterial Hypertension on the effect of occupational stress in BMCs by means of Ambulatory Blood Pressure Monitoring (ABPM).

Methods

This is a case-control study carried out between January and August 1999, in which 66 healthy male BMCs were distributed into two groups according to the presence or not of FSAH. All subjects underwent ABPM during 12 consecutive working hours at the CC. Group 1 (case group) consisted of 34 BMCs with FSAH, and group 2 (control group) consisted of 32 BMC without FSAH. The healthy status was determined through anamnesis, physical examination, and past medical history (medical charts review). Inclusion criteria were age between 21 and 49, healthy status confirmed by periodic medical check-ups provided by the CBMERJ, normal physical examination, systolic blood pressure (SBP) < 140 mmHg, and

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Manuscript received December 28, 2004; revised manuscript received August 21, 2005; accepted February 4, 2006.

diastolic blood pressure (DBP) < 90 mmHg. Exclusion criteria were cardiac arrhythmias, SAH, past history of hypertensive crisis or of any other heart disease and suspected FSAH. Body mass index (BMI) was calculated by dividing body weight (in kilograms) by squared height (in meters) ($BMI = w/h^2$). The presence of FSAH was based on the BMC's father and/or mother having a history of SAH and/or use of antihypertensive drugs (interviews with study participants and their relatives⁴). Conventional BP was measured before work shift at the CC by using a mercury sphygmomanometer after a five-minute rest, in the seated position, on the right arm supported at heart level. Cuff size was selected according to arm circumference. Blood pressure was taken three times, two minutes apart, and the lowest reading was considered as criterion for study inclusion. Ambulatory blood pressure monitoring (ABPM) was performed using oscillometric devices, validated by the Association for the Advancement of Medical Instrumentation (AAMI) and British hypertension Society (BHS)¹⁵, and cuff sizes similar to those used for BP auscultatory measurements. The device was set to obtain automatic readings every 10 minutes, with a cuff deflation rate of 8 mmHg/second. Measurement rejection followed criteria set forth by the II Brazilian Consensus for Ambulatory Blood Pressure Monitoring (*II Consenso Brasileiro para o Uso da Monitorização Ambulatorial da Pressão Arterial*)¹⁶ (HR > 125 bpm or < 40 bpm, SBP > 240 mmHg or < 50 mmHg, and DBP > 140 mmHg or < 40 mmHg). Examinations were considered valid when a minimum of 48 readings at least every two hours were valid. Mean measurement in both groups was 60 readings by examination. Examinations with mean SBP > 140 mmHg and/or DBP > 90 mmHg were repeated 36 hours after baseline monitoring, but this time for 24 hours during the subject's day off. The concept of systolic pressure load (SPL) and diastolic pressure load (DPL) were those described in the II Brazilian Consensus for Ambulatory Blood Pressure Monitoring¹⁶ (% of readings > 140 mmHg for SBP and > 90 mmHg for DBP).

Statistical analysis - Results were analyzed using the Student's t-test or Mann-Whitney test (means), chi-square test or Fisher's exact test (proportions), Pearson's correlation coefficient, multiple linear regression analysis and covariance analysis (heterogeneous groups). P values < 0.05 were considered statistically significant. The ethical principles for medical research established by the Helsinki Declaration and ratified by the Brazilian Code of Medical Ethics were followed.

Results

Group 1 differed from group 2 in that it had higher mean age (37.8 ± 5.5 X 31.7 ± 6.4 ; $p = 0.0001$), BMI (26.8 ± 3.3 X 24.5 ± 3.7 ; $p = 0.007$), and length of time on the job (14.1 ± 6.4 X 8.9 ± 7.9). Mean conventional measurements obtained at the CC before study entry did not differ between group 1 (120.6 mmHg \pm 9.0 mmHg for SBP and 75.2 mmHg \pm 8.2 mmHg for DBP) and group 2 (118.7 mmHg \pm 8.9 mmHg for SBP and 72.8 mmHg \pm 8,5 mmHg for DBP). However, during work period at the CC, ABPM showed that mean SBP and DBP of group 1 (134.1 mmHg \pm 9.9 mmHg and 83.8 mmHg \pm 8.3 mmHg) were higher than those of group 2 (120.8 mmHg \pm 9.9 mmHg and 72.9 mmHg \pm 8.6 mmHg), with $p = 0.0001$ for SBP and

$p = 0.001$ for DBP (Figure 1). Mean SBP and DBP of eleven subjects of group 1 (32.3%) were higher than 140 mmHg and/or 90 mmHg during the work period, and all subjects had SPL and/or DPL above 50%. In group 2, also during the work period, no subject showed mean SBP and/or DBP above 140 mmHg and/or 90 mmHg, respectively. Monitored on their day off, they showed normal mean SBP, DBP, SPL, and DPL. Comparing mean SPL and DPL of group 1 ($31.4\% \pm 25.6\%$ and $28.2\% \pm 26.6\%$) with those of group 2 ($9.4\% \pm 9.4\%$ and $6\% \pm 8.9\%$), both were found to be higher in group 1, with $p = 0.0001$ for SPL and $p = 0.001$ for DPL (Figure 2). Table 1 presents Pearson's correlation coefficient and significance level for every association evaluated. Table 2 shows the subgroup of variables selected, in order of importance, by multiple linear regression analysis and their significance level for mean SBP, DBP, SPL and DPL. It has been demonstrated that FSAH influence is independent of the other variables, which may explain the higher mean SBP, DBP, SPL and DPL found in group 1. Table 3 presents the covariance analysis for FSAH relative to mean SBP, DBP, SPL and DPL. It was noted that even when the covariable effect, either individually or collectively, is under control, mean SBP, DBP, SPL and DPL are affected by the FSAH.

Discussion

Although mean SBP and DBP of group 1 are higher than those of group 2, both are considered normal for the awake period (SBP < 135 mmHg and DBP < 85 mmHg)¹⁶⁻¹⁹. Countless ABPM studies comparing subjects with hypertensive parents with subjects with normotensive parents have found higher values in the former, although still within normal limits^{20,21}. Normalization of mean SBP and DBP during the day off of those subjects whose readings were higher than 140 mmHg and/or 90 mmHg (32.3%) at the workplace, coupled with the fact that they showed absolutely normal mean conventional measurements before beginning their workday (120.9 mmHg \pm 9.2 mmHg for SBP and 75.6 mmHg \pm 8,4 mmHg for DBP – Figure 3), suggests the diagnosis of latent hyperreactivity (functional hypertension)²², which

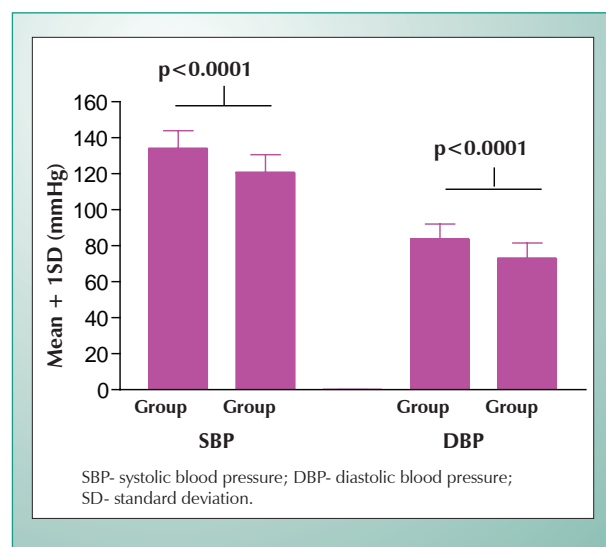


Fig. 1 – Mean SBP and DBP during work.

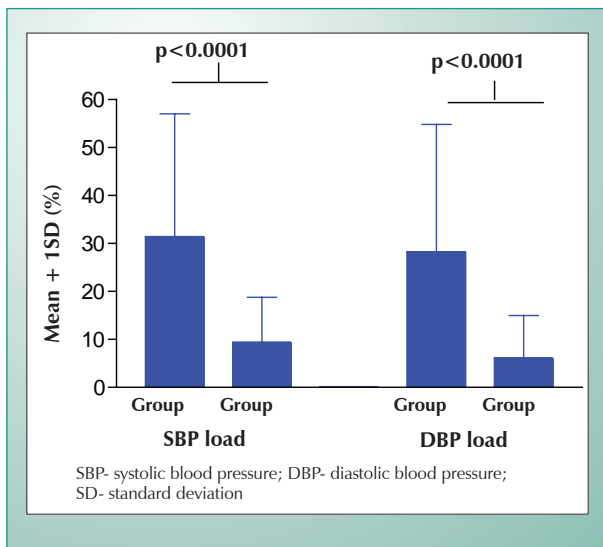


Fig. 2 – Mean SBP and DBP loads during work.

Variables	Work time	Age	BMI
	0.35870	0.41088	0.41894
SBP mean	0.0031	0.0006	0.0005
	0.47204	0.54280	0.39704
DBP mean	0.0001	0.0001	0.0010
	0.34182	0.36875	0.41029
SBP load	0.0050	0.0023	0.0006
	0.43941	0.44575	0.35783
DBP load	0.0002	0.0002	0.0032

SBP- systolic blood pressure; DBP- diastolic blood pressure; BMI- Body mass index.

Table 1 - Pearson's correlation coefficient (above) and the respective significance level (below) for every association assessed.

ABPM variables	Selected variables	p value
SBP mean	1a FSAH	0.0001
	2 ^a BMI	0.015
DBP mean	1a FSAH	0.0001
	2 ^a Age	0.001
SBP load	1a FSAH	0.0001
	2 ^a BMI	0.014
DBP load	1a FSAH	0.0001
	2 ^a Length of time on the job	0.01

SBP- systolic blood pressure; DBP- diastolic blood pressure; BMI- body mass index; FSAH- familial systemic arterial hypertension.

Table 2 - Multiple linear regression analysis and its respective significance level for ABPM variables

warrants close clinical follow-up. If these subjects progress to established SAH in the future, the prevalence of this syndrome among BMC with hypertensive parents will be higher than that found among journalists and advertising people (21%), public transportation workers (18.9%), bank employees and insurance workers (18.6%, steel workers (17.3%), automobile industry workers (11.4%), merchants (12.1%), textile workers (12.9%), and professionals (11%)²³, prison inmates (26.3%), and navy officers (6.7%)²⁴; nurses (27.9%)²⁵. Although one of the limitations of this study was that sympathetic tone, intensity of alarm reaction and operational responses of peripheral effector organs were not assessed, the initial hypothesis is that because of their genetic inheritance, these BMC have high sympathetic tone, with consequent circulatory hyperkinetic manifestation and greater blood pressure reactivity to environmental psychological stresses²⁶⁻³⁰. This fact itself is very important, since it may show, early on, the tendency of these BMC to develop established SAH. It is currently agreed that hyperkinesis is related to neurogenic activity exacerbation, despite diverging opinions on the actual origin of this phenomenon. While some researchers^{31,32} believe that there is a predominance of psychological and behavioral aspects, others³³ say that the increase in peripheral sympathetic nervous system activity is more relevant, the latter being thought to be the early change in SAH development and/or dysfunctions of its receptors' sensitivity. The theory of the neurogenic origin of SAH, albeit widespread, is controversial in studies evaluating sympathetic nervous system activity through plasma and urinary catecholamine levels. Many authors have detected higher plasma noradrenaline levels in subjects with hypertensive parents³⁴⁻³⁶, although others did not confirm an elevation in plasma or urinary noradrenaline; in addition, microneurography shows no differences in sympathetic activity among subjects with hypertensive parents and subjects with normotensive parents³⁷⁻⁴¹. Mean SPL and DPL of group 1 are within borderline values¹⁶ (between 25% and 50%), while other authors¹⁷ classify mean normal SPL (>30%) and borderline DPL (between 15% and 30%). Mean SPL and DPL of group 2, on the other hand, are normal^{16,17} (<25%). The importance of these findings lays in the fact that subjects in group 1, with mean SPL and DPL ranging from 20% to 40%, will develop hypertension in the future, despite the low correlation with left ventricular (LV) anatomical and functional changes⁴². According to some studies⁴², blood pressure loads (BPL) lower than 20% have normal clinical and prognostic significance; if less than 40%, they present a correlation of less than 17% in LV functional changes, whereas if greater than 40%, the likelihood of changes in cardiac parameters is as high as 61%. The increase in BPL also correlates well with reduced peak LV filling rate⁴². In addition, when casual BP is high, target organ changes reach at most 19%, whereas when casual BP and BPL are altered, 64% are diagnosed with target organ damage⁴³. Thus, BPL greater than 50% translates, clinically, into established ASH with prognosis of a 60% to 90% correlation with target organ lesions and, therefore, treatment is indicated⁴². However, even if a direct relationship between BPL values, especially greater than 50%, and target organ lesions is documented, the tendency in the most recent guidelines for ABPM is not to take these values into account in the clinical interpretation, since this criterion has been subject

ABPM variables	Covariables	F value	p value
SBP mean	Age	17.3	0.0001
	BMI	20.8	0.0001
	Length of time on the job	20.7	0.0001
DBP mean	The three of them simultaneously	13.5	0.0005
	Age	11.8	0.001
	BMI	18.8	0.0001
SBP load	Length of time on the job	16.2	0.0002
	The three of them simultaneously	8.9	0.004
	Age	11.8	0.001
DBP load	BMI	13.6	0.0005
	Length of time on the job	13.8	0.0004
	The three of them simultaneously	9.0	0.003
SBP load	Age	9.4	0.003
	BMI	13.7	0.0005
	Length of time on the job	11.4	0.001
DBP load	The three of them simultaneously	7.9	0.006

SBP- systolic blood pressure; DBP- diastolic blood pressure; BMI- body mass index.

Table 3 – Covariance analysis for Family History of Systemic Arterial Hypertension relative to ABPM variables

to much criticism, the foremost of which is that BPL values alone do not reflect the SAH importance, because similar BPL values fail to reveal the magnitude of SAH, that is, subjects with SAH and the same BPL may actually have distinct BP variations and, therefore, different prognoses⁴⁴. Because of the cross-sectional nature of this study, it is suggested that prospective BP measurements be performed regularly for a better clinical course monitoring of both groups and, in particular, of the BMC considered functionally hypertensive. Based on that stated above regarding mean SBP, DBP, SPL,

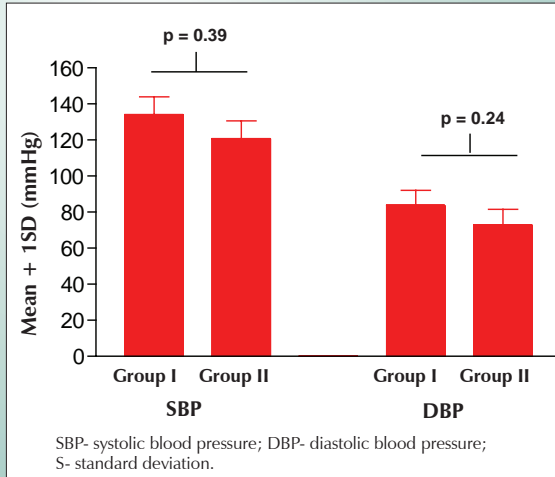


Fig. 3 – Mean SBP and DBP before beginning work.

and DPL in the BMC population, it may stand to reason that group 1, in general, despite the low likelihood of target organ involvement, should be followed more closely and preventive measures be adopted to avoid SAH development. This group has a BMC subpopulation with abnormally high mean SBP, DBP, SPL and DPL on the job that normalizes during day off (hyperreactors) and, therefore, stricter prophylactic measures should be focused so that chronic elevated BP does not trigger adaptive and structural responses, turning these workers into future hypertensives. According to the methodology used in this study and based on the analysis of the results obtained, it is possible to conclude that higher BP in BMC with hypertensive parents is independently explained by the FSAH. In addition, subjects who developed SAH during their work at the CC may be considered functionally hypertensive, while those with normotensive parents, yet who underwent psychological stress, are free of BP changes. Moreover, the CC is not an appropriate place to work for a BMC with SAH or FSAH to work. And ABPM is an effective method for identifying clinically healthy subjects who develop SAH at the workplace.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

References

1. Van-Egeren LF. The relationship between job strain and blood pressure at work, at home, and during sleep. *Psychosom Med.* 1992; 54: 337-43.
2. Andrade Filho A, Santos Junior AE. Aparelho cardiovascular. In: Mendes R. (Ed.) *Patologia do trabalho.* São Paulo: Atheneu; 1997. p. 311-27.
3. Mehta SK, Super DM, Anderson RL, Harcar-Sevcik RA, Badjak M, Liu X, et al. Parental hypertension and cardiac alterations in normotensive children and adolescents. *Am Heart J.* 1996; 131: 81-8.
4. Almeida CM, Siqueira-Filho AC, Rachid MB, Rachid J, Kamel CS. Avaliação morfo-funcional cardíaca em jovens normotensos, filhos de hipertensos: estudo Doppler-ecocardiográfico prospectivo. *Arq Bras Cardiol.* 1998; 71: 681-6.
5. Radice M, Allì C, Avanzini F, Tullio M, Mariotti G, Taioli E, et al. Left ventricular structure and function in normotensive adolescents with a genetic predisposition to hypertension. *Am Heart J.* 1986; 111:115-20.
6. Gottdiener JS, Brown J, Zoltick J, Fletcher RD. Left ventricular hypertrophy in men with normal blood pressure: relation to exaggerated blood pressure response to exercise. *Ann Intern Med.* 1990; 112: 161-6.

7. Radice M, Alli C, Avanzini F, Di Tullio M, Mariotti G, Zussino A. Role of blood pressure response to provocative tests in the prediction of hypertension in adolescents. *Eur Heart J*. 1985; 6: 490-6.
8. Molineux D, Steptoe A. Exaggerated blood pressure responses to submaximal exercise in normotensive adolescents with a family history of hypertension. *J Hypertens*. 1988; 6: 361-5.
9. Bond V JR, Franks BD, Tearney RJ, Wood B, Melendez MA, Johnson L, et al. Exercise blood pressure response and skeletal muscle vasodilator capacity in normotensives with positive and negative family of hypertension. *J Hypertens*. 1994; 12: 285-90.
10. Taskinen MR, Kuusi T, Helve E, Nikkila EA, Yki-Jarvinen H. Insulin therapy induces antiatherogenic changes of serum lipoproteins in noninsulin-dependent diabetes. *Arteriosclerosis*. 1988; 8: 168-77.
11. Ohno Y, Suzuki H, Yamakawa H, Nakamura M, Otsuka K, Saruta T. Impaired insulin sensitivity in young, lean normotensive offspring of essential hypertensives: possible role of disturbed calcium metabolism. *J Hypertens*. 1993; 11: 421-6.
12. Anderson EA, Mahoney LT, Lauer RM. Progeny of hypertensives have altered hemodynamic mechanisms during mental challenge. *Circulation*. 1985; 72 (Suppl): III-259.
13. Graettinger WF, Neutel JM, Smith DH, Weber MA. Left ventricular diastolic filling alterations in normotensive young adults with a family of systemic hypertension. *Am J Cardiol*. 1991; 68: 51-6.
14. Ferrier C, Cox H, Esler M. Elevated total body noradrenaline spillover in normotensive members of hypertensive families. *Clin Sci*. 1993; 84: 225-30.
15. Jones CR, Taylor K, Chowieniczky P, Poston L, Shennan AH. A validation of the Mobil O Graph (version 12) ambulatory blood pressure. *Blood Press Monit*. 2000; 5: 233-8.
16. Amodeo C, Giorgi DM, Mion JR D. II Consenso brasileiro para o uso da monitorização ambulatorial da pressão arterial. *Arq Bras Cardiol*. 1997; 69: 359-67.
17. Pickering TG. Recommendations for the use of home (self) and ambulatory blood pressure monitoring. American Society of Hypertension Ad Hoc Panel. *Am J Hypertens*. 1996; 9: 1-11.
18. The sixth report of the joint national committee on prevention, evaluation and treatment of high blood pressure. *Arch Intern Med*. 1997; 157: 2413-45.
19. Staessen JA, Fagard RH, Lijnen PJ. Ambulatory blood pressure and blood pressure measured at home: progress report on a population study. *J Cardiovasc Pharmacol*. 1994; 23 (Suppl. 5): S5-11.
20. Ravogli A, Trazzi S, Villani A, Mutti E, Cuspidi C, Sampieri L, et al. Early 24-hour blood pressure elevation in normotensive subjects with parental hypertension. *Hypertension*. 1990; 16: 491-7.
21. Schwartz GL, Turner ST, Sing CF. Twenty-four-hour blood pressure profiles in normotensive sons of hypertensive parents. *Hypertension*. 1992; 20: 834-40.
22. Fiedler N, Favata E, Goldstein BD, Gochfeld M. Utility of occupational blood pressure screening for the detection of potential hypertension. *J Occup Med*. 1988; 30: 943-8.
23. Ribeiro MD, Ribeiro AB, Neto CS, Chaves CC, Karter CE, Iunes M, et al. Hypertension and economic activities in São Paulo, Brazil. *Hypertension*. 1981; 3: 233-7.
24. Carvalho JJ, Silva NA, Oliveira JM, Arquelles E, Silva JA. Pressão arterial e grupos sociais: estudos epidemiológicos. *Arq Bras Cardiol*. 1983; 40: 115-20.
25. Aquino EM, Magalhães LC, Araújo MJ. Confiabilidade da medida de pressão arterial sanguínea em um estudo de hipertensão arterial. *Arq Bras Cardiol*. 1996; 66: 21-4.
26. Falkner B, Onesti G, Angelakos ET, Fernandes M, Langman C. Cardiovascular response to mental stress in normal adolescents with hypertensive parents: hemodynamics and mental stress in adolescents. *Hypertension*. 1979; 1: 23-30.
27. Ohlsson O, Henningsen NC. The effect on blood pressure; ECG and heart rate of psychological stress; static and dynamic muscle work: a study on members of families with known aggregation of essential hypertension. *Acta Med Scand*. 1982; 211: 113-20.
28. Horikoshi Y, Tajima I, Igarashi H, Inui M, Kasahara K, Noguchi T. The adreno-sympathetic system; the genetic predisposition to hypertension; and stress. *Am J Med Sci*. 1985; 289: 186-91.
29. Manuck SB, Proletti JM. Parental hypertension and cardiovascular response to cognitive and isometric challenge. *Psychophysiology*. 1992; 19: 481-9.
30. Perini C, Muller FB, Rauchfleisch U, Battegay R, Hobi V, Buhler FR. Psychosomatic factors in borderline hypertensive subjects and offspring of hypertensive parents. *Hypertension*. 1990; 16: 627-34.
31. Julius S, Esler M. Autonomic nervous cardiovascular regulation in borderline hypertension. *Am J Cardiol*. 1975; 36: 685-95.
32. Jorgensen SR, Houston BK. Family history of hypertension, personality pattern, and cardiovascular reactivity to stress. *Psychosom Med*. 1986; 48: 102-17.
33. De Champlain J, Petrovich M, Gonzales M, Lebeau R, Nadeau R. Abnormal cardiovascular reactivity in borderline and mild hypertension. *Hypertension*. 1991; 17 (Suppl.III): III 22-8.
34. McCrory, WW, Klein AA, Rosenthal RA. Blood pressure, heart rate, and plasma catecholamines in normal and hypertensive children and their siblings at rest and after standing. *Hypertension*. 1982; 4: 507-13.
35. Ferrier C, Cox H, Esler M. Elevated total body noradrenaline spillover in normotensive members of hypertensive families. *Clin Sci*. 1993; 84: 225-30.
36. Masuo K, Mikami H, Ogihara T, Tuck ML. Familial hypertension; insulin; sympathetic activity; and blood pressure elevation. *Hypertension*. 1998; 32: 96-100.
37. Ferrier C, Beretta-Piccoli C, Weidmann P, Bianchetti MG. Different blood pressure responses to diuretic treatment in normotensive subjects with and without a family history of hypertension. *J Hypertens*. 1983; 1(Suppl): 31-4.
38. Umemura S, Uchino K, Yasuda G, Ishikawa Y, Hatori Y, Tochikubo O, et al. Altered platelet alpha 2-adrenoceptors and adrenaline response in adolescents with borderline hypertension who have a family history of essential hypertension. *J Hypertens*. 1988; 6(Suppl.): S568-71.
39. Ferrara LA, Moscato TS, Pisanti N, Marotta T, Krogh V, Capone D, et al. Is the sympathetic nervous system altered in children with familial history of arterial hypertension? *Cardiology*. 1988; 75: 200-5.
40. Noll G, Wenzel RR, Schneider M, Oesch V, Binggeli C, Shaw S, et al. Increased activation of sympathetic nervous system and endothelin by mental stress in normotensive offspring of hypertensive parents. *Circulation*. 1996; 93: 866-9.
41. Hausberg M, Sinkey CA, Mark AL, Hoffman RP, Anderson EA. Sympathetic nerve activity and insulin sensitivity in normotensive offspring of hypertensive parents. *Am J Hypertens*. 1998; 11: 1312-20.
42. White WB, Dey HM, Schulman P. Assessment of the daily blood pressure load as a determinant of cardiac function in patients with mild to moderate hypertension. *Am Heart J*. 1989; 118: 782-95.
43. Floras JS, Jones JV, Hassam MD, Osikowska B, Server PS, Sleight P. Cuff and ambulatory blood pressure in subjects with essential hypertension. *Lancet*. 1981; 2: 107-9.
44. Pickering TG. Ambulatory monitoring and blood pressure variability. *Science Press*. 1991; 1: 5-9.