Is obesity deflating?

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CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) is a progressive disease that leads to a spiral of decline following a pattern of obstruction → hyperinflation → dyspnea → inactivity → deconditioning → muscle impairment → worsening dyspnea. In addition, there are increases in a number of biochemical mediators as the disease worsens that influence oxidative stress, inflammation, and neurohumoral function. If hypoxemia develops, sensitive genes are stimulated that may further accentuate maladaptive pathways. Thus the disease not only influences the pulmonary system but develops into a systemic illness that in turn plays a role in driving the pathophysiology.

Obesity has typically been thought to be a comorbidity in patients with COPD, and intuitively this would seem linked to further derangements in respiratory mechanics due to reduced expansion of the thorax from the increased inspiratory elastic load and a diminished caudal movement of the diaphragm as well as accentuated breathing against narrowed and closed airways on expiration, all further contributing to the spiral of decline in systemic function.

However an interesting controversy has developed on the role of obesity in the COPD population, and even a possible protective role for obesity against mortality has emerged (7). This has been portrayed as the “obesity paradox” since this is contrary to data from the general population where obesity is associated with decreased life expectancy (although this may be linked to related issues rather than obesity per se, e.g., reduced physical activity). In addition, recent studies suggest that exercise capacity may be maintained in obese COPD patients with similar or decreased levels of dyspnea as non-obese patients (8). This is important, but puzzling since exercise breathing mechanics are altered in otherwise healthy obese adults and is by itself predictive of mortality in the COPD population.

To further understand the interaction between obesity, lung mechanics, exercise tolerance, and dyspnea, Ora and colleagues (9), in an article in this issue of the Journal of Applied Physiology, carried out a study with carefully matched obese and normal-weight COPD patients based on disease severity (defined by pulmonary function measures), but differing in weight by 23 kg and in body mass index (BMI) by 8.8 kg/m². Both patient groups had moderate COPD (class IIa by the GOLD classification guidelines). Extensive measurements were made of respiratory mechanics and symptoms at rest and during exercise. Total lung capacity (TLC) and functional residual capacity (FRC) were reduced in the obese patients as was the expiratory reserve volume (ERV). The sniff esophageal pressure (Pes) and transdiaphragmatic pressures (Pdi) were similar, and lung static recoil pressure was also increased in obese patients.

Both groups performed constant-work exercise at 75% of the maximal incremental work rate, while measures of lung mechanics, gas exchange, and symptoms were recorded. Interestingly, the groups worked at similar absolute workloads, and had relatively well-matched minute ventilations through the constant-work exercise, with relatively similar oxygen consumption (V̇O₂) values. Exercise duration was similar between groups as was the degree of dyspnea. Furthermore, both groups demonstrated dynamic hyperinflation with exercise of a similar degree and the majority of measures of lung mechanics were similar between groups, including the apparent degree of ventilatory constraint. However, abdominal pressures were higher at rest but failed to reach significance during exercise, unlike the elevated gastric pressures seen during exercise in otherwise healthy obese men and women (1, 4). This, combined with slightly greater tidal swings in Pes (albeit not statistically significant), suggests an increased work and presumably oxygen cost of breathing in obese COPD patients. The authors concluded that three important parameters may have offered an advantage that allowed preservation of exercise tolerance in the obese COPD patients. This included the greater static lung elastic recoil, the persistently lower operational lung volumes, and an improved ventilatory efficiency.

A number of interesting questions arise from their work. First, one might expect the obese COPD patient to have a reduced exercise tolerance, linked to a greater mechanical load and/or negative consequences of increased airway closure. Second, are there truly benefits from obesity, such as breathing at lower lung volumes, that allow maintenance of exercise tolerance or are we simply observing a heterogeneous group of COPD patients that generally will exercise up to a given level of mechanical constraint to breathing that is defined by their baseline respiratory mechanics? Third, how well does BMI really identify obesity vs. identifying stockier individuals with greater lean muscle mass with perhaps greater preservation of other key variables related to exercise performance (a different COPD phenotype)? And fourth, what would be the mechanism for the apparently improved ventilatory efficiency in obese vs. normal-weight COPD patients? Finally, could the obese COPD patients actually be exercise trained at low work rates due to carrying the extra 23 kg during activities of daily living? It is also unclear if the same results would have been found in more severely obese patients or in obese COPD patients with less emphysema and that were not as hyperinflated at rest. Many mild to moderate COPD patients do not have an increased

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TLC, and the effects of obesity may be more deleterious to them.

The most consistent finding in obesity is the shifting of the vital capacity and tidal breathing to lower lung volumes, and in the COPD population, the apparent ability to breathe at these lung volumes without greater flow limitation. We know from work in lung volume-reduction surgery that small improvements in the length-tension relationships of the respiratory muscles can have important implications, and previous studies have reported the impact of lung volume (muscle length) and flow rate (velocity of shortening) on the ability to generate inspiratory pressure as well as on the energetics of the respiratory muscles (2, 3, 6). Figure 1 gives an example of the theoretical impact of the lower-lung-volume, tidal breathing in the obese subjects on the ability to produce inspiratory pressure and presumes a similar TLC could exist if obesity were not contributing to the reduction. Although the obese subjects in the study of Ora et al. (9) did not demonstrate an increased ability to generate maximal inspiratory muscle pressure, it is unclear how lung volume and flow rate influenced the dynamic pressure-generating capacity in the obese group and if a true mechanical advantage exists. The mechanism for the apparent fall in TLC in obesity remains somewhat unclear and is also variable (10). In a previous imaging study by Watson and colleagues (12), it appeared that this was primarily due to an inability to further expand the thorax and thus presumably there is a sharp rise in elastic load as the tidal breaths in the obese subjects approach TLC.

Could the elevated gastric pressures influence loading conditions and muscle mechanics, length-tension relationships, and synergies across accessory muscles to provide a more efficient pump? Previous studies using abdominal binders or strapping in COPD patients have been variable, with one study suggesting a reduction in exercise tolerance with abdominal binding (5). On the other hand, pursed lip breathing is often taught to COPD patients to increase expiratory time and potentially keep airways from collapsing, while at the same time increasing the gastric pressure (as well as Pes) (11). Thus this may point to greater benefits from improving gas exchange in COPD vs. the negative consequences of increased expiratory muscle work, or once again point to the benefits of improved inspiratory muscle length.

Going forward, the work of Ora and colleagues (9) highlights an important area for further research. There remain many unanswered questions regarding obesity and COPD and the apparent paradox. Do the obese patients truly have mechanical advantages to breathing as a result of less hyperinflation? Are the respiratory muscles more fatigue resistant? What happens to lung function and exercise tolerance with a controlled, healthy weight loss in obese COPD patients? Would these patients become like the normal-weight patients in the Ora study with higher TLC, FRC, and ERV, or are there differences in lung parenchyma that are specific to these patients? Why doesn’t the proinflammatory state of obesity further drive the disease process in obese patients with COPD? What is the role of fat distribution and the impact on respiratory muscle geometry vs. impeding thoracic expansion or competing for intrathoracic space with the lungs? How does abdominal fat (visceral or subcutaneous) alter loading conditions and muscle mechanics, length-tension relationships, and the energetics of the diaphragm and accessory inspiratory muscles?

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).
REFERENCES


