HEAVY BREATHING

Robert B Banzett¹, Stephen H Loring¹,²

¹Division of Pulmonary & Critical Care, Dept. of Medicine
²Dept. of Anesthesia and Critical Care
Harvard Medical School
Beth Israel Deaconess Medical Center

Corresponding author:
Robert B Banzett
Division of Pulmonary and Critical Care
Beth Israel Deaconess Medical Center
330 Brookline Ave
Boston, MA 02215 USA
dyspnea@hsph.harvard.edu
Phone: 617 667-0572
FAX: 617 667-0579
In this issue, Ofir et al (6) examine the mechanism of dyspnea due to obesity. Obesity is a well-recognized threat to public health in developed countries; for example, the NIH estimates that a third of Americans are obese (5). Dyspnea on exertion is a common problem in obesity (9). Because dyspnea is extremely unpleasant, most humans will alter their behavior to avoid experiencing prolonged or severe dyspnea. In the case of obesity, this leads to an unfortunate positive feedback -- avoidance of exercise leads to greater weight gain, which in turn leads to stronger avoidance of exercise, etc. Understanding the mechanisms causing dyspnea in obese subjects might help us to find ways to break this vicious cycle.

The American Thoracic Society defines dyspnea as “a subjective experience of breathing discomfort”; they also note that dyspnea “consists of qualitatively distinct sensations that vary in intensity” (1). At least three distinct forms of ‘dyspnea sensations’ have been described; each has a different neural mechanism (2). Because dyspnea is a subjective, multi-component experience, adequate measurement is challenging. Few subjects understand the word “dyspnea”, and their ratings of dyspnea may include any or all of the individual uncomfortable respiratory sensations that comprise their experience. Thus, researchers must specify what sensation is studied. The present authors do this in two ways: they describe for us exactly what they asked subjects to rate (“labored or difficult breathing”) and they provide debriefing information that helps us understand what the subjects actually felt.

The authors set out to test a hypothesis regarding the respiratory mechanical factors that might increase dyspnea in obese subjects. As often happens in science, the investigators were trying to distinguish between possibilities 1 and 2, but instead found possibility Y, something completely different. That is, the original hypothesis was rendered moot by an unexpected finding: for a given minute ventilation, obese subjects rated the same amount of dyspnea as controls.

Ofir et al (6) designed their study to isolate the respiratory impairment of obesity. Many studies of exercise in obesity employ treadmill grade exercise as the stimulus. Although this is very relevant to daily life, it also introduces an important difficulty to those wishing to examine the effect of obesity on the respiratory system: the added work of weight-bearing locomotion places added demand on breathing; this metabolic demand may dwarf the effects of respiratory mechanical derangement. The present investigators used cycle exercise to minimize this difficulty. The authors report that the obese subjects could produce 17% less external work on the cycle, yet they achieved similar maximum oxygen uptake. Perhaps surprisingly, obese subjects were not more breathless at a given oxygen
uptake, or at a given minute ventilation; they simply expended more energy for a given external work rate.

So why doesn’t the added chest wall mass cause more dyspnea? The respiratory mechanical impact of obesity does not easily fit the standard terms ‘obstructive’ or ‘restrictive’. Many people assume that obesity will increase the work of breathing because the added chest-wall mass must be lifted and accelerated. Closer consideration of chest-wall mechanics challenges this assumption. Most studies show that passive elastance of the chest wall is not much altered by obesity (10). Although the respiratory muscles must accelerate the mass of the chest wall, velocities are low. Maximum chest-wall displacements during 1.5 liter breaths are about 0.5 cm; if breathing is sinusoidal and inspiratory time is 1 sec, chest wall acceleration will be .007m/sec/sec, only about 0.1% of the acceleration of gravity. In the upright posture in normal subjects, the effect of gravity is expiratory on the rib-cage and inspiratory on the abdomen (4); the net effect of these opposing effects is inspiratory (3). Thus simply adding mass to the ribcage and abdomen would likely increase relaxation volume. However, the increased volume of intra-abdominal and intrathoracic fat raise pleural pressure and reduce resting end-expiratory lung volume (FRC). As a result, obese subjects at rest show significant expiratory flow limitation; the increased pulmonary resistance and increased pulmonary elastance at rest are mainly due to operating at a volume low enough to cause flow limitation in normal lungs (11) (7, 8). This suggests that the obese person could restore normal pulmonary function to meet the demand of exercise simply by elevating end-expiratory volume. Ofir et al (6) showed that they do just this. This solution is much less costly than those available to patients with pulmonary fibrosis or chronic obstructive lung disease. The perception of respiratory work by obese subjects should thus be little greater than that experienced by non-obese subjects, as Ofir et al. (6) clearly show.

The finding that obese subjects can achieve similar oxygen uptake with similar respiratory discomfort is, perhaps, good news – the benefits of exercise are largely related to oxygen uptake rate. Exercise programs for obese subjects can be designed to achieve similar physiological benefit, and can be easily scaled by the individual’s tolerable level of exercise. The present result, obtained in moderately obese women who were motivated enough to participate in a study, will not apply to everyone, but it will apply to a large portion of the overweight population. As often happens, this finding leads to another question – why do obese subjects consume more energy to do the same amount of external work? Altered leg mechanics or altered muscle fiber composition are possibilities. Other possibilities include added cost of thermoregulation (cost of pumping more blood to the skin,
and/or cost of secreting more sweat), or metabolic rate increase due to greater temperature rise (Q10 effect). We look forward to the answer, or even better, to the next unexpected question!

Acknowledgments
Supported by HL46690 and HL63737

References