Response to A New Exercise Central Hemodynamics Paradigm: Time for Reflection or Expansion?

We thank Drs Heffernan and Lefferts1 for their interesting comments and request to expand on the implications of the late-systolic forward decompression (suction) wave (FDW) during exercise. Parker et al3 were the first to suggest that the FDW generated by the left ventricle is related to left ventricular (LV) performance and that wave reflections from the peripheral circulation were less likely to play a dominant role in the modulation of the central blood pressure waveform. Other work has shown that the FDW is closely related to LV mechanics during mid-to-late systole3 and that FDW generation is attributed to LV relaxation and storage of energy in the left ventricle (ie, the same mechanism facilitating early diastolic filling). Notably, the total FDW energy is related to the rate of diastolic relaxation (τ) as well as end-systolic volume6 and correlates closely with peak reverse ejection intraventricular pressure difference.5 It, therefore, seems plausible that the FDW may provide insight into aspects of LV relaxation and filling in addition to late-systolic pressure loading, as suggested by Drs Heffernan and Lefferts.1

During exercise, aortic pressure is increased (raising LV afterload) and because of elevated heart rate, LV diastolic filling time tends to shorten. Under normal circumstances, the positive lusitropic effects of exercise, and optimization of LV relaxation, would be expected to generate greater late-systolic suction wave energy; as was observed in our data.6 Adding further complexity, the positive inotropy produced to overcome raised LV afterload and maintain adequate stroke volume may compensate a relative reduction in end-systolic volume via the Anrep effect. With all this in mind, the negative relation between FDW intensity and end-systolic pressure observed by Drs Heffernan and Lefferts1 in their data may not be unexpected. Indeed, additional analysis of invasive exercise data from our study6 revealed a similar correlation (r = -0.56; P = 0.09) to theirs.

Another interesting aspect of the FDW is evidence showing reduction in magnitude from the carotid to brachial and radial arteries.7 This dissipation occurs to a greater extent than the forward compression wave and is inversely related to vessel diameter. Therefore, FDW intensity is greatest in the aorta, where local suction energy generated by LV relaxation is greatest. Our results show that aortic wave reflection magnitude does not change during exercise, despite large increases in FDW and forward compression wave intensity,5 leading us to conclude that forward propagating waves present in the aorta, that are generated by LV contraction and relaxation forces, are primary contributors to the shape of the exercise central pressure waveform. We agree with the suggestion by Drs Heffernan and Lefferts1 that the FDW has physiological relevance to appropriate ventricular–vascular interaction. Moreover, the accentuation of the FDW with stress induced by exercise underscores the usefulness of exercise as a modality to gain further understanding on the physiology of arterial wave travel.

References

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