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Brief Communications

Squatting: The Hemodynamic Change Is Induced By Enhanced Aortic Wave Reflection

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Background: Although "squatting" in patients with tetralogy of Fallot is famous, the hemodynamic mechanism of the phenomenon has not been fully elucidated. To clarify the mechanism of squatting, the change in aortic pulsatile property induced by squatting was investigated.

Methods: Twelve healthy young adults were enrolled in this study. The change in central pressure waveform induced by squatting was investigated using SphygmoCor (PWV Medical, Sydney, Australia).

Results: The augmentation index during squatting was higher than that during standing (mean ± SEM, 6.3 ± 1.1 *v* 25.0 ± 1.1; $P < .0001$). The heart rate corrected augmentation index during squatting was also higher than that during standing (7.9 ± 0.9 *v* 25.6 ± 1.0; $P < .0001$).

Conclusion: Squatting enhances the aortic wave reflection. It may cause a rise in aortic pressure and may improve cyanotic spells in patients with tetralogy of Fallot. Am J Hypertens 2002;15:986–988 © 2002 American Journal of Hypertension, Ltd.

Key Words: Squatting, wave reflection, afterload, tetralogy of Fallot.

The hemodynamic change induced by squatting is a prompt rise in arterial pressure and cardiac output.[1–3] In patients with tetralogy of Fallot, it is well known that this hemodynamic intervention increases the pulmonary blood flow and improves cyanotic spell.[4,5] However, the mechanism of these hemodynamic changes (blood pressure [BP] rise and increase in cardiac output) has not been fully elucidated.

Many textbooks explain that squatting increases left ventricular preload and afterload.[4,5] Numerous reports have shown that squatting increases preload by augmentation of venous return.[1–3,6] On the other hand, there is no study that demonstrates the increase of afterload by squatting.[6] In fact, Hansen et al demonstrated that the systemic vascular resistance does not change by squatting.[6] Ventricular afterload is defined not only by resistance but also by aortic stiffness and wave reflection. Thus, this study was carried out to elucidate the pulsatile hemodynamic change induced by squatting.

Methods

Study Population

Twelve healthy young adults were enrolled in this study. Eight were men and four were women, with a mean age of 32 years (range 25 to 43 years). They had a physical checkup within 6 months, and no abnormality was pointed out. None of them took any medication.

Measurements

Blood pressure was measured in the right brachial artery using an automated oscillometric method (BP-8800SF, Nippon Colin, Komaki, Japan) during standing and squatting. Aortic pressure waveform was recorded using the applanation tonometry technique and generalized transfer function (SphygmoCor, PWV Medical) during standing and squatting.[7,8] Using the sampled aortic pressure waveform, the inflection time was measured and the augmentation index calculated[9] (Fig. 1).

Baseline upright measurements were obtained after 3 min of quiet standing, including BP and radial artery pressure waveform. Squatting was then performed for 3 min, with body weight positioned over the heels. Both BP and radial artery pressure waveform were re-examined during squatting.

Statistical Analysis

Values are expressed as mean ± SEM. Comparisons between parameters during standing and squatting were performed by paired *t* analysis. Significance was accepted at a value of $P < .05$.

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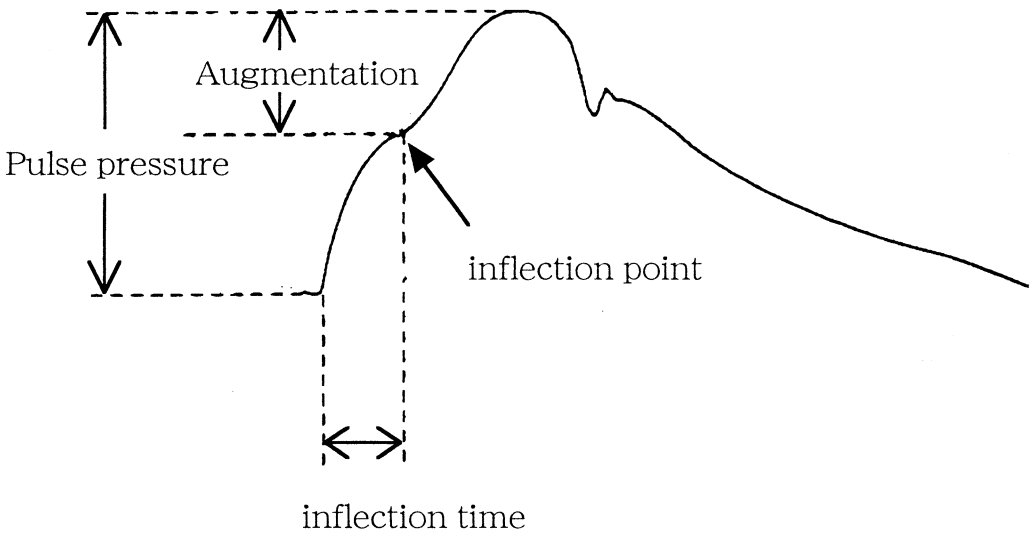

FIG. 1. Schematic representation of augmentation index and inflection time. The augmentation index is the ratio of the augmentation to the pulse pressure. The inflection time is defined as the interval between the onset of a systolic blood pressure waveform and the inflection point.

Results

Fig. 2 shows differences in the central aortic pressure waveform during standing and squatting. There was no significant difference about heart rate between two situations (Table 1). Although systolic BP, diastolic BP, and pulse pressure during squatting were higher than those during standing, only the difference in systolic BP was statistically significant (113.7 ± 1.0 *v* 121.8 ± 1.2; $P = .0081$) (Table 1).

In all patients, the augmentation index during squatting was higher than that during standing (6.3 ± 1.1 *v* 25.0 ± 1.1; $P < .0001$) (Fig. 3). Because the augmentation is influenced by heart rate, the index was transferred at a heart rate of 75 beats/min.[10] The corrected augmentation index during squatting was also higher than that during standing (7.9 ± 0.9 *v* 25.6 ± 1.0; $P < .0001$) (Fig 3). There was no difference in the inflection time between two situations (146.3 ± 1.3 *v* 139.6 ± 1.0; $P = .2107$).

Discussion

The present study shows that squatting increases the left ventricular afterload. According to many textbooks, the hemodynamic mechanism of squatting is an increase in afterload to the left ventricle.[4,5] However, the research that these textbooks cited did not demonstrate an increase in afterload but, rather, an elevation in systemic BP. Without a doubt, the elevation of BP does not always mean an increase of afterload. Only one article examined the change in afterload induced by squatting.[6] It verified that the systemic vascular resistance does not change by squatting. Because vascular resistance is calculated using mean pressure, in the current study, the change in the pulsatile property of systemic circulation induced by squatting was examined. It was found that the aortic augmentation increased and that the systolic BP became elevated by squatting. These outcomes may imply that squatting raises the

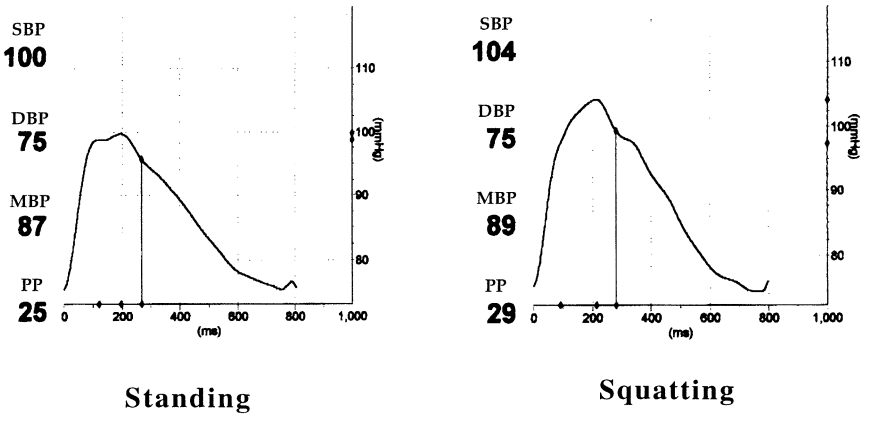

FIG. 2. Difference in central aortic pressure waveform between during standing and squatting. SBP = central systolic blood pressure; DBP = central diastolic blood pressure; MBP = central mean blood pressure; PP = central pulse pressure.

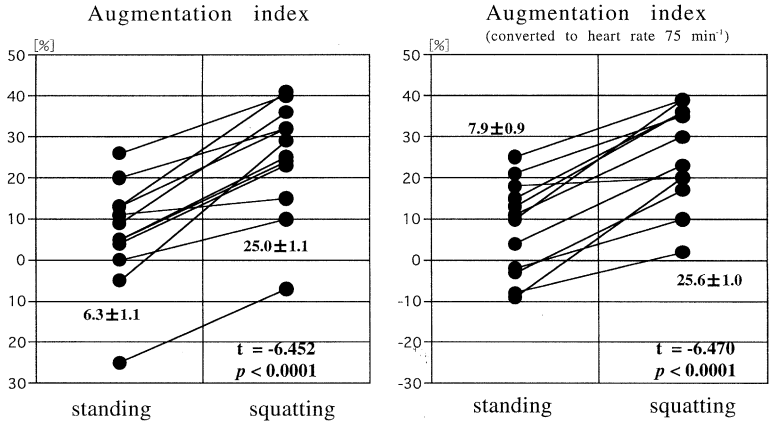

FIG. 3. Difference in augmentation index between during standing and squatting.

afterload to the left ventricle by enhancing aortic wave reflection.

Murgo et al measured the ascending aortic pressure waveform during bilateral external compression of the femoral arteries.[9] They noted that "the end-diastolic pressure and the pressure at the reflection point do not change significantly, but the late systolic portion of the ascending aortic wave form immediately increases," indicating an increase in aortic augmentation. These investigators concluded that augmentation of the reflection phenomenon was accomplished by external occlusion of the vessels in the region of the effective reflection site. Squatting compresses the region of the effective reflection area and may cause augmentation of the reflecting wave.

In conclusion, squatting enhances the aortic wave reflection, and leads to an increase in afterload for the left ventricle. In patients with tetralogy of Fallot, this hemodynamic change shifts the cardiac output from the systemic circulation to the pulmonary circulation.

Table 1. Changes in hemodynamic parameters induced by squatting (n = 12)

	Standing	Squatting	*P* Value
Heart rate (beats/min)	78.2 ± 1.1	76.3 ± 1.1	0.32
SBP (mm Hg)	113.7 ± 1.0	121.8 ± 1.2	0.0081
DBP (mm Hg)	89.9 ± 1.1	92.9 ± 1.0	0.36
PP (mm Hg)	23.8 ± 0.4	28.9 ± 0.7	0.0759

SBP = central systolic blood pressure; DBP = central diastolic blood pressure; PP = central pulse pressure.

Data are given as mean ± SEM.

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