Hypertension Research (2010) 33, 544–545

www.nature.com/hr

COMMENTARY

Continuous *vs.* interval exercise training in hypertensive subjects

Kenneth M Madden

Hypertension Research (2010) **33,** 544–545; doi:10.1038/hr.2010.54; published online 30 April 2010

Sedentary behaviors have a well-established association with an increase in overall cardiovascular risk.[1] This connection is likely because of numerous factors, including improved lipid control, improved blood pressure, improved endothelial function and increased heart rate variability.[2] Aerobic fitness has been shown to be an independent predictor of lower mortality regardless of blood pressure or other cardiovascular risk factors.[3] This suggests that there may be additional independent beneficial mechanisms underlying aerobic exercise; one possible explanation would be an exercise-related reduction in arterial stiffness.

The normal aging process is associated with an increase in vascular stiffness, a process that is accelerated by the presence of type II diabetes, hypercholesterolemia and hypertension.[4] The relationship between increased arterial stiffness and cardiovascular mortality is well established.[5] Arterial stiffness is a measure of the total effect of all cardiovascular risk factors on the entire arterial tree, as well as any synergistic relationships between the various risk factors. The arterial wall is composed of both low-compliance collegen fibers that give the arteries strength at higher pressures and high-compliance elastin fibers that provide elasticity at lower pressures (Figure 1).[6] It has been hypothesized that high cardiovascular risk is associated with glycation-related crosslinking of collegen fibers in the arterial wall, resulting in a change in the mechanical properties of the arterial wall. Crosslinking of the collegen fibers means that the high-compliance elastin fibers no longer provide a 'cushion' when the arterial wall is deformed, resulting in overall lower arterial compliance. Theoretically, mechanical distension during aerobic exercise sessions could result in pulsatile 'stretching' of collagen fibers. This 'stretching' could then reverse the glycation-related collagen crosslinking that is responsible for reduced arterial compliance.[7] Increases in the amount of collegen in the arterial wall, hypertrophy of the smooth muscle present in arterial walls and dysfunction of the endothelium have also been hypothesized to underlie increases in arterial stiffness in older adults with hypertension,[7] although the role of exercise in reversing these mechanisms remains uncertain.

Although there is little to no consensus on a standard measure of arterial stiffness, the most commonly used measure in both the literature and consensus statements is pulse wave velocity.[8] Most measures of pulse wave velocity use pressure transducers that are held in place by velcro straps that allow them to be fixed over the skin. Each pressure transducer measures the pulse waveform at each site, allowing one to measure transit time of the pulse wave between the two locations. A higher pulse wave velocity indicates greater arterial stiffness. The transducers are placed over the carotid and femoral arteries for a measure of central arterial stiffness and over the carotid and radial arteries for a measure of peripheral arterial stiffness.[8] Femoral pulse wave velocity is one of the only indices of arterial stiffness directly linked with cardiovascular mortality and morbidity.[8]

Previous prospective examinations of the effect of aerobic exercise on arterial stiffness have shown benefits with vigorous exercise in hypertensive older adults with multiple cardiovascular risk factors,[9] but the effect of less vigorous interventions in hypertensive adults failed to show any benefit.[10] The current paper by Vlachopoulos *et al.*[11] in this edition of *Hypertension Research* provides an explanation for these contradictory findings. Vlachopoulos *et al.* compared the effect on arterial stiffness (n=65) of continuous exercise training *vs.* interval training in middle-aged subjects with hypertension. This paper showed that although both continuous and interval training resulted in improved blood pressure control, only interval training resulted in a reduction in arterial stiffness (when compared with a control group). This result helps to explain the seemingly contradictory results in previous studies, as both interval training and high-intensity exercise would result in increased shear stress and a consequent breaking down of collegen fiber crosslinking.

The improvement in arterial stiffness observed by Vlachopoulos *et al.*[11] with interval training helps to emphasize the fact that the benefits of exercise are due to more than just changes in other well-known cardiac risk factors such as blood pressure, glucose tolerance and body mass index. The results shown by Vlachopoulos *et al.* are in keeping with other examinations of high cardiovascular risk populations. Aerobic fitness was shown to predict mortality in patients with known cardiovascular disease, independent of other known cardiovascular risk indices.[3] Similarly, a vigorous exercise intervention also resulted in improvements in arterial stiffness in older adults with type II diabetes, hypertension and hyperlipidemia without any observed changes in glucose control, blood pressure or lipid profile.[9] Clearly, aerobic exercise in and of itself improves cardiovascular risk, and Vlachopoulos *et al.* provide support for the hypothesis that increased arterial compliance is the underlying mechanism.

Dr KM Madden is at the VITALiTY (Vancouver Initiative to Add Life to Years) Research Laboratory, Division of Geriatric Medicine, Department of Medicine, University of British Columbia, University of British Columbia, Room 7185, 2775 Laurel Street, Vancouver, Canada BC V5Z 1M9.
E-mail: kmmadden@interchange.ubc.ca

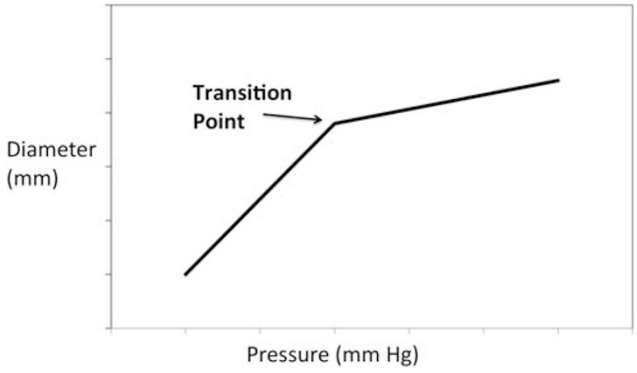

Figure 1 The arterial wall is composed of both low-compliance collegen fibers that give the arteries strength at higher pressures and high-compliance elastin fibers that provide elasticity at lower pressures. The transition point indicates the diameter at which the collegen fibers begin to dominate over the elastin fibers.

There are some limitations to the study presented by Vlachopoulos *et al.* The subject numbers are small and the dropout rate was quite large. However, the subject numbers were quite robust compared with other exercise studies, which illustrates the logistical difficulties in examining standardized exercise interventions over time. There was also no follow-up measures of arterial stiffness after the subjects had completed the intervention, leaving the question open of whether the changes observed in arterial stiffness were permanent over the long term. If the observed decrease in arterial stiffness was maintained several months after the exercise intervention was halted, it might indicate that more permanent changes in arterial structure were occurring as opposed to merely short-term functional changes. The results of this study also cannot be extended to hypertensive subjects with poorly controlled blood pressure (unfortunately a large portion of the population) as these subjects were excluded from the current study.

Sedentary behaviors are responsible for a significantly large proportion of reversible morbidity and mortality, resulting in significant personal and financial costs to society. Hypertensive patients are particularly vulnerable to the effect of cardiovascular disease, and current guidelines and standard practice recommend the prescription of continuous aerobic exercise as nonpharmacological therapy. Vlachopoulos *et al.* present some convincing data that this approach is flawed and the prescription of more vigorous interval training might be more effective in the well-controlled hypertensive population. These results are also important scientifically because they help suggest a mechanistic explanation as to why more vigorous exercise interventions have shown more efficacy in attenuating arterial stiffness than more moderate interventions in the hypertensive population.[9,10] Hopefully, these findings will help stimulate further development of more efficient and effective exercise prescriptions.

CONFLICT OF INTEREST

The author declares no conflict of interest.

ACKNOWLEDGEMENTS

This research was supported by the Canadian Institutes of Health Research, as well as the Academic Enhancement Fund (Department of Medicine, University of British Columbia).

1 Warren TY, Barry V, Hooker SP, Sui X, Church TS, Blair SN. Sedentary behaviors increase risk of cardiovascular disease mortality in men. *Med Sci Sports Exerc* 2010; **42**: 879–885.
2 Madden KM, Levy WC, Stratton JK. Exercise training and heart rate variability in older adult female subjects. *Clin Invest Med* 2006; **29**: 20–28.
3 Vivekananthan DP, Blackstone EH, Pothier CE, Lauer MS. Heart rate recovery after exercise is a predictor of mortality, independent of the angiographic severity of coronary disease. *J Am Coll Cardiol* 2003; **42**: 831–838.
4 Mitchell GF, Parise H, Benjamin EJ, Larson MG, Keyes MJ, Vita JA, Vasan RS, Levy D. Changes in arterial stiffness and wave reflection with advancing age in healthy men and women: the Framingham Heart Study. *Hypertension* 2004; **43**: 1239–1245.
5 Benetos A. Pulse pressure and arterial stiffness in type 1 diabetic patients. *J Hypertens* 2003; **21**: 2005–2007.
6 Woodman RJ, Watts GF. Measurement and application of arterial stiffness in clinical research: focus on new methodologies and diabetes mellitus. *Med Sci Monit* 2003; **9**: RA81–RA89.
7 Cencetti S, Lagi A, Cipriani M, Fattorini L, Bandinelli G, Bernardi L. Autonomic control of the cerebral circulation during normal and impaired peripheral circulatory control. *Heart* 1999; **82**: 365–372.
8 Laurent S, Cockcroft J, Van Bortel L, Boutouyrie P, Giannattasio C, Hayoz D, Pannier B, Vlachopoulos C, Wilkinson I, Struijker-Boudier H. Expert consensus document on arterial stiffness: methodological issues and clinical applications. *Eur Heart J* 2006; **27**: 2588–2605.
9 Madden KM, Lockhart C, Cuff D, Potter TF, Meneilly GS. Short-term aerobic exercise reduces arterial stiffness in older adults with type 2 diabetes, hypertension, and hypercholesterolemia. *Diabetes Care* 2009; **32**: 1531–1535.
10 Ferrier KE, Waddell TK, Gatzka CD, Cameron JD, Dart AM, Kingwell BA. Aerobic exercise training does not modify large-artery compliance in isolated systolic hypertension. *Hypertension* 2001; **38**: 222–226.
11 Guimarães GV, Ciolac EG, Carvalho VO, D'Avila VM, Bortolotto LA, Bocchi EA. Effects of continuous *vs.* interval exercise training on blood pressure and arterial stiffness in treated hypertension. *Hypertens Res* 2010; **33**: 627–632.