COMMENTARY

Dynamic whole-body vibration training: a unique upstream treatment from the muscle to the arterial system and central hemodynamics

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Hypertension Research (2017) 40, 436–438; doi:10.1038/hr.2017.17; published online 23 February 2017

Obesity is a serious health problem all over the world. This is the case not only for adults but also for children. Childhood obesity is generally carried over to adulthood and increases the risk of cardiovascular death in later life. It has been recognized, however, that the impact of obesity on cardiovascular death diminishes with increasing age. Thus, adequate intervention for young obese individuals may be particularly important.

Arterial stiffness is a measure to quantify the rigidity of the arterial wall. The arterial wall progressively stiffens due to aging, which induces various types of biochemical and histological changes. Moreover, increased intraluminal pressure, or hypertension, could increase arterial wall stress and functionally stiffen the arterial wall. Thus, arterial stiffness reflects the summation of both the organic and functional stiffness of the arterial wall. Pulse wave velocity (PWV) is the most commonly used measure to evaluate arterial stiffness. It has been well recognized that PWV increases with age, high blood pressure and hyperglycemia and thus reflects the summation of major cardiovascular risks.

Carotid-femoral PWV (aortic stiffness) is the gold standard for the evaluation of arterial stiffness, but brachial-ankle PWV (systemic arterial stiffness) has been recognized as useful as well. Both PWV measures have been reported to be independent predictors for cardiovascular morbidity in the general population as well as in hypertensive, diabetic, and other high-risk populations, such as end-stage renal disease patients. Thus, PWV has now been considered as a novel biomarker in the management of cardiovascular diseases.

Previous reports on the relationship between obesity and PWV have been contradictory. Some reports have shown that high body mass index is associated with higher carotid-femoral PWV, while others have demonstrated an opposite relationship. Similar findings have been reported for brachial-ankle PWV. The reason for the discrepancy is not fully understood, but central and peripheral fat may exert opposite influences on arterial stiffness. It has been shown in apparently healthy adults that greater trunk fat is associated with higher carotid-femoral PWV, while the opposite relationship has been observed for peripheral fat and carotid-femoral PWV. Moreover, peripheral lean body mass was more strongly and negatively related to carotid-femoral PWV than peripheral fat. In other words, some degree of protection can be provided either by fat or lean body mass in the limbs. Thus, the influence of higher body mass index on PWV could be very different, depending on whether central obesity or lower body obesity is present. These data strongly suggest that body composition could modulate arterial stiffness. Exercise intervention to reduce trunk fat and/or to increase muscle mass could be a new modality for the improvement of arterial stiffness.

Muscle mass and strength decrease with age. This phenomenon is called sarcopenia. It has been reported that sarcopenia predisposes individuals to disability, dependence, and institutionalization. Several studies have shown that sarcopenia is associated with advanced arterial stiffening. Thus, the increased cardiovascular risk in elderly people can be derived, in part, from decreased muscle mass. This relationship could be further worsened by the accumulation of visceral fat with aging. Therefore, a safe and effective modality to reduce visceral fat and increase muscle mass is likewise important for elderly people to prevent not only disabilities but also cardiovascular diseases.

Advanced systemic arterial stiffness is associated with elevated wave reflection and increased central blood pressure, which reflects a more accurate load to the heart and thereby more closely correlates with adverse cardiovascular events than brachial blood pressure. Exercises to increase muscle mass may have favorable effects on arterial function and central hemodynamics. Evidence suggests that high-intensity resistance training is the preferred exercise modality to improve muscle strength and mass. However, the effects of high-intensity resistance training on arterial function may be adverse or controversial and has not been well established. Thus, an effective and safe training modality that leads to arterial destiffening and the lowering of central blood pressure would be of great clinical importance.

In this issue of Hypertension Research, Alvarez-Alvarado et al. examined the effects of dynamic whole-body vibration training (WBVT) on systemic arterial stiffness, central blood pressure, wave reflection and muscle volume and strength in young, sedentary overweight/obese women. Thirty-eight young (21 years old) overweight/obese women were randomized to WBVT ($n = 25$) or a non-exercising control ($n = 13$) for 6 weeks. The WBVT group performed dynamic leg exercises on a vertical vibration
platform. The training volume progressively increased over the 6-week training period. For arterial stiffness measures, brachial-ankle (systemic arterial stiffness), carotid-femoral (aortic stiffness) and femoral-ankle (leg arterial stiffness) PWVs were examined. Aortic pressure, augmented pressure and augmentation index were derived from radial pressure waveform analysis. Arm and leg lean masses as well as total body fat were evaluated by a whole-body dual-energy X-ray scan.

Leg muscle strength was significantly increased in the WBVT group than in the control group, while none of the body composition measures changed in either group. WBVT significantly reduced carotid-femoral PWV ($P<0.05$), femoral-ankle PWV ($P<0.01$) and brachial-ankle PWV ($P<0.01$) compared with controls. The reduction in the brachial and aortic systolic blood pressures and the augmentation index ($P<0.05$) following WBVT was significant compared to controls ($P<0.05$). The reduction in the carotid-femoral PWV was correlated with a relative increase in leg muscle strength ($r=-0.41$, $P<0.05$). Moreover, the reduction in the brachial-ankle PWV was correlated with decreases in the femoral-ankle PWV ($r=0.53$ $P<0.001$), aortic systolic blood pressure ($r=0.46$, $P<0.001$), augmented pressure ($r=0.28$ $P<0.05$) and augmentation index ($r=0.37$ $P<0.01$).

This unique, randomized interventional study demonstrated for the first time that dynamic WBVT could reduce not only aortic stiffness but also muscular artery stiffness, which was associated with an increase in leg muscle strength. This means that isometric exercise is not mandatory with WBVT to reduce arterial stiffness. This is very important because isometric exercise often elicits exaggerated blood pressure increases, which could be harmful for high-risk individuals such as obese individuals. Moreover, the increase in muscle strength, but not muscle mass, was enough to reduce arterial stiffness. Thus, dynamic WBVT is a safe and effective modality for the improvement of arterial function in young overweight/obese women.

Another important finding of this study is that decreases in the aortic systolic blood pressure, augmented pressure and augmentation index were related to the decrease in brachial-ankle PWV. These relationships with carotid-femoral PWV were not described in the text, but the authors indicated that a reduction in peripheral arterial stiffness was important for the decrease in reflex wave from the periphery. The constant mechanical stimulation to the leg arteries during the vibration exposure could result in greater shear stress-mediated increases in circulating nitric oxide, which could explain the improvements in leg arterial stiffness and the augmentation index.14,16 Thus, it is clear that the leg arteries are not just conduits for blood supply; rather, in cooperation with the leg muscles, they could be involved in the regulation of central hemodynamics. We should focus not only on aortic stiffness but also on leg artery stiffness to more correctly understand the origin of reflex waves and central hemodynamics. Figure 1 summarizes the possible influences of dynamic WBVT on arterial function and central hemodynamics in young overweight/obese women.

Finally, there are two important issues that deserve clarification. First, there are many populations that need safe and effective exercise modalities to improve arterial function. As noted above, elderly people with sarcopenia are a large candidate population because society is progressively aging in many countries. Hemiplegic patients after stroke may also be considered, because it has been reported that muscle mass is decreased and leg artery stiffness is increased at hemiplegic sites compared with non-hemiplegic sites. Second, the mechanism linking muscle function and large-artery function should be clarified. In a previous study that showed the negative relationship between peripheral lean mass and carotid-femoral PWV in apparently healthy adults, higher peripheral lean mass was associated with greater femoral artery diameter. They speculated that an increase in muscle mass demands more blood supply, resulting in a larger size adaptation of the artery, which would potentially slow pulse wave propagation. Unfortunately, the changes in arterial size were not investigated in this study. The close link between an increase in muscle strength and a reduction in carotid-femoral PWV following WBVT suggests that some humoral interaction may exist between muscles and large arteries.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

![Figure 1](image-url)

**Figure 1** Possible influences of dynamic WBVT on arterial function and central hemodynamics in young overweight/obese women.

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1. Twig G, Yaniv G, Levine H, Leiba A, Goldberg N, Derazne E, Ben-Ami Shir D, Tzur D, Afek A, Shamis A, Haskal Z, Kark JD. Body-mass index in 2.3 million adolescents and cardiovascular death in adulthood. *N Engl J Med* 2016; **374**: 2430-2440.

2. Ahmadi SF, Streja E, Zaimatkeghe G, Streja D, Kashyap M, Moradi H, Molnar MZ, Reddy U, Amiri AN, Kovesdy CP, Kalantar-Zadeh K. Reverse epidemiology of traditional cardiovascular risk factors in the geriatric population. *J Am Med Dir Assoc* 2015; **16**: 933-939.

3. Munakata M. Brachial-ankle pulse wave velocity in the measurement of arterial stiffness: recent evidence and clinical applications. *Curr Hypertens Rev* 2014; **10**: 49-57.

4. Vlachopoulos C, Xaplanteris P, Aboyan V, Brodmann M, Cifkova R, Cosentino F, De Carlo M, Gaillo M, Landmesser U, Laurent S, Lekakis J, Mikhailidis DP, Naka K, Pagonides AD, Rizos D, Schmidt-Trucksas A, Van Bortel L, Weber T, Yamashina A, Zimlichman R, Boutouyrie P, Cockroft J, O’Rourke M, Park JB, Schillaci G, Sillehus E, Townsend RR. The role of vascular biomarkers for primary and secondary prevention. A position paper from the European Society of Cardiology Working Group on peripheral circulation: endorsed by the Association for Research into Arterial Structure and Physiology (ARTERY) Society. *Atherosclerosis* 2015; **241**: 507-532.

5. Wildman RP, Mackey RH, Boston A, Thompson T, Sutton-Tyrrell K. Measures of obesity are associated with vascular stiffness in young and older adults. *Hypertension* 2003; **42**: 468-473.

6. Lurbe E, Torro I, Garcia-Vicent C, Rivero J, Fernández-Fornos JA, Redon J. Blood pressure and obesity exert independent influences on pulse wave velocity in youth. *Hypertension* 2012; **60**: 550-555.

7. Tomiyama H, Arai T, Koji Y, Yambe M, Hirayama Y, Yamamoto Y, Yamashina A. The relationship between high-sensitive C-reactive protein and pulse wave velocity in healthy Japanese men. *Atherosclerosis* 2004; **174**: 373-377.
8 Lee M, Choh AC, Demerath EW, Towne B, Siervogel RM, Czerwinski SA. Associations between trunk, leg and total body adiposity with arterial stiffness. Am J Hypertens 2012; 25: 1131–1137.

9 Ferreira I, Snijder MB, Twisk JW, van Mechelen W, Kemper HC, Seidell JC, Stehouwer CD. Central fat mass versus peripheral fat and lean mass: opposite (adverse versus favorable) associations with arterial stiffness? The Amsterdam Growth and Health Longitudinal Study. J Clin Endocrinol Metab 2004; 89: 2632–2639.

10 Ochi M, Kohara K, Tabara Y, Kido T, Uetani E, Ochi N, Igase M, Miki T. Arterial stiffness is associated with low thigh muscle mass in middle-aged to elderly men. Atherosclerosis 2010; 212: 327–332.

11 Williams B, Lacy PS, Thom SM, Cruickshark K, Stanton A, Collier D, Hughes AD, Thurston H, O’Rourke MCAFE Investigators., CAFE Steering Committee and Writing Committee. Differential impact of blood pressure-lowering drugs on central aortic pressure and clinical outcomes: principal results of the Conduit Artery Function Evaluation (CAFE) study. Circulation 2006; 113: 1213–1225.

12 Cortez-Cooper MY, DeVan AE, Anton MM, Farrar RP, Beckwith KA, Todd JS, Tanaka H. Effects of high intensity resistance training on arterial stiffness and wave reflection in women. Am J Hypertens 2005; 18: 930–934.

13 Heffernan KS, Fahs CA, Iwamoto GA, Jae SY, Wilund KR, Woods JA, Femhail B. Resistance exercise training reduces central blood pressure and improves microvascular function in African American and white men. Atherosclerosis 2009; 207: 220–226.

14 Alvarez-Alvarado S, Jaime SJ, Ormsbee MJ, Campbell JC, Post J, Pacilio J, Figueroa A. Benefits of whole-body vibration training on arterial function and muscle strength in young overweight/obese women. Hypertens Res 2017; 40: 487–492.

15 Michishita R, Ohta M, Ikeda M, Jiang Y, Yamato H. An exaggerated blood pressure response to exercise is associated with nitric oxide bioavailability and inflammatory markers in normotensive females. Hypertens Res 2016; 39: 792–798.

16 Perdomo SJ, Moody AM, McCoy SM, Barinas-Mitchell E, Jakicic JM, Gibbs BB. Effects on carotid-femoral pulse wave velocity 24 h post exercise in young healthy adults. Hypertens Res 2016; 39: 435–439.

17 Okabe R, Inaba M, Sakai S, Ishimura E, Moriguchi A, Shoji T, Nishizawa Y. Increased arterial stiffening and thickening in the paretic lower limb in patients with hemiparesis. Clin Sci 2004; 106: 613–618.