

Original articles

**RELATIONSHIP BETWEEN ORTHOSTATIC BLOOD PRESSURE CHANGES
AND POSTURAL SWAY DURING STANDING UP FROM A CHAIR IN OLDER
ADULT FEMALES**

Short running title: Change of blood pressure and postural sway

Jun Murata¹, Shin Murata², Jun Horie², Hiroshi Ohtao³, Junya Miyazaki⁴

1) Department of Physical and Occupational Therapy, Graduate School of Biomedical Sciences,
Nagasaki University

2) Department of Physical Therapy, Faculty of Rehabilitation Science, Nishikyushu
University

3) Department of Physical Therapy, Faculty of Health and Welfare, Prefectural
University of Hiroshima

4) Department of Physical Therapy, Faculty of Health Sciences, Mejiro University

***Correspondence to:** Dr. Jun Murata, Department of Physical and Occupational Therapy,

Graduate School of Biomedical Sciences, Nagasaki University

1-7-1 Sakamoto, Nagasaki 852-8520, Japan.

E-mail : jmura@nagasaki-u.ac.jp

Tel: +81-95-819-7923 / Fax: +81-95-819-7907

Disclosure of Conflicts of Interest

No potential conflicts of interest were disclosed.

ABSTRACT

Background: Orthostatic reductions in blood pressure upon standing are common among the elderly. This orthostatic blood pressure changes may relate to the augmentation of postural sway and be an important risk factor for falls. Thus, to clarify whether orthostatic blood pressure change on standing up from a chair is relevant to postural sway, we simultaneously measured changes in blood pressure and the movement of a weighted center upon standing.

Methods: A total of 63 older adult females were investigated. Blood pressure (systolic blood pressure: SBP, diastolic blood pressure: DBP) measured in a sitting position were defined as the baseline levels. The movement of center of pressure (COP) was measured using a stable force platform to quantify postural stability. Subjects were instructed to stand up from a chair on the platform and maintain an upright position with their eyes open for 40s. Upon standing, blood pressure and the movement of COP were recorded. Pearson's correlation was performed to determine relationships between the changes in BP and the movement of COP (distance of the movement of COP: LNG, envelopment area traced by the movement of COP: AREA).

Results: SBP was reduced while maintaining an upright position for 40s (-5.0 ± 8.6 mmHg), but not DBP (0.6 ± 4.3 mmHg). Moreover, the change in SBP showed a negative

relationship with LNG ($r=-0.43$, $P<0.01$) and AREA ($r=-0.31$, $P<0.05$).

Conclusions: These results suggested that postural change influenced SBP and that the drop of SBP was associated with augmentation of postural instability in older adult females.

Key words: Orthostatic stress, blood pressure, Balance, Posture, Elderly

Introduction

Standing results in the displacement of 300-1000 ml of blood from the central body compartments to the lower parts of the body, which in turn causes a reduction in venous return to the heart, resulting in decreased cardiac output and a subsequent lowering of blood pressure (BP)¹⁻³. The phenomenon, called orthostatic hypotension (OH), is often associated with the following symptoms: dizziness, fatigue, light-headedness, and/or syncope⁴⁻⁶. OH (defined as a decrease in systolic BP (SBP) of ≥ 20 mmHg and/or decrease in diastolic BP (DBP) of ≥ 10 mmHg within 3 min of standing⁷⁻⁸) is a common condition with a prevalence as high as 30% in elderly people⁹⁻¹³. Moreover, OH is considered an important risk factor for falls¹⁴⁻¹⁷, which are one of the principal causes of reduced activities of daily living and a lower quality of life in the elderly.

On the other hand, it is known that elderly people display increased postural swaying during movement of the center of pressure (COP)¹⁸⁻²⁰. This age-related augmentation of postural instability may be affected by orthostatic BP changes and increase the risk of falls¹⁴⁻¹⁷. However, the relationship between the change in BP and the increase in postural sway upon standing has not been thoroughly investigated. Therefore, this study aimed to clarify whether the orthostatic BP change upon standing up from a

chair is relevant to postural instability in older adult females.

Materials and Methods

Subjects

A total of 63 older adult females (mean age: 71.4 ± 6.5 years) were investigated in this study. All subjects professed to be in good health on a standard medical examination questionnaire and lived independently in the community. The characteristics of the study population including their anthropometric data are shown in Table 1. None of the subjects had diseases that are known to influence the neuromuscular function of the lower extremities, such as lumbar spondylosis-related lumbar radiculopathy or a cerebrovascular-related accident. However, three subjects had diet-controlled diabetes. Another twenty-seven subjects were on regular medication for cardiovascular problems (heart disease, $N=9$; high blood pressure, $N=27$). Thirty-three elderly individuals did not take regular medication. Moreover, prior to the study, we administered the Mini-Mental State Examination, and all subjects passed (threshold for a passing score = 25).

Each subject was informed in advance of the purpose of the study and of the procedures involved, and their consent was obtained. This study was performed in accordance with the Declaration of Helsinki and approved by the Institutional Ethical

Committee of Nagasaki University.

Measurements

SBP, DBP, and pulse rate (PR) were measured during sitting and standing with an automated blood pressure monitor (HEM770A, Omron Co., Tokyo, Japan). SBP, DBP, and PR were measured for approximately 30 s. A brachial cuff was placed around the left arm, which was kept in a fixed position at heart level with a sling. The movement of the center of pressure (COP) was measured using a stable force platform (GS-30, Anima Co., Tokyo, Japan) to quantify postural stability. The equipment consisted of a triangular steel platform connected to a feedback unit. The machine was designed to assess the movement of the COP using three verticality load sensors placed on the corners of an isosceles triangle in the horizontal plane with the center of gravity of the subject located within the center of the triangle. Distance of the movement of the COP (LNG) and the area traced by the movement of the COP (AREA) were measured for 30 s and are represented both numerically and graphically.

Procedure

All experiments were performed in a quiet room, which was controlled at an

ambient temperature of 22-25°C, between 10:00 a.m. and 12:00 noon. After the instruments had been prepared, each subject was allowed to sit on a chair for more than 10 min in order to allow their cardiovascular variables to stabilize. After resting, the first measurements of SBP, DBP, and PR were performed in a sitting position. The values of SBP, DBP, and PR measured in a sitting position were defined as the baseline levels. The subjects were instructed to stand up from a chair on a platform without their shoes and to maintain an upright position for 40 s with their eyes open, their right arm at their side, and their feet close together. The examination was performed with the subjects' eyes focused on a round red dot (3 cm in diameter) located on a white wall, which was set at a distance of 2 m away from the subject. The changes in SBP, DBP, PR, and COP (LNG and AREA) were simultaneously measured for 30 s from 2 s after the onset of standing (shown in Fig. 1).

Statistics

The values of SBP, DBP, and PR during standing were statistically compared with the baseline levels using the paired-*t* test. Correlations between the changes in BP (Δ SBP and Δ DBP) and PR from the baseline values and the movement of the COP (LNG and AREA) were performed using the Pearson's correlation coefficient. Furthermore, the

subjects were divided into two groups (hypertensive medication users and non-users), and comparisons of the anthropometric data and the changes in SBP, DBP, PR, LNG, and AREA that occurred in response to orthostatic challenge between the two groups were performed using the unpaired-*t* test. Moreover, comparisons of categorical variables were carried out using the chi-square test (or Fisher's exact test). The level of statistical significance was defined as $P < 0.05$. The data are expressed as the mean \pm standard deviation.

Results

Cardiovascular responses to orthostatic challenge

The baseline values of SBP, DBP, and PR during sitting on a chair were 149.5 ± 20.6 mmHg, 87.5 ± 10.1 mmHg, and 71.7 ± 11.7 beats/min, respectively. On the other hand, SBP was reduced by -5.0 ± 8.6 mmHg ($P < 0.05$) while the subjects maintained an upright position for 40s, whereas DBP did not change (0.6 ± 4.3 mmHg) (Fig. 2A, 2B). PR increased by 5.0 ± 3.9 beats/min during standing (Fig. 2C). The prevalence of OH in these community-dwelling older adult females (N=63) was 13%.

Relationships between cardiovascular responses and the movement of the

COP at the onset of rising from a chair

The mean values of LNG and AREA were 48.5 ± 13.0 cm and 3.0 ± 1.3 cm², respectively. Moreover, the change in SBP showed negative relationships with LNG ($r=-0.43$, $P<0.01$) and AREA ($r=-0.31$, $P<0.05$) (Fig. 3). However, there was no relationship between the change in DBP and LNG ($r=-0.07$, $P=0.96$) or AREA ($r=0.13$, $P=0.31$). Similarly, the change in PR did not show a significant association with LNG ($r=0.17$, $P=0.19$) or AREA ($r=0.21$, $P=0.11$).

The differences in cardiovascular responses to and postural sway after orthostatic challenge in the subjects with or without hypertensive medication

The characteristics of the hypertensive medication users and non-users are summarized in Table 1. There were no differences in the anthropometric data between the two groups. On the other hand, many of the hypertensive medication users had cardiovascular problems (hypertension, $N=27$, 100%, $P < 0.001$; heart disease, $N=9$, 33%, $P < 0.001$).

The changes in SBP, DBP, PR, LNG, and AREA in the hypertensive medication users ($N=27$) at the onset of rising from a chair were -6.7 ± 7.9 mmHg, 0.2 ± 3.0 mmHg, 4.3 ± 3.3 beats/min, 46.6 ± 11.8 cm, and 3.2 ± 1.3 cm², respectively. The responses to orthostatic challenge of the hypertensive medication users did not differ from the

responses of the non-users (N=36, SBP; -4.5 ± 9.1 mmHg, DBP; 0.9 ± 5.0 mmHg, PR; 5.6 ± 4.4 beats/min, LNG; 49.9 ± 13.8 cm, AREA; 2.9 ± 1.3 cm²) (Table 2).

Discussion

The purpose of this study was to clarify whether the orthostatic BP change that occurs during standing up from a chair is relevant to postural instability in older adult females. Our major findings were that SBP was reduced while the subjects maintained an upright position and that a negative correlation was observed between the change in SBP and the movement of the COP. These results suggested that the orthostatic reduction in SBP evoked by standing was associated with the augmentation of postural sway in older adult females.

OH was detected in 13% elderly participants (8 of the 63 participants) in this study. This incidence rate agreed with those found in previous studies, which reported that OH occurred in 6-30 % of elderly people⁹⁻¹³. Neural dysregulation of BP strongly affects the incidence of OH. In patients with autonomic dysfunction, OH results from an impaired capacity to increased vascular resistance during standing¹. Neural dysfunction leads to augmented downward pooling of venous blood and a consequent reduction in stroke volume and cardiac output that exaggerates the orthostatic fall in BP¹. In addition,

multiple factors have been linked to the incidence of OH including age, bed rest, low body mass index, medication, and the timing of food intake^{14,21}. The changes in BP that occur after postural challenge are probably modulated by the interaction of these factors in elderly people.

On the other hand, the present results indicated an orthostatic fall in SBP, but not DBP, at the onset of standing up from a chair. Since elderly subjects show increased sympathetic and decreased parasympathetic activity as well as impaired arterial baroreflex function²²⁻²⁶, elderly subjects maintain their blood pressure in the upright position essentially through increased peripheral resistance, whereas they increase their heart rate less than the young²⁷⁻²⁸. Therefore, the orthostatic decline in SBP might be more prominent than that in DBP.

OH is known to be one of the underlying causes of falls¹⁴⁻¹⁷. Heitterachi et al.¹⁷ reported that elderly fallers (subjects who had experience of falling during a 12 month follow-up survey) displayed significantly greater decreases in systolic blood pressure when they tilted their head up by 60° than non-fallers. Other studies have also indicated that OH is prevalent in older people who fall frequently¹⁴⁻¹⁶. In the present study, the orthostatic change in SBP was associated with an increase in the movement of the COP. Falls in the elderly may be affected by postural instability due to a drop in SBP at the

onset of rising from a chair. Several studies, using transcranial Doppler sonography or near infrared spectroscopy, have shown cerebral vasoconstriction and lower cerebral perfusion during orthostatic stress in healthy adult subjects²⁹⁻³². Cerebral hypoperfusion due to an orthostatic decline in SBP may influence postural control, thereby contributing to the augmentation of postural instability.

Changes in the motor and sensory functions are also considered to be responsible for the augmentation of postural instability³²⁻³³. There is some evidence that reduced lower extremity strength is associated with poor balance and a greater risk of falls³⁴⁻³⁵. Furthermore, the sensory inputs from the visual, vestibular, proprioceptor, and somatosensory systems constitute the primary elements necessary for the central nervous system to process postural control information. Aging also affects the sensory systems, resulting in increased postural instability and falls³⁶⁻³⁹. In the present study, a moderate correlation between the change in SBP and the movement of the COP was observed. These results suggest that postural instability at the onset of rising from a chair is influenced by not only the orthostatic change in SBP but also other factors including changes in motor and sensory functions. Further studies are required to investigate the effects of other factors and to verify the major determinant of postural sway upon standing.

In this study, the twenty-seven subjects were on regular medication, and most of the subjects were taking medication for hypertension. Several studies have suggested that the use of anti-hypertensive medication triggers falls in hypertensive subjects⁴⁰⁻⁴¹. Blood volume depletion due to diuretics or blood pressure fluctuations caused by anti-hypertensive drugs may augment orthostatic BP changes. However, the changes in BP and the COP at the onset of rising from a chair did not differ between anti-hypertensive medication users and non-users in this study. Furthermore, some previous cross-sectional studies have found no association between anti-hypertensive therapy and postural changes in SBP⁴²⁻⁴³. Considered together, these studies suggest that the use of anti-hypertensive medication has little influence on orthostatic SBP control and postural instability, at least immediately after rising. The effects of the other medications on orthostatic BP control and postural instability are unclear but warrants examination.

One limitation of our study is that we only examined female subjects, since few male subjects were enrolled. Previous study has reported that females display a lower tolerance to orthostatic challenge than males⁴⁴. This gender effect may also influence the relationship between orthostatic BP changes and postural sway. Moreover, this study was too small to detect any differences in the relationships between the type of anti-hypertensive medications used and orthostatic BP control or postural instability.

Further studies are necessary to clarify these points.

In conclusion, the orthostatic change in SBP evoked immediately after rising from a chair is related to postural instability in older adult females, which in turn may increase the risk of falls.

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References

1. Smit AA, Halliwill JR, Low PA, Wieling W. Pathophysiological basis of orthostatic hypotension in autonomic failure. *J Physiol* 1999; 519: 1-10.
2. Mathias CJ. To stand on one's own legs. *Clin Med* 2002; 2: 237-45.
3. van Lieshout JJ, Wieling W, Karemaker JM, Secher NH. Syncope, cerebral perfusion, and oxygenation. *J Appl Physiol* 2003; 94: 833-48.
4. Mathias CJ, Kimber JR. Postural hypotension: causes, clinical features, investigation, and management. *Annu Rev Med* 1999; 50:317-36.
5. Gupta V, Lipsitz LA. Orthostatic hypotension in the elderly: diagnosis and treatment. *Am J Med* 2007;120:841-7.
6. Maule S, Papotti G, Naso D, Magnino C, Testa E, Veglio F. Orthostatic hypotension: evaluation and treatment. *Cardiovasc Hematol Disord Drug Targets* 2007; 7: 63-70.
7. The Consensus Committee of the American Autonomic Society and the American Academy of Neurology. Consensus statement on the definition of orthostatic hypotension, pure autonomic failure, and multiple system atrophy. *Neurology* 1996; 46:1470.
8. Lahrman H, Cortelli P, Hilz M, Mathias CJ, Struhal W, Tassinari M. EFNS guidelines on the diagnosis and management of orthostatic hypotension. *Eur J Neurol* 2006; 13: 930-6.

9. Johnson RH, Smith AC. Effect of posture on blood-pressure in elderly patients. *Lancet* 1965; 731-3.
10. Lipsitz LA, Storch HA, Minaker KL, Rowe JW. Intra-individual variability in postural blood pressure in the elderly. *Clin Sci* 1985; 69: 337-41.
11. Mader SL, Josephson KR, Rubenstein LZ. Low prevalence of postural hypotension among community-dwelling elderly. *JAMA* 1987; 258: 1511-4.
12. Low P. The effect of aging on the autonomic nervous system. In: Low P editor. *Clinical Autonomic Disorders*. Philadelphia: Lippincott-Raven; 1997; p. 161-78.
13. Luukinen H, Koski K, Laippala P, Kivela SL. Prognosis of diastolic and systolic orthostatic hypotension in older persons. *Arch Intern Med* 1999; 159:273–80.
14. Rutan GH, Hermanson B, Bild DE, Kittner SJ, LaBaw F, Tell GS. Orthostatic hypotension in older adults. The Cardiovascular Health Study. CHS Collaborative Research Group. *Hypertension*. 1992; 19: 508-19.
15. Ooi WL, Hossain M, Lipsitz LA. The association between orthostatic hypotension and recurrent falls in nursing home residents. *Am J Med*. 2000; 108: 106-11.
16. Kario K, Tobin JN, Wolfson LI, Whipple R, Derby CA, Singh D, Marantz PR, Wassertheil-Smoller S. Lower standing systolic blood pressure as a predictor of falls in the elderly: a community-based prospective study. *J Am Coll Cardiol* 2001; 38: 246-52.

17. Heitterachi E, Lord SR, Meyerkort P, McCloskey I, Fitzpatrick R. Blood pressure changes on upright tilting predict falls in older people. *Age Ageing* 2002; 31: 181-6.
18. Era P, Heikkinen E. Postural sway during standing and unexpected disturbance of balance in random samples of men of different ages. *J Gerontol* 1985; 40: 287-95.
19. Ring C, Nayak US, Isaacs B. The effect of visual deprivation and proprioceptive change on postural sway in healthy adults. *J Am Geriatr Soc* 1989; 37: 745-9.
20. Masui T, Hasegawa Y, Matsuyama Y, Sakano S, Kawasaki M, Suzuki S. Gender differences in platform measures of balance in rural community-dwelling elders. *Arch Gerontol Geriatr* 2005; 41: 201-9.
21. Hajjar I. Postural blood pressure changes and orthostatic hypotension in the elderly patient: impact of antihypertensive medications. *Drugs Aging* 2005; 22:55-68.
22. Shimada K, Kitazumi T, Sadakane N, Ogura H, Ozawa T. Age-related changes of baroreflex function, plasma norepinephrine, and blood pressure. *Hypertension* 1985; 7: 113-7.
23. Byrne EA, Fleg JL, Vaitkevicius PV, Wright J, Porges SW. Role of aerobic capacity and body mass index in the age-associated decline in heart rate variability. *J Appl Physiol* 1996; 81: 743-50.
24. Laitinen T, Hartikainen J, Vanninen E, Niskanen L, Geelen G, Länsimies E. Age and

gender dependency of baroreflex sensitivity in healthy subjects. *J Appl Physiol* 1998; 84: 576-83.

25. James MA, Potter JF. Orthostatic blood pressure changes and arterial baroreflex sensitivity in elderly subjects. *Age Aing* 1999; 28: 522-30.

26. Hotta H, Uchida S. Aging of the autonomic nervous system and possible improvements in autonomic activity using somatic afferent stimulation. *Geriatr Gerontol Int* 2010; 10: S127-36.

27. Smith JJ, Hughes CV, Ptacin MJ, Barney JA, Tristani FE, Ebert TJ. The effect of age on hemodynamic response to graded postural stress in normal men. *J Gerontol* 1987; 42: 406-11.

28. Shannon RP, Maher KA, Santinga JT, Royal HD, Wei JY. Comparison of differences in the hemodynamic response to passive postural stress in healthy subjects greater than 70 years and less than 30 years of age. *Am J Cardiol* 1991; 67: 1110-6.

29. Grubb BP, Gerard G, Roush K, Temesey-Armos P, Montford P, Elliott L, Hahn H, Brewster P. Cerebral vasoconstriction during head-upright tilt-induced vasovagal syncope: a paradoxical and unexpected response. *Circulation* 1991; 84: 1157-64.

30. Levine BD, Giller CA, Lane LD, Buckey JC, Blomqvist CG. Cerebral versus systemic hemodynamics during graded orthostatic stress in humans. *Circulation* 1994; 90:

298–306.

31. Mehagnoul-Schipper, DJ, Vloet LC, Colier WNJM, Hoefnagels WHL, Jansen RWMM Cerebral oxygenation declines in healthy elderly subjects in response to assuming the upright position. *Stroke* 2000; 31: 1615-20.

32. Shumway-Cook A, Woollacot MH. *Motor control: theory and practical applications*. 2nd edition. Baltimore, MD: Lippincott Williams & Wilkins; 2001; p. 172–6.

33. Horvat M, Ray C, Ramsey V, Miszko T, Keeney R, Blasch B. Compensatory analysis and strategies for balance in individuals with visual impairments. *J Vis Impair Blind* 2003; 97:695–703

34. Maki PJ, Holliday AK, Topper M. A prospective study of postural balance and risk of falling in an ambulatory and independent elderly population. *J Gerontol* 1994; 49:72–84.

35. Perry MC, Carville SF, Smith CH, Rutherford OM, Newman DJ. Strength, power output and symmetry of leg muscles: effect of age and history of falling. *Eur J Appl Physiol* 2007; 100: 553–61.

36. Anacker SL, Di Fabio RP. Influence of sensory inputs on standing balance in community-dwelling elders with a recent history of falling. *Phys Ther* 1992; 72: 575-81.

37. Brooke-Wavell K, Perrett LK, Howarth PA, Haslam RA. Influence of the visual environment on the postural stability in healthy older women. *Gerontology* 2002;

48:293–7.

38. Kulmala J, Viljanen A, Sipilä S, Pajala S, Pärssinen O, Kauppinen M, Koskenvuo M, Kaprio J, Rantanen T. Poor vision accompanied with other sensory impairments as a predictor of falls in older women. *Age Ageing* 2009; 38: 162-7.

39. Ricci NA, de Faria Figueiredo Gonçalves D, Coimbra AM, Coimbra IB. Sensory interaction on static balance: a comparison concerning the history of falls of community-dwelling elderly. *Geriatr Gerontol Int* 2009; 9: 165-71.

40. Lipsitz LA. Abnormalities in blood pressure homeostasis that contribute to falls in the elderly. *Clin Geriatr Med* 1985; 1: 637-48.

41. King MB, Tinetti ME. Falls in community-dwelling older persons. *J Am Geriatr Soc* 1995; 43: 1146-54.

42. Applegate WB, Davis BR, Black HR, Smith WM, Miller ST, Burlando AJ.

Prevalence of postural hypotension at baseline in the Systolic Hypertension in the Elderly Program (SHEP) cohort. *J Am Geriatr Soc* 1991; 39:1057-64.

43. Burke V, Beilin LJ, German R, Grosskopf S, Ritchie J, Puddey IB, Rogers P. Postural fall in blood pressure in the elderly in relation to drug treatment and other lifestyle factors. *Q J Med* 1992; 84:583-91.

44. Fu Q, Arbab-Zadeh A, Perhonen MA, Zhang R, Zuckerman JH, Levine BD.

Hemodynamics of orthostatic intolerance: implications for gender differences. *Am J*

Physiol Heart Circ Physiol 2004; 286:H449-57.

Table 1. Characteristics of the hypertensive medication users and non-users

	Hypertensive medication users (n=27)	Non-users (n=36)	Difference between users and non-users (P value)
Age (years)	71.5 ± 4.9	71.4 ± 7.6	0.939
Height (cm)	147.9 ± 5.3	148.5 ± 7.5	0.741
Weight (kg)	49.2 ± 5.3	48.1 ± 7.6	0.527
Body mass index	22.5 ± 2.0	22.0 ± 3.2	0.537
Medical conditions (number)			
Hypertension	27	0	< 0.001
Heart disease	9	0	< 0.001
Hyperlipidemia	2	1	0.572
Diabetes	1	2	> 0.999

Mean ± standard deviation.

Table 2. Comparison of postural sway and the cardiovascular responses to orthostatic challenge between the hypertensive medication users and non-users

	Hypertensive medication users (n=27)		Non-users (n=36)		Difference between users and non-users (<i>P</i> -value)
	sitting	standing	sitting	standing	
SBP (mmHg)	153.5±19.2	146.9±18.4	146.7±21.4	142.2±24.1	0.337
DBP (mmHg)	87.6±7.1	87.8±6.8	87.4±11.8	88.3±13.2	0.548
PR (beats/min)	66.3±7.1	70.5±8.0	76.2±12.8	81.8±13.8	0.218
LNG (cm)		46.6±11.8		49.9±13.8	0.321
AREA (cm ²)		3.2±1.3		2.9±1.3	0.409

Mean ± standard deviation.

Figures

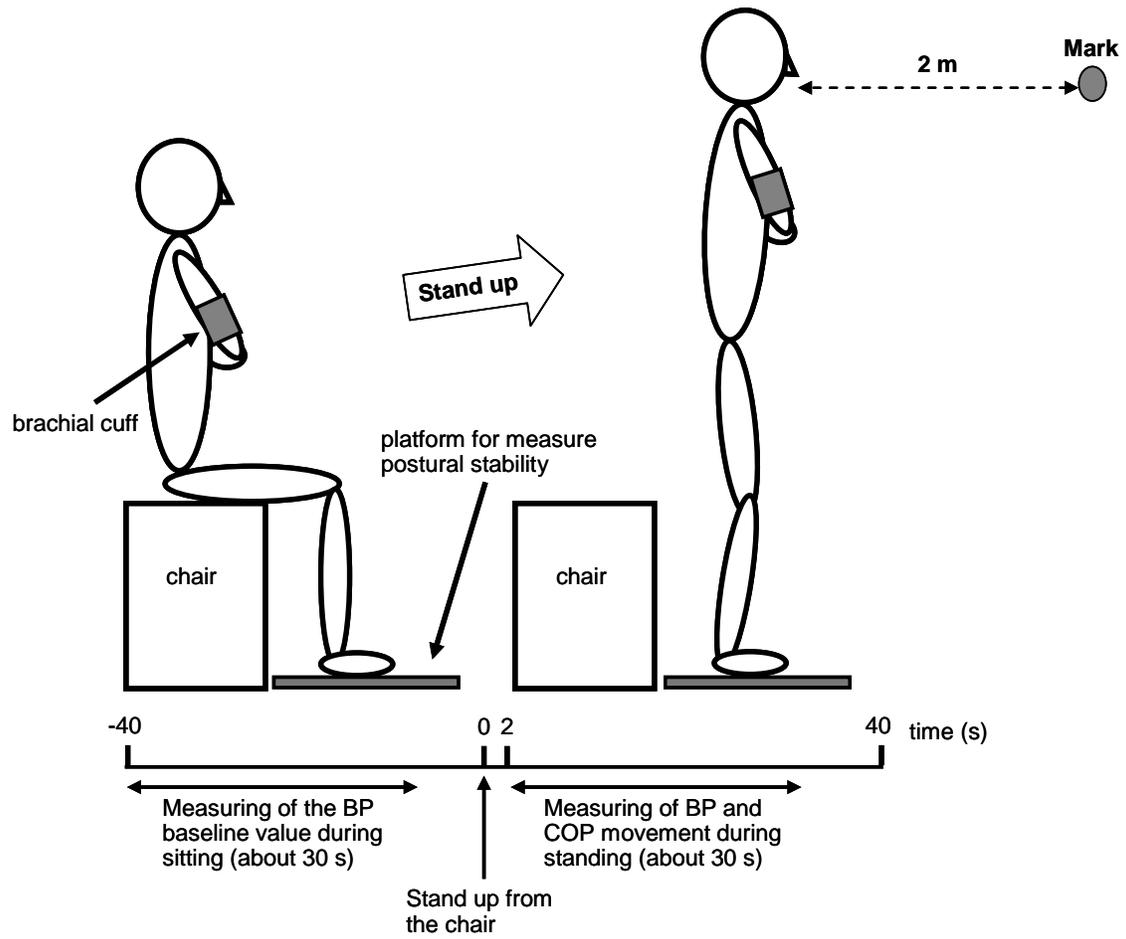


Figure 1. Experimental protocol.

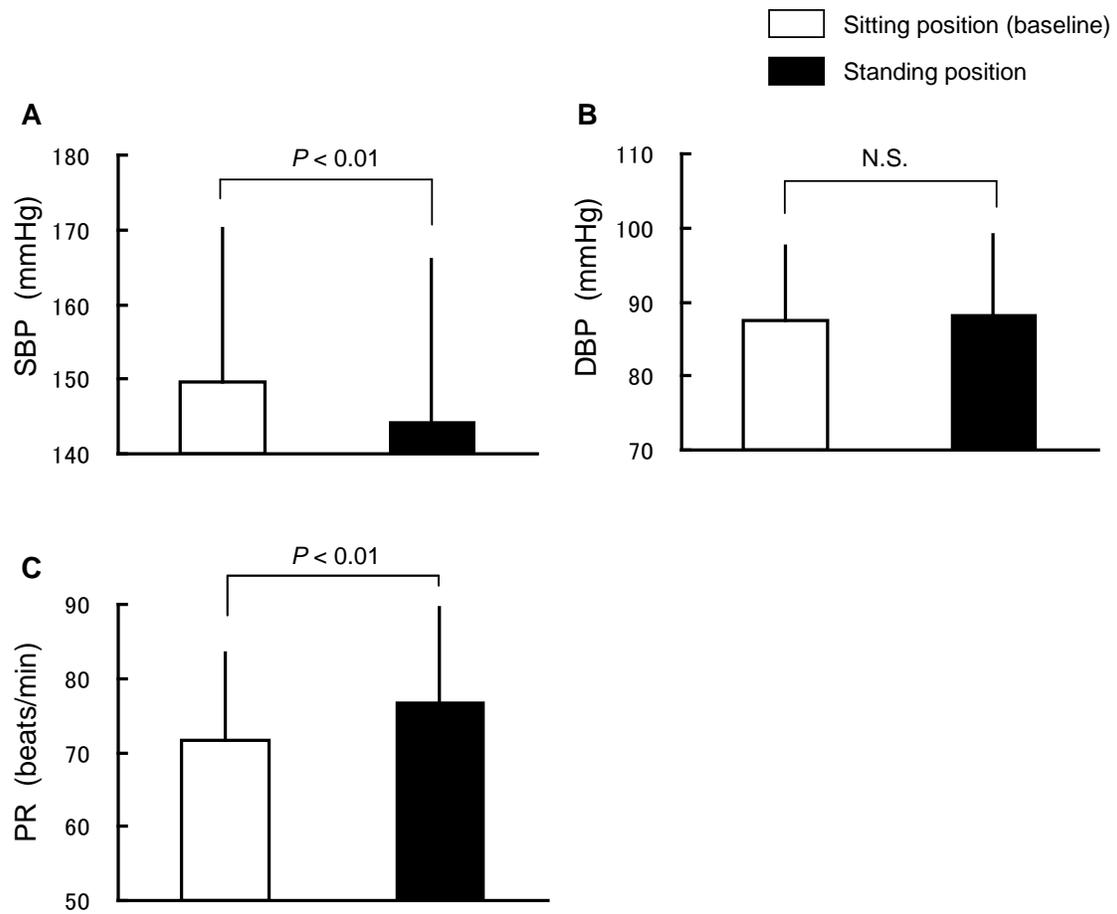


Figure 2. Responses of SBP (A), DBP (B), and PR (C) to orthostatic challenge.

Each parameter was compared with the baseline value obtained during sitting (open bar) and the value produced after orthostatic challenge (closed bar).

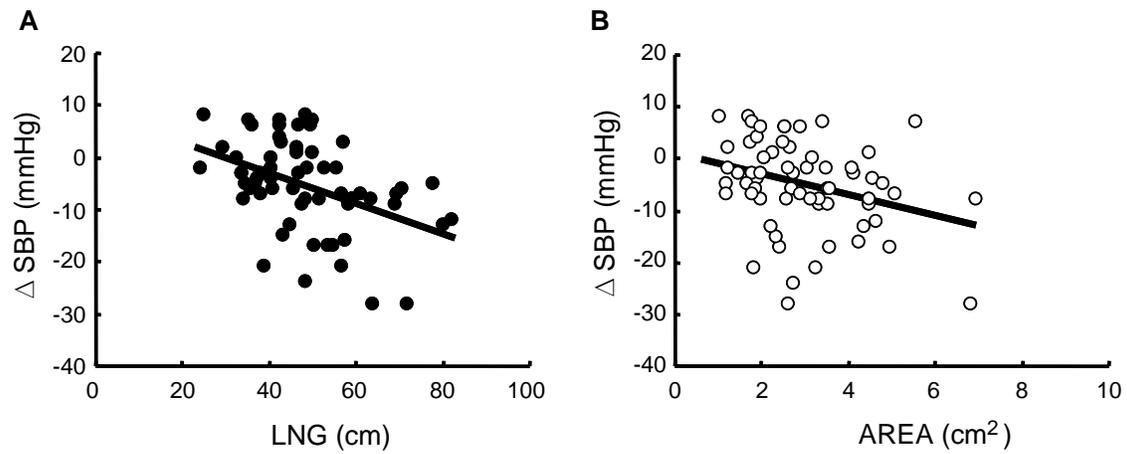


Figure 3. Associations between the orthostatic SBP change and LNG (A) or AREA (B). The linear regression lines shown in A and B are represented by $y = -0.29x + 8.50$ ($r = 0.43$, $P = 0.01$) and $y = -1.99x + 0.57$ ($r = 0.31$, $P < 0.05$), respectively.