TO THE EDITOR: The clear and resounding conclusion of the correspondents who weighed in on this debate is that exercise limitation in COPD is multifactorial. Protagonists who advance a single dominant mechanism of activity limitation in this phenotypically heterogeneous population do so at their peril. My esteemed colleagues, Drs. Maltais/Debigaré and Macklem/Aliverti must accept (however grudgingly!) that a mechanical limitation to ventilation is an important and partially reversible contributor to poor exercise performance in COPD. In fact, Maltais was lead investigator in a multicenter study that demonstrated that exercise improvement following bronchodilator treatment in COPD was partially explained by reduced lung hyperinflation (5); similarly, Macklem is a pioneer in the development of innovative endoscopic methods of lung volume reduction in advanced COPD (4). In the same confessional vein, our own group has more than a passing interest in peripheral muscle dysfunction in COPD (2).

We were so intrigued by Aliverti and Macklems’ hypothesis that excessive expiratory muscle activity limits exercise, we decided to test it formally in a recent study (3). We conducted mechanical measurements during symptom-limited cycle exercise in 16 patients with moderate to severe COPD (FEV1 = 48% predicted). In our preliminary analysis: intolerable dyspnea was the main exercise-limiting symptom; dynamic pulmonary hyperinflation from rest-to-peak exercise was 0.83 liters, end-inspiratory lung volume reached 93% of total lung capacity at a low peak ventilation; peak inspiratory pleural pressures reached 29% of maximal inspiratory pressures; and end-expiratory gastric pressures increased smoothly throughout exercise in all patients to reach a peak of 19% of maximal gastric pressure. We found no evidence of abdominal muscle derecruitment even in those with the most advanced disease and could find no correlation between the magnitude of expiratory muscle force generation and perceived dyspnea intensity (described mainly as inspiratory difficulty).

In considering integrated mechanisms of exercise limitation, we cannot lose sight of the reality that disabling dyspnea is the primary preoccupation of the COPD patient. Our knowledge of the mechanisms of this symptom remains woefully deficient and effective management will remain an elusive goal unless research in this area develops substantially beyond its current deplorable state. In the meantime, the caregiver can at least derive some reassurance from the fact that modern long-acting bronchodilators achieve sustained pharmacological lung volume reduction with attendant improvements in activity-related dyspnea and exercise tolerance (5). A remaining challenge is to convert this newfound increase in functional capacity into longer term improvements in functional status. To do this we must reverse inactivity-related, global skeletal muscle deconditioning with structured multimodality exercise training. This, however, is particularly difficult to achieve in patients with critical ventilatory limitation and severe exertional dyspnea. New approaches that reduce the rate of dynamic pulmonary hyperinflation during exercise beyond that achieved by maximal bronchodilatation [i.e., hyperoxia, heliox and ventilation-feedback methods (1)] deserve further scrutiny as potential adjuncts to exercise training in such patients.

As we pursue increasingly sophisticated models of exercise limitation in COPD we should be careful not to neglect the fundamental reality—When you can’t breathe nothing else matters!

REFERENCES