

EDITORIAL

Take a Deep, Resisted, Breath

Michael J. Joyner , MD; Sarah E. Baker , PhD

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

See Article by Craighead et al.

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

Key Words: Editorials ■ exercise training ■ high-risk populations ■ hypertension ■ high blood pressure

The opinions expressed in this article are not necessarily those of the editors or of the American Heart Association.

Correspondence to: Michael J. Joyner, MD, Department of Anesthesiology and Perioperative Medicine, Mayo Clinic, 200 First St SW, Rochester, MN 55905. E-mail: joyner.michael@mayo.edu

For Disclosures, see page 2.

© 2021 The Authors. Published on behalf of the American Heart Association, Inc., by Wiley. This is an open access article under the terms of the Creative Commons Attribution-NonCommercial-NoDerivs License, which permits use and distribution in any medium, provided the original work is properly cited, the use is non-commercial and no modifications or adaptations are made.

JAHA is available at: www.ahajournals.org/journal/jaha

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; \approx 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 (\approx 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 \approx 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

Affiliation

Mayo Clinic, Rochester, MN.

Disclosures

None.

REFERENCES

- Feldman DI, Al-Mallah MH, Keteyian SJ, Brawner CA, Feldman T, Blumenthal RS, Blaha MJ. No evidence of an upper threshold for mortality benefit at high levels of cardiorespiratory fitness. *J Am Coll Cardiol*. 2015;65:629–630. DOI: 10.1016/j.jacc.2014.11.030.
- Dawes GS, Comroe JH Jr. Chemoreflexes from the heart and lungs. *Physiol Rev*. 1954;34:167–201. DOI: 10.1152/physrev.1954.34.2.167.
- Eckberg DL. Physiological basis for human autonomic rhythms. *Ann Med*. 2000;32:341–349. DOI: 10.3109/07853890008995937.
- Mitchell JH. Neural circulatory control during exercise: early insights. *Exp Physiol*. 2013;98:867–878. DOI: 10.1113/expphysiol.2012.071001.
- Chen YF, Huang XY, Chien CH, Cheng JF. The effectiveness of diaphragmatic breathing relaxation training for reducing anxiety. *Perspect Psychiatr Care*. 2017;53:329–336. DOI: 10.1111/ppc.12184.
- Anderson BE, Bliven KCH. The use of breathing exercises in the treatment of chronic, nonspecific low back pain. *J Sport Rehabil*. 2017;26:452–458. DOI: 10.1123/jsr.2015-0199.
- Wilson T, Baker SE, Freeman MR, Garbrecht MR, Ragsdale FR, Wilson DA, Malone C. Relaxation breathing improves human glycemic response. *J Altern Complement Med*. 2013;19:633–636. DOI: 10.1089/acm.2012.0603.
- Zou Y, Zhao X, Hou YY, Liu T, Wu Q, Huang YH, Wang XH. Meta-analysis of effects of voluntary slow breathing exercises for control of heart rate and blood pressure in patients with cardiovascular diseases. *Am J Cardiol*. 2017;120:148–153. DOI: 10.1016/j.amjcard.2017.03.247.
- Beaumont M, Forget P, Couturaud F, Reychler G. Effects of inspiratory muscle training in COPD patients: a systematic review and meta-analysis. *Clin Respir J*. 2018;12:2178–2188. DOI: 10.1111/crj.12905.
- Hsu B, Emperumal CP, Grbach VX, Padilla M, Enciso R. Effects of respiratory muscle therapy on obstructive sleep apnea: a systematic review and meta-analysis. *J Clin Sleep Med*. 2020;16:785–801. DOI: 10.5664/jcsm.8318.
- Nicolò A, Massaroni C, Passfield L. Respiratory frequency during exercise: the neglected physiological measure. *Front Physiol*. 2017;8:922. DOI: 10.3389/fphys.2017.00922.
- McKenzie DRE. Cardiorespiratory and metabolic responses to exercise on a rowing ergometer. *Aust J Sports Med*. 1982;14:21–23.
- Aaron EA, Seow KC, Johnson BD, Dempsey JA. Oxygen cost of exercise hyperpnea: implications for performance. *J Appl Physiol (1985)*. 1992;72:1818–1825. DOI: 10.1152/jappl.1992.72.5.1818.
- Craighead DH, Heinbockel TC, Freeberg KA, Rossman MJ, Jackman RA, Jankowski LR, Hamilton MN, Ziemba BP, Reisz JA, D'Alessandro A, et al. Time-efficient inspiratory muscle strength training lowers blood pressure and improves endothelial function, nitric oxide bioavailability and oxidative stress in midlife/older adults with above-normal blood pressure. *J Am Heart Assoc*. 2021;10:e020980. DOI: 10.1161/JAHA.121.020980.
- Pierce GL, Eskurza I, Walker AE, Fay TN, Seals DR. Sex-specific effects of habitual aerobic exercise on brachial artery flow-mediated dilation in middle-aged and older adults. *Clin Sci (Lond)*. 2011;120:13–23. DOI: 10.1042/CS20100174.
- Santos-Parker JR, Strahler TR, Vorwald VM, Pierce GL, Seals DR. Habitual aerobic exercise does not protect against micro- or macrovascular endothelial dysfunction in healthy estrogen-deficient postmenopausal women. *J Appl Physiol (1985)*. 2017;122:11–19. DOI: 10.1152/jappphysiol.00732.2016.