

APPENDIX

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DISCUSSION

Neuromuscular theory of WBV

The transmission of vibration mechanical energy applicable to the skeletal muscle or tendon stimulates sensory receptors on muscle spindle primary endings (mainly Ia afferent nerve endings) [1, 2]. Direct excitation of the Ia afferent nerve endings of the muscle spindle, through the vibration of the muscle, stimulates the α -motor neurons, producing reflex contractions of this muscle [3]. The consequence is a tonic contraction of the muscle (i.e., tonic vibration reflex (TVR)) [4–10]. The primary recruitment of the α -motor neurons is done through signals from the brain to the muscles, and the α -motor neurons engender muscle contractions (i.e., TVR) [11, 12]. Neuro-muscular spindles are habitually excited when a muscle is under a static stretch or speedily stretched or overstretched, leading to a rapid and powerful TVR [13]. Some studies reported that once a muscle is subject to vibration, the TVR is constantly excited by contracting and relaxing the muscle, until the vibration stimulus ends [14, 15]. Several studies have established a rise in electromyography activity in reaction to acute WBV, demonstrating that a rise in neuromuscular activity has happened under WBV [16–18]. Consequently, repeating this TVR cycle might improve voluntary skeletal muscle activation with the muscle “stiffening” in an effort to dampen out the consequence of vibration. By improving some facets of the neuromuscular system (e.g., muscle strength and power, speed, flexibility, body stability and composition) [19–34], WBV training improves both muscle performance and function whatever the sex, age and the training status.

Possible mechanisms underlying the effects of WBV training on cardiovascular function

Vibration stimulus excites the TVR and therefore activates and improves the neuromuscular system [16–18, 30]. This activation necessitates the turnover of energy in the muscle (metabolic activity), and therefore an upsurge in the request for energy. Generating the extra energy commanded by the muscle requires some adaptations of the HR and blood pressure and needs additional blood flow (BF) to be supplied to the skeletal muscle. Numerous studies have investigated the impacts of vibration training on cardiovascular function [8, 35–50]. Among these studies, some have indicated that several phenomena (e.g., itchiness, redness, erythema, and oedema) occurred transitorily dur-

ing the first few sessions of WBV training [39, 42, 46–50]. According to some authors [36, 37, 39–43, 45, 47, 49, 51] the chief contributor of the aforementioned phenomena is the rise in BF.

The probable mechanisms underlying the effects of WBV training on arterial function and blood pressure are the improvement of endothelial and autonomic functions [52]. The possible mechanisms underlying the vasodilation effects of WBV exercise may be related to an acute increase in the production of vasodilatory substances including metabolites and nitric oxide [37, 53]. Another possible explanation is related to the fact that skin blood flow might also increase due to friction forces applied by the mechanical vibration on the endothelial cells at the cellular level [43]. WBV may acutely increase local nitric oxide production [53]. Nitrate administration decreases SBP via a reduction in vascular tone of small arteries independently from aortic pulse wave velocity (PWV) [54]. Figueroa et al. [55], have established that acute WBV diminished the increases in reflected wave magnitude, aortic SBP, leg and brachial-ankle PWVs during metaboreflex-induced sympathetic activation succeeding a continuous 4-min period of static squat. This suggests that vibration-related factors induced functional sympatholysis in the legs [55]. A previous study [52] showed that WBV training reduced the sympathovagal balance due to a simultaneous reduction in sympathetic and a rise in cardiovagal modulation. After WBV training, the reductions in sympathovagal balance and brachial-ankle PWV were powerfully linked [52]. Figueroa et al. [52] suggested that the improvement in brachial-ankle PWV would be moderately attributed to a reduction in sympathetic dominance after WBV training. The mechanisms responsible for the reduced arterial stiffness after WBV exercise training could be a localized improvement in arteries exposed to exercise and vibration [56]. In fact, a 10-min period of intermittent static exercises with WBV exercise training immediately reduces brachial-ankle and leg PWV [40, 57]. Moreover, short-term WBV training with and without exercise may decrease leg PWV through a greater discharge of local contraction-related vasodilatory factors and nitric oxide [53, 55]. The special decrease in leg PWV suggests that WBV exercise training may improve leg artery vasomotor tone [56]. The possible mechanisms underlying the impact of WBV exercise training on HR variability may include a rise in baroreflex sensitivity [58, 59]. The evidence strongly suggests that WBV exercise training limits the reduction in the spontaneous baroreflex sensitivity caused by prolonged head-down bed rest in healthy subjects [60]. An additional possible mechanism could be a rise in nitric oxide levels. Numerous lines of evidence indicate that nitric oxide may play a role in cardiac autonomic function by rising vagal and reducing sympathetic activity [61–63]. In fact, WBV exercise training was found to raise circulating nitric oxide [64, 65].

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