Large Artery Stiffness and Baroreflex Function

To the Editor:

We read with interest the recent article by Monahan et al.1 that examined the relationship between carotid arterial compliance and baroreflex sensitivity in the context of aging and fitness. The authors demonstrate that age-related carotid artery stiffening is associated with a smaller carotid diameter change in response to a given increase in systolic blood pressure. Thus, baroreceptor activation is attenuated and likely accounts for the lower cardio-vagal baroreflex sensitivity reported in older individuals. Endurance-trained individuals are known to have large arteries that are more elastic than their sedentary counterparts, a phenomenon likely to reduce age-related impairment in baroreflex function in older athletes.1,2

The findings of Monahan et al.1 are of particular relevance in disease states associated with large artery stiffening. We have previously explored the relationship between the cardiac baroreflex and large artery stiffness in hypertension individuals who are known to have stiffer large vessels than normotensive individuals.3 In this study, modeled changes in arterial circumference based on systemic arterial compliance measurements predicted the impaired gain and reduced bradycardic response to pressure elevation. Interestingly, although stiffer large vessels are predicted to impair the heart rate reduction to pressure elevation due to the nonlinear arterial pressure-volume relationship, we have shown that the effect on responsiveness to pressure reduction is less marked.3 Thus, patients with stiff large vessels are likely to adequately respond to orthostatic stimuli, but impaired responses to a sudden pressure rise may contribute to long-term pressure elevation by upward baroreceptor resetting.4 In addition to hypertension, this mechanism is likely relevant to all states of increased large artery stiffness, including diabetes and coronary disease. With recent studies highlighting large arterial stiffness as an independent predictor of cardiovascular mortality,5 the impaired buffering of sudden pressure elevation should be considered an important contributing mechanism and aerobic exercise training a relevant preventive strategy.

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Response

We would like to thank Dr Kingwell and colleagues for providing us with the opportunity to further discuss our recent report published in Circulation.1 We agree that mechanical properties of the carotid artery associated with age- and habitual exercise-related differences in cardio-vagal baroreflex sensitivity (BRS) may also be important in disease states associated with large artery stiffening. In this regard, we acknowledge the important contribution made by Kingwell et al.,2 who reported that reduced arterial compliance appears to contribute to the depressed baroreflex function in young and middle-aged hypertensive individuals relative to age-matched physically active peers. Additionally, recent experimental evidence indicates that reduced large artery compliance contributes importantly to the depressed cardio-vagal BRS observed in patients with coronary artery disease.3 In other states, such as diabetes, it is possible that other mechanisms in addition to reductions in reduced large artery compliance, such as muscarinic receptor sensitivity/number, may contribute to depressed cardio-vagal BRS as outlined in our Discussion.1 Prospective studies appear warranted to elucidate whether reduced large artery compliance contributes to impaired baroreflex function in other prevalent clinical disorders such as congestive heart failure.

Collectively, the currently available experimental data indicate that large artery compliance may contribute to age-,1,4 physical activity-,1,2,4 coronary artery disease-,3 and resting arterial blood pressure-related2 population differences in BRS. These data should be of great clinical interest in light of recent data demonstrating that depressed cardio-vagal BRS independently predicts mortality after myocardial infarction in humans.5 Furthermore, as suggested by Dr Kingwell and colleagues, interventions capable of preventing or attenuating declines in large artery compliance (ie, regular aerobic exercise) may prove to be powerful primary and secondary strategies to prevent or reverse the associated impairments in cardio-vagal BRS.

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Circulation. 2002;105:e56
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/105/8/e56

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