

# Effects of obesity on respiratory mechanics at rest and during exercise

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Obesity is a major health problem worldwide. The prevalence of obesity is increasing and its association with multiple comorbidities is now firmly established. It is considered as an independent risk factor for reduced survival. Obesity directly affects respiratory mechanics at rest and during exercise. Obese individuals usually show changes in certain lung volumes, respiratory compliance and ventilatory behaviour. Dyspnoea is the limiting symptom usually experienced by these individuals.

## Introduction

Obesity is a chronic disease characterized by the excessive accumulation of body fat that is harmful to the individuals [1]. Usually, body fat above 25% in men and body fat above 30% in women is considered to be obese; therefore, on the basis of this definition, obesity should be determined by measuring body fat. Unfortunately, measures of body composition are not universally and readily available in all clinical settings and therefore most data on the effects of obesity on health rely on the relationship of weight to height, such as the BMI, which is generally useful for describing different magnitudes of obesity. On the basis of BMI, obesity can be classified into mild/class I (30–34.99 kg/m<sup>2</sup>), moderate/class II (35–39.99 kg/m<sup>2</sup>) and morbid/class III (>40 kg/m<sup>2</sup>) [2]. In addition to the degree of obesity, the distribution of excess fat affects the health risks of obesity. A waist-to-hip ratio greater than 1.0 in men and greater than 0.85 in women identify individuals with abdominal fat accumulation and also indicates increased risk for cardiovascular and metabolic disturbances [3].

## Obesity and respiratory mechanics at rest

Obesity can profoundly alter pulmonary function and diminish exercise capacity by its adverse effects on respiratory mechanics, resistance within the respiratory system, respiratory muscle function, lung volumes, work and energy cost of breathing. The mass loading effects of excess adipose tissue on the chest wall and abdomen results in reduced compliance of the respiratory system. This is partially caused by a fall in lung compliance, which is decreased by ~25% in simple obesity and 40% in severe obesity [4,5]. Increased pulmonary blood volume [6], as well as increased closure of dependent airways [7], probably contributes to this decreased lung compliance.

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Airway, chest wall and respiratory system resistances are elevated in simple obesity and are higher with more elevated BMI. The primary mechanism for increased lung and total respiratory system resistance is likely related to increased resistance at the level of the small, rather than large, airways (due to reduced lung volume) [8].

Maximal inspiratory and expiratory pressures (PI<sub>max</sub> and PE<sub>max</sub>, respectively), measured to test respiratory muscle strength, are normal in eucapnic mild-to-moderate obese individuals, but diminished in morbid obesity [9]. Given the elevated total respiratory resistance, decreased compliance and increased inspiratory threshold load associated with obesity, it is not surprising that the mechanical work and oxygen cost of breathing are increased [10]. The work of breathing is three to four times higher in moderate obesity compared with that in normal-weight individuals [11].

The effect of obesity on spirometry and lung volumes is complex and influenced by the degree of obesity, age and type of body fat distribution (central or peripheral). The most frequent pulmonary function test abnormality associated with obesity is decreased expiratory reserve volume, most probably due to displacement of the diaphragm into the chest by the obese abdomen [1]. There is an exponential relationship between increasing BMI and decreasing expiratory reserve volume in a healthy population [1]. The impact of obesity on other lung volumes is modest. Total lung capacity (TLC) may slightly decline with morbid obesity [12]. Residual

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volume (RV) and vital capacity are usually within the normal limits, and forced vital capacity (FVC) is usually in the lower limits of normal. RV/TLC may be increased in obesity, reflecting air-trapping secondary to increased volume-dependent airway closure [1]. The inspiratory capacity (IC) at rest increases about 0.35–0.55 l [13], and the IC/TLC ratio increases with increasing BMI [14]. Forced expiratory volume in 1 s (FEV<sub>1</sub>) is usually in the lower range of normal. The FEV<sub>1</sub>/FVC ratio is generally normal or slightly elevated, indicating the absence of airway obstruction associated with obesity [15,16]. However, obstruction of the small airways may be detected on the flow volume loop in obese individuals [17]. Table 1 summarizes the effects of obesity on pulmonary functions at rest.

**Obesity and respiratory mechanics during exercise**

During exercise, there is an increase in ventilatory requirements, reflecting the higher metabolic cost of external work with the increase in oxygen consumption (VO<sub>2</sub>) and carbon dioxide production (VCO<sub>2</sub>) [18,19].

Obese individuals show different operational lung volumes (Fig. 1) and different breathing pattern responses to incremental cycle exercise, which is more shallow and rapid, compared with that in normal-weight individuals [18]. The larger resting IC and inspiratory reserve volume indicate that obese individuals can increase their end expiratory lung volume without end-inspiratory lung volume prematurely encroaching on the TLC. Thus, there is no mechanical limitation in tidal volume during exercise compared with that in normal weight individuals. This ventilatory behaviour with a more rapid and shallow breathing during exercise may be simply a compensatory adaptation to decrease the

elastic work of breathing [20]. The pulmonary dynamic hyperinflation that can be detected in obese patients during exercise is potentially a cause of decreased maximal performance, but it was demonstrated that it has no direct impact on breathlessness in those patients. The presence of higher operational lung volumes in obese patients during exercise, with higher IC, works as a mechanism of adaptation [21].

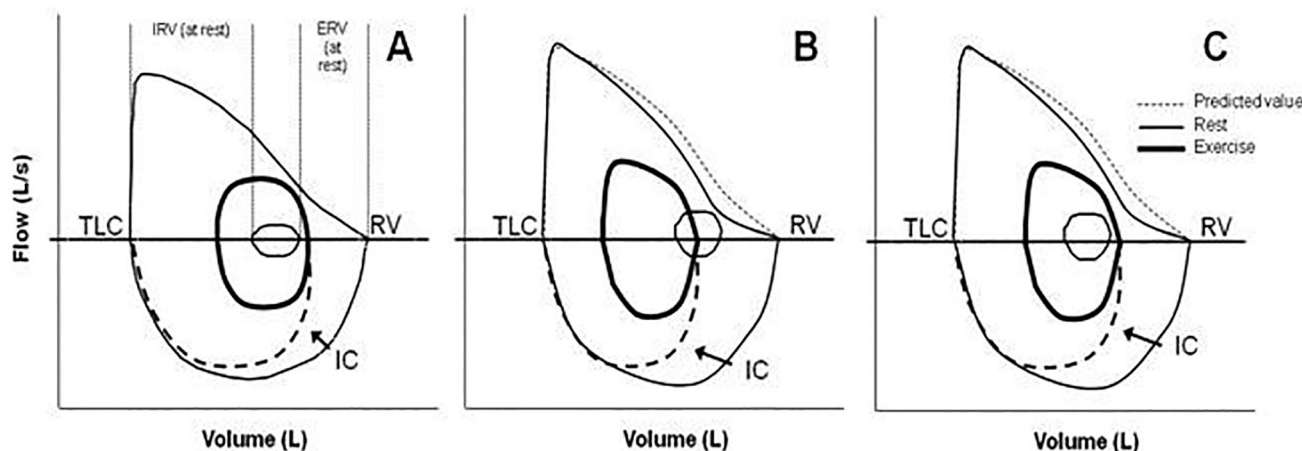
Exertional dyspnoea is a common complaint among obese individuals, but the mechanisms that drive breathlessness are not well defined. In severely obese individuals, it was demonstrated that, even during maximal exercise on a cycle ergometer, they were able to increase their ventilation enough to avoid hypercapnia [11]. There was no difference in peak exercise capacity, in terms of both peak work rate and oxygen consumption, and is normal in healthy obese individuals with and without exertional breathlessness. However, breathlessness was associated with increased ventilatory demand and greater increase in the oxygen cost of breathing during exercise. Breathlessness at rest may also be reported by obese individuals [11].

**Table 1 Effects of obesity on pulmonary functions at rest**

Respiratory compliance	↓
Small airway resistance	↑
Work of breathing	↑
Respiratory muscle strength	↓ ou ← →↔
FEV <sub>1</sub> /FVC	↑ ou ← →
FEV <sub>1</sub>	↓ ou ← →
FRC	↓
ERV	↓
TLC	↓ ou ← →
IC	↑

ERV, expiratory reserve volume; FEV<sub>1</sub>, forced expiratory volume in 1 s; FRC, functional residual capacity; FVC, forced vital capacity; IC, inspiratory capacity; TLC, total lung capacity.

**Fig. 1**



Flow volume curves in healthy and obese individuals and summary of the effects of obesity on pulmonary functions.

The respiratory mechanical factors related to obesity do not contribute to breathlessness in obese individuals. When obese and normal-weight women were compared during cycle exercise, it was found that, although oxygen consumption and minute ventilation were greater in obese individuals at all work rates, the relationships between breathlessness scores and both oxygen consumption and minute ventilation were no different in the obese and normal-weight individuals [18].

## Conclusion

Obesity results in decreased compliance and increased resistance of the respiratory system and in pulmonary dynamic hyperinflation secondary to small airway obstruction. These mechanical effects increase with the increase in BMI. The work of breathing is increased in obese patients, and the ventilatory requirements are higher during exercise due to higher metabolic cost. Certain mechanisms of adaptation help in decreasing the consequences of these mechanical effects; the higher IC at rest and the modification of the ventilatory behaviour lead to decrease in the elastic work of the respiratory muscles. However, obesity and especially severe obesity remain an important potential cause of dyspnoea, which is essentially associated with increased ventilatory demand during exercise.

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## Conflicts of interest

There are no conflicts of interest.

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