Original Article

Hypotension Associated with Prone Body Position: A Possible Overlooked Postural Hypotension

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Conditions related to the dysregulation of blood pressure (BP), such as orthostatic hypotension, have been shown to be significantly associated with cardiovascular disease. Recently, the prone body position has been recognized as a possible postural factor leading to BP dysregulation. We conducted a cross-sectional study to investigate the BP response to a change in body position from supine to prone. The study subjects consisted of 271 middle-aged healthy males, randomly selected from the employees of a large manufacturing enterprise in Ehime Prefecture, Japan. Brachial BP and heart rate were measured in a sitting, supine and prone position, in that order, and each difference was defined as a postural change. The postural changes in aortic hemodynamics were also assessed using a SphygmoCor system. The basal BP measured in the sitting position was significantly decreased in the supine position (132±18 to 130±17 mmHg, $p<0.001$). A further reduction was observed after the postural change from supine to prone (130±17 to 125±16 mmHg, $p<0.001$). The heart rate was increased with the supine-to-prone postural change (4.1±5.8 beats/min, $p<0.001$), while it showed a significant decrease with the sitting-to-supine postural change (−7.6±5.6 beats/min, $p<0.05$). The impact of BP reduction was more prominent in the aortic artery (−3.3±6.7%) than the brachial artery (−3.0±6.3%, $p=0.020$). Multiple regression analysis showed that basal systolic BP was a solely significant determinant of the prone-hypotension ($\beta=-0.309$, $p<0.001$). In conclusion, these results indicate that lying in a prone posture could lead to unregulated postural hypotension, which has the possibility of being a novel predictor of cardiovascular frailty. (*Hypertens Res* 2005; 28: 741–746)

***Key Words*: prone position, postural hypotension, cardiovascular disease, risk factor**

Introduction

The response of blood pressure (BP) to postural change has been shown to be associated with cardiovascular diseases in the Atherosclerosis Risk in Communities (ARIC) study (*1*). Several studies have suggested that orthostatic hypotension could be a potent predictor for stroke, cardiovascular disease, cognitive dysfunction, and even death (*2*–*5*). Recently, Kario *et al*. (*6*) reported a significant association between orthostatic BP variation and abnormal diurnal BP variation in elderly hypertensive subjects. Since the prognostic significance of abnormal nighttime BP, such as nondipping status with diminished nocturnal BP fall, has also been well established (*7*), BP variation could be a useful phenotype to detect impairment of the cardiovascular system as well as physical frailty.

As another postural factor leading to mortality, previous studies have drawn attention to the sleeping position as a risk related to sudden infant death syndrome (*8*, *9*). Although various mechanisms have been postulated to explain the association between prone sleeping position and sudden death, a

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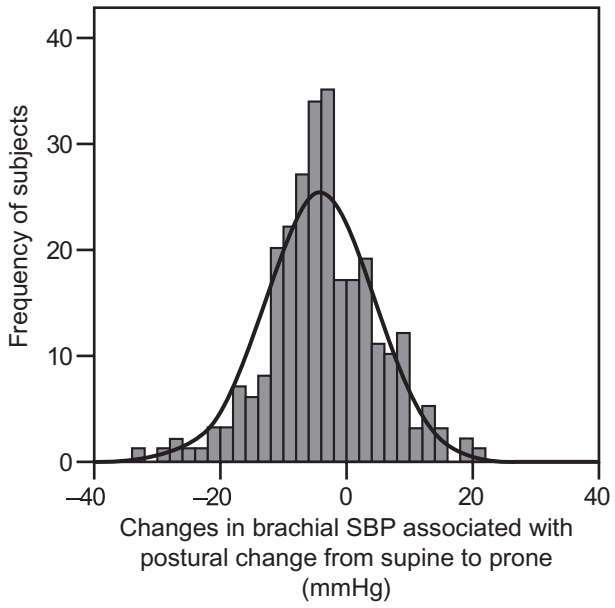

Fig. 1. *Frequency histogram of the postural changes in brachial systolic BP (SBP). The solid line indicates the normal distribution curve. The distribution curve had a symmetric shape with a pivot at –4.2 mmHg.*

reduction of vasomotor tone accompanied with hypotension and tachycardia have been considered the underlying mechanisms (*10*). Similar observations have also been reported in young adult subjects (*11*); the prone position reduced the stroke volume associated with reflex activation of the sympathetic nervous system, possibly due to an impediment to the arterial filling caused by the mechanical compression of the thorax. Furthermore, it has also been demonstrated that changing the posture from supine to prone causes a significant decrease in cardiac index under anesthesia (*12*, *13*).

Considering the results of these previous studies, we hypothesized that BP could be decreased in subjects in a prone body position, and that such a posture-related BP decrease could be a novel marker of BP dysregulation to be used in conjunction with orthostatic BP variation and diurnal BP variation. In the present cross-sectional study, we investigated the BP response to a change in body position from supine to prone.

Methods

Study Subjects

The present study is derived from the Shimanami Health Promoting Program (J-SHIPP), which was designed as a longitudinal epidemiological study evaluating factors relating to cardiovascular disease, dementia, and death (*14*, *15*). A random sample of 309 male employees was obtained from a large manufacturing enterprise in Ehime Prefecture, Japan, one of the study cohorts of the J-SHIPP study. From these subjects, 271 males (mean age: 50±11 years old [19 to 64]; body mass index (BMI): 23±3 kg/m^2 [16.1 to 39.2]) free from any history or symptoms of cardiovascular disease, such as stroke, transient ischemic attack, myocardial infarction, angina, congestive heart failure, or peripheral vascular disease, and not under anti-hypertensive treatment were enrolled in this study. Among these subjects, 92 (33.9%) persons were regarded as hypertensive; these individuals had a seated systolic BP (SBP)≥140 mmHg and/or diastolic BP (DBP)≥90 mmHg. Informed consent for the procedure was obtained from each subject. All procedures were approved by the ethical committee of Ehime University School of Medicine.

Hemodynamic Measurements

Brachial BP was measured using an automatic cuff-oscillometric device (HEM-907; OMRON HEALTHCARE Co., Ltd., Kyoto, Japan). The validity and reproducibility of this device have been confirmed in a previous report (*16*). The peripheral pressure waveform was recorded from the radial artery using a SphygmoCor system (AtCor Medical Pty., Ltd., Sydney, Australia). The aortic pressure waveform was derived from a radial waveform by using a previously validated transfer function (*17*, *18*).

To assess the postural changes in aortic hemodynamics, an augmentation index (AIx) of both the radial and aortic pressure waveforms was calculated as an index of wave reflection (*17*). The AIx was calculated as the ratio of the augmented pressure (AP) by the reflection pressure wave to the pulse pressure. Larger Alx values indicated an increased wave reflection from the periphery or an earlier return of the reflected wave as a result of increased arterial stiffness (*19*). At the aortic artery, AP was also calculated as an absolute value of AIx (*20*). Details of the method used to assess the reflection pressure wave have been described elsewhere (*19*).

All measurements were carried out between 9:00 AM and 12:00 AM under a fasting condition. Subjects were not permitted to drink water or smoke for at least 30 min prior to the measurements.

Evaluation of Postural Change in BP

After more than a 5-min rest in a sitting position, brachial BP was measured twice with a 1-min interval. The mean value of these measurements was regarded as the basal BP. Then subjects were asked to lie in a supine position by themselves, and the brachial BP and radial waveform were measured in that posture. All measurements were repeated on the same arm at 1 min after the posture was changed from supine to prone. The difference between the measurements was defined as the postural change.

Table 1. Hemodynamic Responses to Change in Body Position from Sitting to Supine and Supine to Prone

		Sitting position		Supine position		Prone position	Postural changes		
							Sitting to supine		Supine to prone
Brachial	SBP (mmHg)	132±18	#	130±17	§	125±16	−2.3±10	¶	−4.1±8.5
	DBP (mmHg)	83±12	#	76±12	§	75±11	−7.1±6.4	¶	−1.3±5.9
	PP (mmHg)	49±10	#	54±9	§	51±10	4.8±8.7	¶	−2.9±8.0
Radial	AIx (%)			80±19	§	79±19			
Aortic	SBP (mmHg)			119±19	§	115±18			
	PP (mmHg)			42±10	§	39±10			
	AIx (%)			25±13	§	24±13			
	AP (mmHg)			12±8	§	10±7			
Heart rate (beats/min)		71±10	#	63±9	§	67±10	−7.6±5.6	¶	4.1±5.8

Statistical significance of postural changes in hemodynamics was assessed by repeated measure of ANOVA. #: $p<0.05$ between sitting and supine position. §: $p<0.05$ between supine and prone position. ¶: $p<0.05$ between sitting-to-supine and supine-to-prone postural change. SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure; AIx, augmentation index; AP, augmented pressure.

Statistical Change

All values are expressed as the mean±SD unless otherwise specified. The changes in hemodynamics among the sitting, supine and prone posture were assessed by the repeated measure of analysis of variance (ANOVA). A multiple regression analysis was used to determine the factors independently associated with postural BP change. All analyses were performed with a commercially available statistical package (SPSS ver. 12.0; SPSS Inc., Chicago, USA). p values less than 0.05 were defined as statistically significant.

Results

The frequency histogram of the change in brachial systolic BP (ΔbSBP) during the postural change from supine to prone is shown in Fig. 1. The histogram had a Gaussian distribution with a pivot at −4.2 mmHg. Details of the postural changes in hemodynamic variables are summarized in Table 1. The basal BP and heart rate (HR) measured at the sitting position were significantly decreased in the supine position. Further reduction was observed in both SBP and DBP after the subject changed from a supine to a prone body position. During the change from a sitting to a supine body position, the reduction of DBP was more prominent than that of SBP, though a marked reduction of SBP was observed after the change from a supine to a prone position. The HR was decreased during the sitting-to-supine postural change, and increased during the supine-to-prone postural change. The aortic SBP (−4.2±8.3 mmHg) and AP (−1.3±3.4 mmHg) were also significantly decreased in the prone position. The impact of BP reduction was more prominent in the aortic artery (−3.3±6.7%) than the brachial artery (−3.0±6.3%, $p=0.020$). In a separate analysis, no significant differences were observed in the series of hemodynamic changes between hypertensive subjects and normotensive controls (Fig. 2).

To further evaluate the factors affecting prone-hypotension, multiple regression analysis was carried out with the following parameters: age, BMI, bSBP, HR and arterial AIx measured in the supine position. As a result, basal bSBP was detected as the solely significant determinant of ΔbSBP ($\beta=-0.309$, $p<0.001$). Consequently, in the sub-group analysis, a marked decline in bSBP was observed in 74 hypertensive patients (−7.3±10 mmHg) showing a supine SBP≥140 mmHg and/or DBP≥90 mmHg compared with normotensive subjects (−2.9±7.4 mmHg, $p<0.001$). The aortic SBP was also remarkably decreased in hypertensives compared with normotensives (−8.4±9.7 *vs.* −2.7±7.1 mmHg, $p<0.001$). These differences were still significant after adjusting for BMI and HR.

To evaluate the possibility that the impact of BP reduction was biased by abdominal obesity, we also investigated the possible association between the waist–hip ratio and ΔbSBP. However, no relationship was found between ΔbSBP and the waist–hip ratio in the multiple regression analysis ($p=0.494$).

Discussion

In the present study, we evaluated the BP variations during a simple postural change from supine to prone. An excessive reduction of both brachial and central aortic BP was observed in spite of an increased HR.

During the postural change from sitting to supine, a significant reduction of both brachial SBP and DBP was observed. The change was more prominent in DBP, and was accompanied by a reduced HR. The posture-induced stimulation of carotid baroreceptors, due to removing the hydrostatic pressure gradient from the carotid sinus to the heart, led to a decreased DBP through a decrease in HR, as well as peripheral vasodilatation (*21*). The time-dependent BP reduction during the serial BP measurement might affect the hemodynamic change together with the hydrostatic effects. Further

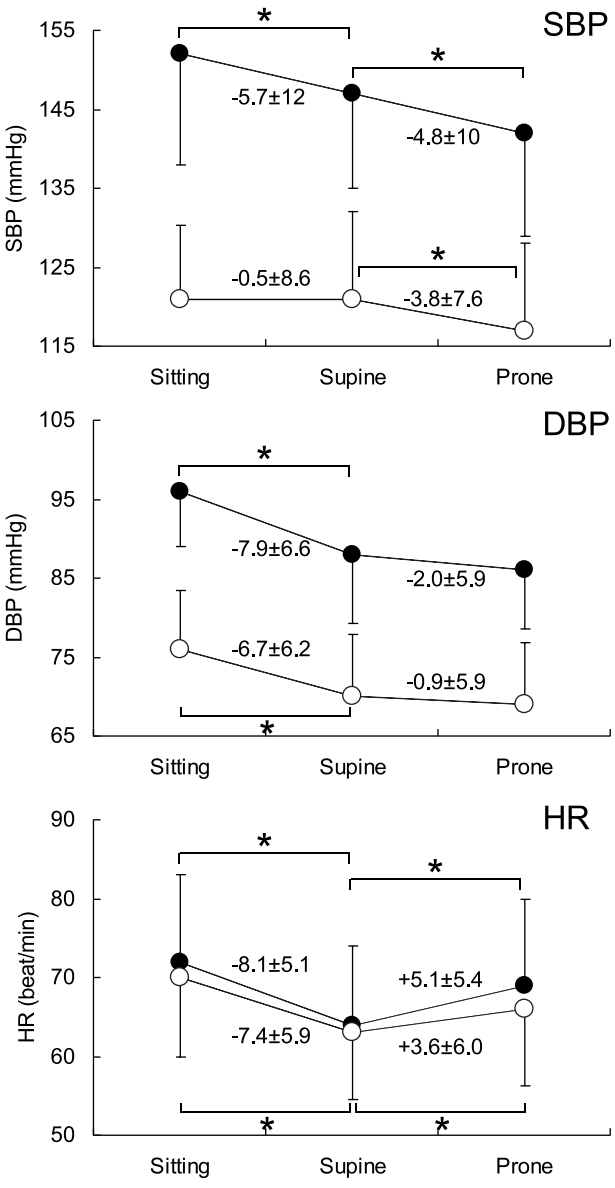

Fig. 2. *Postural changes in hemodynamics by BP levels. Values are the mean±SD. Statistical significance was assessed by repeated measures of ANOVA, and asterisks indicate* $p < 0.05$. *Closed circles, hypertensive subjects; open circles, normotensive subjects.*

reduction of the brachial BP was observed during the postural change from supine to prone. In contrast to the postural change from sitting to supine, the changes were more prominent in SBP, and were accompanied by increased HR. An impediment of arterial filling due to mechanical compression of the thorax and abdomen was thought to be a possible mechanism of the postural BP change (*11*). An insufficient cardiovascular responsiveness to sympathetic stimulation ignited by the impediment arterial filling is thought to be another underlying mechanism of the hemodynamic response during the postural change from supine to prone. In a separate analysis, marked differences were not observed between the hypertensive subjects and normotensive controls. Although it has been demonstrated that hypertension is associated with impaired baroreceptor function, the present hypertensive subjects, free from any end-organ damages or cardiovascular diseases, may be mild enough to show preserved baroreceptor function.

The magnitude of BP reduction was more prominent in the aorta (−3.3±6.7%) than the brachial artery (−3.0±6.3%, $p=0.020$). We have reported a similar phenomenon in orthostatic BP decline (*22*). A postural decline of the reflection component of pulse pressure, represented as a reduced AIx, has been thought to be a principal reason for the larger orthostatic decline in the aortic pressure. In the present study, the radial and aortic AIx, as well as AP, were significantly decreased after the postural change from a supine to a prone position. Thus the alteration of the reflection pressure wave could further precipitate a postural BP decline in the aortic artery. The magnitude of AIx is dependent on the distance from the reflection site, the velocity of wave conduction, as well as the reflection efficacy (reflectance) (*23*). A reduced wave velocity and reflection efficacy, caused by the postural changes in BP and HR, would be the main mechanisms responsible for the postural AIx decline. Since arterial impedance could influence the reflection point, it is also conceivable that the postural change from a supine to a prone position could alter the reflection point through the changes in impedance (*24*).

A fall in systemic BP induces sympathetic stimulation through activation of the baroreceptor reflex. Several lines of evidence have indicated an association between the attenuation of baroreflex sensitivity (BRS) and cardiovascular diseases, such as hypertension, coronary artery disease, and congestive heart failure (*25*). It has also been demonstrated that physiological factors, especially age, have a significant impact on BRS even in healthy subjects. Laitinen *et al*. (*26*) revealed that BRS was inversely related to age ($r=-0.65$) in healthy subjects aged 23 to 77. This observation supports the finding that the prevalence of orthostatic hypotension is more frequent in elderly subjects than young to middle-aged subjects (*5*). However, in the present study, no significant association was observed between age and ΔbSBP. Although it is difficult to explain the reasons for the lack of association, the fact that our study subjects were relatively young is one of the possible explanations.

As a clinical implication of "prone hypotension," we propose that the sleeping posture could be a considerable behavioral source of BP variation, and that it should be considered especially when attempting to make a meaningful assessment of nocturnal BP monitoring. Non-dipping, which is defined as the absence of a normal nocturnal decrease in BP, is known to be one of the potent risk factors for cerebrovascular disease in both hypertensive and normotensive subjects (*27*). A significant association with chronic heart failure and renal impairment has also been demonstrated (*6*, *28*, *29*). Non-dipping

status has been defined as less than a 10% or 10/5 mmHg decline in nocturnal BP compared to daytime BP (*30*, *31*). In the present study, we observed that the mean reduction in SBP in subjects adopting a prone body position was 3.0% (−4.1 mmHg), and 30 subjects (11.1%) showed a more than 10% decline with the change to the prone posture (data not shown). Furthermore, Pump *et al*. (*11*) recently observed a decline in stroke volume and an increase in HR that lasted, along with a slight increase in mean BP, for 6 h after changes in the posture from supine to prone. The inverse change, *i.e.*, a change in the posture from prone to supine, has also been shown to be associated with an increase in mean arterial pressure and cardiac index in subjects under anesthesia (*13*). Although it is well known that the circadian BP rhythm is likely to be a result of the various interactions of neurological and hormonal variations and superimposed exogenous effects, such as physical and mental activities, as well as sleep disturbance (*32*), our findings further suggest that sleeping posture could be a considerable behavioral factor, and that it should be assessed in order to make a meaningful monitoring of ambulatory BP variation.

Several studies have proposed that ambulatory BP variability is an independent determinant of target organ damage (*33*), cardiovascular diseases (*34*), and the presence of silent cerebral white matter lesions (*35*). Although the precise mechanisms of these prognostic associations remain unclear, they might be explained in part by the diminished cardiovascular responsiveness associated with increased stiffness and decreased compliance of the large elastic arteries caused by aging and hypertension (*36*). On the other hand, it has also been reported that several specific components of ambulatory BP variability, such as orthostatic hypotension (*2*, *3*), nondipping status in nocturnal BP monitoring (*29*), and enhanced morning serge (*37*), could be potent risk factors for cardiovascular diseases. Since abnormal diurnal BP variability and other shorter BP variability are associated with each other (*38*), prone-hypotension could be a novel risk factor for cardiovascular frailty as one of the specific components of excessive BP instability.

There are several limitations in the present study. We could not eliminate the possible influences of the time-dependent BP reduction during the serial BP measurement, because we did not examine the postural BP measurement in a cross-over fashion. It was also a limitation that only a single measurement was carried out after the change from the supine to the prone position, *i.e.*, the measurement at 1 min after the change. Sequential measurements would be necessary to evaluate BP variability occurring later than 1 min after the postural change. The validity as well as the reproducibility of the prone hypotension also needs further investigation.

In summary, the present study demonstrated an association between the prone body posture and an asymptomatic reduction in BP. Although the precise mechanisms are still unclear, the BP variation under the prone posture could be a new potential phenotype reflecting BP dysregulation together with orthostatic and diurnal BP variations. Further longitudinal investigations are needed to assess the physiological and prognostic significance of prone-hypotension.

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