Last Word on Point:Counterpoint: Exercise-induced intrapulmonary shunting is imaginary vs. real

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TO THE EDITOR: We thank our colleagues from UCSD for this enjoyable discourse as well as the additional commentaries from colleagues around the world. In reviewing the Point: Counterpoint (3, 5) and the commentaries submitted (see Ref. 4), it appears that there is general agreement that some type of large-diameter intrapulmonary arteriovenous pathways are recruited with exercise. However, their size, magnitude, regulation, as well as their contribution to pulmonary gas exchange in health and disease remains to be fully understood. As Dr. Jones points out (see Ref. 4), comparisons between healthy subjects who demonstrate a positive contrast echocardiogram vs. those healthy subjects who do not would add to our understanding of the significance of these yet unidentified vessels. In reference to comments made by Drs. Sheel and Dr. Hughes (see Ref. 4), quantification of intrapulmonary arteriovenous shunt has been done in dogs (57) and most recently in humans (6); however, the ability to accurately size these intrapulmonary arteriovenous pathways in humans is limited because albumin macroaggregates have a variable size distribution, whereas polymer microspheres are of a known and a very specific diameter, but are not injectable in humans.

Drs. Naeije and Faoro (see Ref. 4) point out that there may be more plasticity within the pulmonary circulation than previously assumed and highlight the pulmonary-related complications associated with chronic liver disease as an example of this plasticity. In this example, Drs. Naeije and Faoro also bring attention to the fact that in a pathophysiological condition, gas exchange-dependent techniques detect a shunt fraction inconsistent with the shunt fraction detected by the anatomic-based methods. It is of interest that observations dating back more than 30 yr in patients with hemorrhagic hereditary telangiectasia previously highlighted how “shunt” calculated anatomically is different than “shunt” determined with gas exchange techniques (2). With respect to pathophysiology, Dr. Bates (see Ref. 4) suggests a role for these pathways in both cardiovascular and pulmonary pathologies, highlighting the need for further studies of these intrapulmonary arteriovenous anastomoses in both health and disease.

In summary, with general agreement between all parties, the next steps will be to 1) determine regulation of these vessels, 2) determine their role in pulmonary gas exchange in health and disease, 3) better quantify magnitude of vessel blood flow, and 4) establish anatomic origin of these vessels. Undoubtedly the role of the Journal of Applied Physiology’s Point:Counterpoint is to search for truth within controversies that exist in physiology at present so that our understanding will evolve. Our colleagues at UCSD suggested that our shunts are much like Horton’s Whos, small and insignificant, leaving the reader to decide if they should even care about this controversy, while explanations for pulmonary gas exchange efficiency during exercise remain unresolved. In keeping with the spirit of this Point:Counterpoint, we would be remiss to not include a final closing quotation from Dr. Seuss, which we feel sums up the collective interest of ourselves and our colleagues regarding the area of pulmonary gas exchange, pulmonary circulation, and exercise: “Unless someone like you cares a whole awful lot, nothing is going to get better. It’s not. (1)”

GRANTS
This work was supported by the American Heart Association Scientist Development Grant SDG2280238 (to A. T. Lovering), American Heart Association Grant-In-Aid 0550176Z (to M. W. Eldridge), and Canadian Institutes of Health Research New Investigator Award (to M. K. Stickland).

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