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REVIEW

The respiratory muscles in eucapnic obesity: Their role in dyspnea

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Summary
Regular exercise appears to be one of the best predictors of successful weight maintenance. Although physical activity and exercise are important components in the prevention and treatment of obesity, many obese adults without coexisting disorders are unable to exercise due to dyspnea on exertion. As a result they may not participate in regular physical activity. Therefore exertional dyspnea in obese adults is also an obstacle to the prevention and treatment of obesity and coexisting comorbidities. The available data suggest that increased respiratory muscle force generation, and the concomitant increase in respiratory neural drive associated with increased ventilation are an important source of sensation of respiratory effort in obese subjects. Whether activity-related breathlessness is due to either abnormal respiratory mechanical factors (flow limitation and/or chest elastic loading) or the increased metabolic demand of locomotion in obesity, or both of these together, the available data indicate that intensity of dyspnea at any given ventilation and oxygen uptake does not increase in obese subjects as compared with normal weight control subjects. Does this mean that respiratory mechanical factors are unlikely to be contributory? Nonetheless, the component of metabolic cost of breathing may not be accounted for in the measured mechanical work of breathing because of the number of included complex variables. That a decrease in efficiency of the respiratory muscles during exercise contributes to dyspnea in hyperinflating obese subjects should not be disregarded.

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Introduction

Although its etiology is not completely understood, it is agreed that obesity results from the chronic imbalance between energy intake and energy expenditure.[1] It is therefore clear that over consumption of calories and inadequate physical activity are the primary factors underlying the rise in the incidence of obesity. A substantial decrease in physical activity dictates a decrease in daily energy expenditure. Regular exercise appears to be one of the best predictors of successful weight maintenance.[2] Nevertheless, since muscles must work to move an obese body, obesity imposes high metabolic demands which are reflected in the fact that the obese subject's oxygen consumption (VO_2) and carbon dioxide production (VCO_2) are higher than normal at rest and during exercise.[3]

Achieving an augmented ventilation (VE) to oxygen uptake (VO_2) ratio imposes an additional physiological burden because the work of breathing is increased in obesity; this potentially results in respiratory muscle inefficiency.[4] Likewise, heightened demands for diaphragm work in obese people, and demonstration that static inspiratory muscle pressure was only 60—70% of normal has been reported as an index of inspiratory muscle inefficiency, if not dysfunction.[5] The respiratory muscles that might be compromised by obesity are those involved in inspiration, but of these only the diaphragm has been extensively studied. Moreover, reports on the effects of obesity on respiratory muscle function are conflicting. Both muscle biopsy specimens[6] and measurement of CT density show fatty infiltration of non respiratory skeletal muscles,[7] but the extent to which this affects muscle strength is unclear. Newham[7] reported an increased density of quadriceps accompanying weight loss after bariatric surgery, but there was no corresponding increase in the strength of the muscle as assessed by maximal voluntary contraction. In contrast, a recent report on respiratory muscle function shows an increase in maximum inspiratory pressure and in respiratory muscle endurance six months after bariatric surgery, with the improvement being closely related to loss in body mass index (BMI).[8] Although it seems likely that respiratory muscle function in obesity may be compromised by the increased load the muscles are required to overcome, and by some reduction in their capacity,[9—12] this view must be tempered by evidence to the contrary.[13—18]

This review will discuss to what extent respiratory muscle energy has to be spent to overcome mechanical characteristics of the lung and chest wall in eucapnic obese subjects. In particular we will review: (i) the mechanical burden on the respiratory muscles; (ii) their potential structural and biological abnormalities, (iii) their efficiency based on energy cost of breathing and (iv) their impact on dyspnea during regular exercise.

Lung and chest wall mechanics at rest

It is generally believed that the burden of moving a large chest wall increases the work of breathing in obese subjects, with the respiratory muscles encountering a greater mechanical load. The increase in the work of breathing could result from work done to overcome chest wall compliance (Cw), or work performed on the airways and lungs. Studies in obese normal subjects have suggested that Cw is decreased by adipose tissues encasing the chest and the abdomen.[19—22] A decrease in Cw would overload the respiratory muscles by increasing the respiratory effort to displace the chest wall in obese subjects. The low lung compliance that occurs in obesity is often thought to be the consequence of a stiff chest wall. However, what is the body of evidence of a lower Cw in obesity? First of all, the accurate measurement of Cw requires complete relaxation of the respiratory muscles. Naimark and Chernicak[19] found that Cw was lower than normal in seated awake obese subjects. To demonstrate that the chest wall was relaxed they measured electromyographic activity of the pectoral, intercostal, scalene and rectus abdominis muscles and found no evidence of activity during the measurements. There are, however, two biases in the study: the first was the known difficulty of detecting respiratory muscle activity during normal breathing using surface electrodes in obese subjects with a thick chest wall. Second, unlikely as usual, they found normal lung compliance.

Sharp et al.[20] also found a low Cw in awake obese subjects in whom, however, Cw was measured by subtracting the reciprocal of lung compliance measured in a seated subject from the reciprocal of total respiratory