Cardiovascular Response to Postural Change in Normotensive Subjects with a Family History of Hypertension

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Abstract

Objective: To observe the magnitude of cardiovascular response to postural changes in normotensive subjects with a family history of hypertension as compared to those without a family history of hypertension.

Methods: Undergraduate normotensive subjects with (n = 16) and without (n = 14) hypertensive parents did postural changes from supine to standing. Cardiovascular parameters such as systolic blood pressure, diastolic blood pressure and pulse rate were measured during supine and immediately after standing. The differences in cardiovascular reactivity to postural changes between the two groups was defined by median value of delta cardiovascular responses across subjects. Delta value was calculated by subtracting supine measure from standing measure. The differences in the proportion of subjects having cardiovascular responses above median value of delta change score between the two groups were analyzed with Chi square. P value < 0.05 was considered significant.

Results: Median values of delta change of systolic and diastolic blood pressure were 3.5 mm Hg and 2 mm Hg, respectively, whereas median value of delta change of pulse rate was 12 bpm. Proportion of normotensive subjects who had exaggerated systolic or diastolic blood pressure responses to postural changes were significantly higher in those with parental history of hypertension as compared to those without parental history of hypertension. Proportion of subjects who had exaggerated pulse rate response to postural changes was not significantly different between the two groups.

Conclusion: Normotensive young adults with a family history of hypertension showed heightened blood pressure, but not of heart rate, reactivity to postural change.

Keywords: Cardiovascular Reactivity; Postural Change; Normotensive; Family History of Hypertension

Introduction

Cardiovascular reactivity is a set of changes in cardiovascular parameters, such as blood pressure, heart rate, and other hemodynamic parameters, in response to a stressor. Cardiovascular reactivity is a non-invasive method to detect the pre-clinical state of cardiovascular disease. Pre-clinical cardiovascular disease state is the pathogenic change in the cardiovascular structure and function that can progress into cardiovascular diseases, such as hypertension, myocardial infarct, and stroke [1,2].

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Essential hypertension occurs more frequently in individuals with a family history of the disease. Subjects with a family history of hypertension have higher prevalence of hypertension than those without. Normotensive subjects with a family history of hypertension can be regarded as a model for prehypertension. To identify the functional changes in the cardiovascular system before the rising of blood pressure is important because when hypertension has already occurred, the initial changes may be obscured by adaptations invoked by the high pressure [3,4].

Postural change from supine to standing position is among various stimuli to stimulate cardiovascular system. The orthostatic challenges the cardiovascular system to the effect of gravity to the blood. It was reported that orthostatic hypotension is associated to an increased risk of hypertension, coronary heart disease and stroke. In the opposite, orthostatic hypertension will cause hemodynamic stress, lead to vascular damage and is associated also to an increased risk of developing hypertension [5,6].

There is no study, however, that reported the cardiovascular responses of normotensive subjects with a family history of hypertension to postural changes. Therefore, this study examined the cardiovascular responses to postural changes from supine to standing in prehypertensive model, i.e. normotensive subjects with a family history of hypertension, as compared to normotensive subjects without a family history of hypertension. This study found that proportion of heightened blood pressure, but not pulse rate, responses to postural changes was significantly higher among normotensive subjects with a family history of hypertension than those without a family history of hypertension.

Materials and Methods

Study design

This was quasi experimental study between-subjects design, which compared cardiovascular reactivity to postural changes from supine to standing in healthy normotensive subjects with and without a family history of hypertension. Blood pressure and pulse rate were measured during pre-test (supine) and test (standing). Similar to our previous study on cardiovascular reactivity to mental and physical tests among normotensive young adult subjects with and without a family history of hypertension [7], in this study we used a delta change score as a measure of cardiovascular reactivity, which was calculated as the difference between the cardiovascular parameters measured in the test and pre-test periods. Mean ± standard deviation was used to summarize the subjects’ characteristics. Exaggerated cardiovascular responses was defined as subjects’ systolic blood pressure, diastolic blood pressure and pulse rate responses that was above the median value of each cardiovascular parameters respectively in responses to postural changes.

Selection criteria

Inclusion criteria

The subject’s age was between eighteen to thirty years old. Female subject had body mass index < 27.3 kg/m² and male subject had body mass index < 27.3 kg/m². The subject had systolic blood pressure (SBP) < 140 mmHg and diastolic blood pressure (DBP) < 90 mmHg in concordance with the criteria of normotensive subjects based on Seventh report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure [8].

Exclusion criteria

Subjects reported whether she/he had renal diseases, hormonal disturbances or neuropsychiatric disorder.
Family history of hypertension

Similar to our previous study on cardiovascular reactivity to mental and physical tests among normotensive young adult subjects with and without a family history of hypertension [7], in this study we defined family history of hypertension by parental hypertension. Subjects were considered having family history of hypertension if she/he reported that either her/his father or mother or both had one of the following criteria: high blood pressure (SBP > 140 mmHg or DBP > 90 mmHg), being diagnosed hypertension, being on medication for hypertension. Subjects were considered not having family history of hypertension if she/he reported that her/his father and mother did not have all the following criteria: high blood pressure (SBP > 140 mmHg or DBP > 90 mmHg), being diagnosed hypertension, being on medication for hypertension.

Postural change

In the pre-test period, subject took a rest in a supine position for ten minutes. At the end of the rest period, cardiovascular parameters were measured for two times. The two closest measurements were being averaged. In the test period, subject stood quickly by the side of the bed. During standing, the subject should not move. Cardiovascular parameters were measured immediately when the subject stood.

Data handling

Microsoft Office Excel 2003 was used for data reduction and Statistical Package for Social Sciences version 12.0 (SPSS Inc., Chicago, USA) for data summarize and analysis.

Data reduction

Delta change score rather than absolute value were chosen to control the baseline level. The magnitude of the reactivity measured by delta change score was not necessarily dependent upon the baseline value. Moreover, delta change score yield reliable measures of blood pressure and heart rate reactivity to laboratory stimuli. A delta change score of cardiovascular reactivity was calculated by subtracting the cardiovascular parameters measured in the pre-test period from the cardiovascular parameters measured during the test period.

Data summary

The results were summarized as mean ± standard deviation.

Data analysis

To determine whether any differences of subjects’ characteristics were existed independent t-test was used. To compare whether any differences in the proportion of exaggerated cardiovascular responses in systolic blood pressure, diastolic blood pressure and pulse rate to postural changes between the two groups were existed Chi-square was used. p value < 0.05 was considered as the statistical significance level.

Results

The sample of this study, i.e. normotensive subjects with and without a family history of hypertension, did not differ significantly (p > 0.05) in the term of age, body mass index, systolic blood pressure, diastolic blood pressure and pulse rate (Table 1).

After the subjects did postural change, the delta change score of systolic blood pressure, diastolic blood pressure and pulse rate were calculated. The median of delta scores of systolic blood pressure, diastolic blood pressure and pulse rate were 3.5 mm Hg, 2 mm Hg and 12 bpm, respectively. These median values were used to define the exaggerated cardiovascular reactivity to postural change.

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Table 1: Characteristics of subjects

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Without a FHoH (n = 14)</th>
<th>With a FHoH (n = 16)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>22.43 ± 2.87</td>
<td>23 ± 3.01</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>2.14 ± 1.55</td>
<td>19.89 ± 2.12</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>116.86 ± 5.22</td>
<td>116.37 ± 7.56</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>79 ± 4.99</td>
<td>84.44 ± 6.13</td>
</tr>
<tr>
<td>Pulse rate (bpm)</td>
<td>74.28 ± 7.84</td>
<td>73.125 ± 6.73</td>
</tr>
</tbody>
</table>

Note: FHoH: Family History of Hypertension; BMI: Body Mass Index; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; bpm: Beats Per Minute.

Data are summarized as mean ± standard deviation.

Based on the median of the delta change score, whether any significant differences in the proportion of exaggerated cardiovascular reactivity to postural change were existed between normotensive subjects with and without a family history of hypertension were analyzed. Normotensive subjects with a family history of hypertension had significantly higher proportion of exaggerated cardiovascular response to postural change in systolic blood pressure (p = 0.001) and diastolic blood pressure (p = 0.001), but not pulse rate (p = 0.44) (Table 2a-2c).

Table 2a: Proportion of exaggerated systolic blood pressure response to the postural change in normotensive subjects with and without a family history of hypertension.

<table>
<thead>
<tr>
<th>∆SBP</th>
<th>Without a FHoH</th>
<th>With a FHoH</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥3.5 mm Hg</td>
<td>2</td>
<td>13</td>
<td>15</td>
</tr>
<tr>
<td>&lt;3.5 mm Hg</td>
<td>12</td>
<td>3</td>
<td>15</td>
</tr>
<tr>
<td>Total</td>
<td>14</td>
<td>16</td>
<td>30</td>
</tr>
</tbody>
</table>

Note. FHoH: Family History of Hypertension; ∆: Delta Change Score; SBP: Systolic Blood Pressure.

Table 2b: Proportion of exaggerated diastolic blood pressure response to the postural change in normotensive subjects with and without a family history of hypertension.

<table>
<thead>
<tr>
<th>∆DBP</th>
<th>Without a FHoH</th>
<th>With a FHoH</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥2 mm Hg</td>
<td>2</td>
<td>12</td>
<td>14</td>
</tr>
<tr>
<td>&lt;2 mm Hg</td>
<td>12</td>
<td>4</td>
<td>16</td>
</tr>
<tr>
<td>Total</td>
<td>14</td>
<td>16</td>
<td>30</td>
</tr>
</tbody>
</table>

Note. FHoH: Family History of Hypertension; ∆: Delta Change Score; DBP: Diastolic Blood Pressure.

Table 2c: Proportion of exaggerated pulse rate response to the postural change in normotensive subjects with and without a family history of hypertension.

<table>
<thead>
<tr>
<th>∆Pulse rate (bpm)</th>
<th>Without a FHoH</th>
<th>With a FHoH</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥12 bpm</td>
<td>3</td>
<td>6</td>
<td>9</td>
</tr>
<tr>
<td>&lt;12 bpm</td>
<td>11</td>
<td>10</td>
<td>21</td>
</tr>
<tr>
<td>Total</td>
<td>14</td>
<td>16</td>
<td>30</td>
</tr>
</tbody>
</table>

Note. FHoH: Family History of Hypertension; ∆: Delta Change Score; bpm: Beats Per Minute.

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Discussion

This is the first study that reported orthostatic hypertension in normotensive subjects with a family history of hypertension (Table 2a and 2b). Previously, orthostatic hypertension is defined as the rise of systolic blood pressure ≥ 20 mm Hg in standing position after immediately move from supine position. Recently, the criteria of orthostatic hypertension is broadened including the rise of diastolic blood pressure ≥ 10 mm Hg in postural change from supine to standing [5,9]. In our study, exaggerated blood pressure response to postural change was classified based on the median value of either the delta systolic blood pressure response (≥ 3.5 mm Hg) or the delta diastolic blood pressure response (≥ 2 mm Hg) or both to postural changes across all the subjects in this study.

Normotensive subjects with a family history of hypertension have been regarded as a pre hypertension model. They are not developed yet high blood pressure; however, they already have structural and functional changes in the cardiovascular system. Therefore, they show exaggerated cardiovascular reactivity in response to various laboratory stimuli or real life stressors. Family history of hypertension is known as an unmodified risk factor to hypertension [4,10,11].

During postural change from a supine to a standing position, gravity load increases transmural pressure in the vein in the lower extremities. Both mean arterial and venous blood pressure increase linearly with vertical distance below the heart level and would reach 180 and 90 mmHg, respectively. When the individual does not move during standing, around 500 mL of blood is pooled in the venous capacitance vessels of the lower extremities, which further reduces venous return. As a consequence, stroke volume decreases and pulse pressure drops [5].

The aortic and carotid baroreceptors detect the decrease in pulse pressure whereas the veno-atrial stretch receptors detect the drop in venous return. The impulses are transmitted to the nucleus tractus solitarius through the IXth and Xth nerve fibres. In turn, the nucleus tractus solitarius will inhibit the cardiac-inhibitory center and stimulate the cardiac-acceleratory centre. As the results, heart rate and contractility increases to limit the fall in the cardiac output as the venous pressure drops. There is also marked vasoconstriction in the skeletal muscle, splanchic and renal vascular beds to raise total peripheral resistance. These compensatory adjustments prevent mean arterial blood pressure to heavily fall during postural change, which can affect the cerebral circulation. In fact, during postural change the systolic blood pressure increases slightly, diastolic blood pressure rises sharply and mean-arterial blood pressure falls only transiently [12].

Regarding orthostatic hypertension, two mechanisms are proposed to explain it. The first is vascular adrenergic hypersensitivity theory. Second is nephroptosis theory. Vascular adrenergic hypertension is related to excessive venous pooling, with an initial drop in cardiac output followed by overcompensation with an excessive release of catecholamine. Nephroptosis is related with activation of the renin-angiotensin-aldosterone system as the perfusion pressure to the kidney drops during postural changes [13-15].

There are strong evidences that showed autonomic nervous system is involved in the heightened response of cardiovascular system to ether laboratory or real life stressors [4,16,17]. Normotensive subjects with a family history of hypertension is a prehypertension model that portrays a significant exaggerated sympathetic activity [3,18]. Rathi, et al. [19] reported that young male normotensive subjects of hypertensive parents shows hyperactive sympathetic nervous system, as assessed by diastolic blood pressure response to cold pressor test and handgrip exercise, as compared to young male normotensive subjects of normotensive parents. In their study, the activity parasympathetic nervous system does not differ significantly between young male normotensive subjects of normotensive and hypertensive parents. In line with it, Jenie and Noor [7] reported that normotensive young adults with a family history of hypertension have exaggerated cardiovascular reactivity to physical test (cold pressor test), but not to mental test (an arithmetic test), as compared to normotensive young adults without a family history of hypertension. It is known that norepinephrine is the main neurotransmitter involved in the cold pressor test, whereas epinephrine is dominant humoral response during mental test [7]. Rafidhah., et al. [20] also showed that normoten-
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Normotensive subjects with a family history of hypertension demonstrate significantly higher norepinephrine level as compared to normotensive subjects without a family history of hypertension. Therefore, these lines of evidences support vascular adrenergic hypersensitivity theory, rather than nephroptosis theory, in explaining normotensive subjects with a family history of hypertension as the prehypertension model. Furthermore, these lines of evidences suggest that the activation of sympathetic nerves, rather than the activation of sympatho-adrenal system, is occurred in normotensive subjects with a family history of hypertension.

Conclusion

Normotensive subjects with a family history of hypertension show more exaggerated systolic blood pressure and diastolic blood pressure to postural changes than those without a family history of hypertension. This finding supports vascular adrenergic hypersensitivity theory.

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Data Availability

None.

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Bibliography
