EDITORIAL

Take a Deep, Resisted, Breath

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The cardiovascular and respiratory systems exist in series with the lungs, situated between the right and left sides of the circulation. Thus, breathing directly effects the cardiovascular system. How the circulation and respiration became separated into different “systems,” disciplines of study, and specialties in clinical medicine is an interesting epistemological and historical question, but the separation aside, one of the main symptoms of congestive heart failure is shortness of breath. Likewise, patients with structural lung disease frequently develop pulmonary hypertension and subsequent right-sided heart failure. In addition, the outcome of a simple cardiopulmonary exercise test is a remarkable predictor of all-cause mortality, and exercise is one of the best things an individual can do to promote cardiovascular health.1

The pressure generated with a breath and the expansion of the lungs influence the volumes and pressures in the chambers of the heart and blood vessels. These changes, in turn, stimulate sensory nerves that influence the autonomic nervous system and the rate and depth of breathing.2 The pattern generators in the brainstem that drive and regulate heart rate, blood pressure, and breathing are also closely aligned.3 With exercise, a “central command” from higher brain centers accelerates the activity of “both” systems and sends feed-forward signals to the brain stem in preparation for the increased metabolic demands of exercise.4

Breathing can also be therapeutic. This ranges from the simple advice to “take a deep breath,” to ancient meditative and religious traditions, to studies showing that structured deep breathing programs can reduce anxiety,5 help manage pain,6 improve blood glucose,7 and lower blood pressure.8 Patients with lung disease and sleep apnea can also benefit from respiratory muscle training.9,10 Respiratory frequency is a key component of perceived exertion, and anecdotal reports indicate that elite athletes sometimes attempt to control or regulate their breathing during competition to maximize their focus and relaxation during peak competitive efforts.11

During whole body exercise, minute ventilation can exceed 100 L/min in healthy young subjects during high-intensity exercise, and values exceeding 250 L/min have been reported in large male rowers.12 Minute ventilations of this magnitude require substantial muscular work and oxygen consumption by the respiratory muscles.13 They also require, like all skeletal muscles, blood flow to sustain the contractions without fatigue.

So, the stage is set to ask if the respiratory muscles can be a target for exercise. If so, what impact does such exercise have on the cardiovascular system, and what sorts of frequency, intensity, and duration of exercise might work to evoke broad-based exercise adaptations?

In this issue of the Journal of the American Heart Association (JAHA), Craighead et al14 investigate the effect of high-resistance inspiratory muscle strength
training on blood pressure, endothelial function, and arterial stiffness in older adults with mildly elevated systolic blood pressure using a double-blind, randomized, sham-controlled trial. The training consisted of 30 inspiratory efforts (5 sets of 6 efforts at 75% of peak inspiratory pressure with 1 minute rest between; ≈5 minutes in duration) 6 days a week for 6 weeks. This training was well tolerated, as demonstrated by no participant dropouts during the intervention and high adherence (94%) for an exercise intervention. This time-efficient training method was sufficient to reduce blood pressure (≈9 mm Hg systolic) in this population. Furthermore, a reduction in blood pressure was maintained in the high-resistance inspiratory muscle training group in a 6-week follow-up visit after the training intervention in which the participants returned after stopping training. The authors also found that endothelial function (flow-mediated dilation) was improved by ≈45% in the high-resistance inspiratory muscle strength training group. Of particular interest was the improvement in endothelial function with high-resistance inspiratory training in estrogen-deficient postmenopausal women, a group that generally does not demonstrate improvements in endothelial function with exercise training.15,16 The participants in the high-resistance inspiratory muscle strength training group also had improvements in NO bioavailability that occurred via a combination of increased endothelial NO synthase activation and decreased oxidative stress. Although there were no significant effects of this training on measures of arterial stiffness, the authors reported decreases in systemic inflammation. Taken together, these data demonstrate that the high-resistance inspiratory muscle strength training protocol used in this study was sufficient to improve blood pressure and endothelial function in this at-risk population.

These observations raise important questions for the future, and several come to mind. First, what combinations of frequency, intensity, and duration of respiratory muscle training evoke training responses? Second, exercise training can have profound effects on glucose tolerance and insulin sensitivity. Can respiratory muscle training improve these parameters in normal subjects, patients with impaired glucose metabolism, and older subjects? In addition to improved endothelial function, exercise training can improve baroreflex control of blood pressure and increase heart rate variability. Will respiratory muscle training evoke these adaptations as well? Third, exercise training can be useful in the treatment of depression; will formal respiratory muscle training be beneficial in this use case as well? Finally, what are the best ways to study and incorporate respiratory muscle training into physical activity programs for patients with mobility issues, time limitations, and other barriers to traditional exercise programs?

Taking a deep, resisted breath offers a new and unconventional way to generate the benefits of exercise and physical activity.

ARTICLE INFORMATION
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Disclosures
None.

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