A New Exercise Central Hemodynamics Paradigm

Time for Reflection or Expansion?

To the Editor:

Current hemodynamic dogma suggests that the increase in central blood pressure during exercise is largely driven by augmentation of pressure from wave reflections. However, this hemodynamic presupposition lacks teleological and physiological support. During dynamic aerobic exercise, there are marked reductions in terminal impedance because of peripheral metabolic vasodilation. Moreover, despite an increase in central artery stiffness and potential muscle pump–mediated pressure counterpulsations during exercise, increases in aortic diameter coupled with peripheral vasodilation and reductions in peripheral artery stiffness result in central-peripheral impedance matching favorably altering pressure wave timing/magnitude. This hemodynamic milieu would foster transmission of antegrade flow into the active skeletal muscle vascular bed.

We recently read with great interest the findings of Schultz et al1 that propose a new paradigm for exercise central hemodynamics. According to findings obtained from invasive hemodynamic appraisal coupled with wave intensity analysis, it was revealed that changes in central blood pressure during exercise are predominantly attributable to forward traveling waves of myocardial origin and not backward traveling waves/wave reflections. We were also very interested to observe the rather large increase in the forward traveling expansion wave (termed the decompression wave herein) with exercise. This wave, generated at the end of systole by the decline in tension-bearing ability of the myocardium coupled with changes in the inertial force/momentum of aortic blood flow,2 creates a suction effect that applies a braking action to the column of blood from behind and decelerates flow while also reducing pressure.2

We wish to underscore the importance of this suction/decompression wave as an additional moderator of the central hemodynamic response to exercise. Recently, a forward traveling decompression wave in midsystole has been implicated in affecting the mid-late systolic shoulder of the pressure waveform.3 Data from our laboratory (n=18, young healthy men) find that the decompression wave in late systole, measured using wave intensity analysis from the simultaneous assessment of carotid distension waveforms and carotid flow waveforms, is inversely associated with end-systolic pressure (ESP) measured from the carotid pressure wave (r=−0.57; P<0.05) and the synthesized aortic pressure wave (r=−0.53; P<0.05) using planation tonometry. ESP is an important parameter that contributes to cardiac performance (ESP–end-systolic volume relationship), arterial elastance (ESP/stroke volume), ventricular elastance (ESP/end systolic volume), and ultimately ventricular–vascular coupling. Indeed, late systolic load is a crucial moderator of left ventricular tissue velocities, particularly early diastolic function (E’ as assessed with tissue Doppler echocardiography).4

During exercise, as heart rate increases, left ventricular filling time may be compromised. Left ventricular untwisting is enhanced with exercise, and subsequent suction increases early left ventricular filling.5 This suction wave may further help reduce late systolic load, and this has important implications for optimal ventricular–vascular coupling during exercise. Therefore when reflecting on the cause(s) of exercise-induced central pressure modulation, authors are encouraged to expand their view and consider the importance of this decompression wave.

Disclosures

None.

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