

PERIOPERATIVE RESPIRATORY PROBLEMS IN MORBIDLY OBESE PATIENTS & THEIR MANAGEMENT

Essay

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List of Abbreviations

1. **AHI:** Apnea-Hypopnea index.
2. **APACHE:** Acute physiology and chronic health evaluation.
3. **BiPAP:** Bi-level positive airway pressure.
4. **BMI:** Body mass index.
5. **CPAP:** Continuous positive airway pressure.
6. **CT:** Computed tomography.
7. **DVT:** Deep vein thrombosis.
8. **EEG:** Electroencephalogram.
9. **EMG:** Electromyogram.
10. **ERV:** Expiratory reserve volume.
11. **ETI:** Endo-tracheal intubation.
12. **ETT:** Endo-tracheal tube.
13. **F_EO₂:** Fractional oxygen concentration in expired gas.
14. **FEV₁:** Forced expiratory volume 1.
15. **F_IO₂:** Fractional oxygen concentration in inspired gas.
16. **FRC:** Functional residual capacity.
17. **FVC:** Forced vital capacity.
18. **GERD:** Gastro-esophageal reflux disease.
19. **HDL:** High-density lipoprotein.
20. **HPV:** Hypoxic pulmonary vasoconstriction.
21. **IBW:** Ideal body weight.
22. **ICU:** Intensive care unit.

- 23.**IVC**: Inferior vena cava.
- 24.**LDL**: Low-density lipoprotein.
- 25.**LVEDP**: Left ventricular end-diastolic pressure.
- 26.**MRI**: Magnetic resonance imaging.
- 27.**NAFLD**: Non-alcoholic fatty liver disease.
- 28.**NIV**: Non-invasive ventilation.
- 29.**OHS**: Obesity hypoventilation syndrome.
- 30.**OSA**: Obstructive sleep apnea.
- 31.**PACU**: Post-anesthesia care unit.
- 32.**P_AO₂**: Partial pressure of oxygen in alveolar gas.
- 33.**PaCO₂**: Arterial carbon dioxide partial pressure.
- 34.**PaO₂**: Arterial oxygen partial pressure
- 35.**PCV**: Pressure-controlled ventilation.
- 36.**PEEP**: Positive end-expiratory pressure.
- 37.**PSG**: Polysomnography.
- 38.**RDI**: Respiratory disturbance index.
- 39.**REE**: Resting energy expenditure.
- 40.**REM**: Rapid eye movement.
- 41.**RV**: Residual volume.
- 42.**RYGBP**: Raux-en-Y gastric bypass.
- 43.**TAI**: Total arousal index.
- 44.**TLC**: Total lung capacity.
- 45.**TTE**: Total energy expenditure.
- 46.**V^oCO₂**: Carbon dioxide production.

- 47.**VCM:** Vital capacity maneuver.
- 48.**VCV:** Volume-controlled ventilation.
- 49. **$\dot{V}_E$:** Expired volume per minute (Minute ventilation).
- 50. **$\dot{V}O_2$:** oxygen consumption.
- 51.**V/Q:** Ventilation-Perfusion ratio.
- 52.**WOB:** Work of breathing.

Chapter 1
Introduction

Morbid obesity and associated respiratory co-morbid conditions, such as obstructive sleep apnea or pulmonary hypertension, may create considerable challenge for the anesthetic management of patients and involve possible complications. A thorough understanding of the pathophysiology and the specific complications associated with the condition should allow more effective and safer treatment for this unique group of patients. **(Sprung J., 2007)**

Obesity increases the likelihood of perioperative hypoxia. The associated respiratory dysfunction is an important cause of postoperative morbidity and mortality. Complications from impaired pulmonary mechanics facilitate the development of pneumonia and respiratory failure and also augment the risk of obstructive sleep apnea, pulmonary hypertension and pulmonary embolism after surgery. **(Kaw et al., 2008)** Obese patients are more likely to be admitted to intensive care units. **Rose and colleagues (1994)** reported that acute postoperative pulmonary events were twice as likely in the obese as in the non-obese, and that hospitalized obese patients were at an increased risk of developing respiratory complications.

Weaning from mechanical ventilation may be difficult because of high oxygen requirement, reduced lung volumes, and ventilation–perfusion mismatching. Interest has emerged in using noninvasive ventilation (NIV) to facilitate earlier removal of the endotracheal tube and decrease complications associated with prolonged intubation. **(Burns et al., 2003)**

NIV represents a rebirth of an old tool for mechanical pulmonary assistance. It appears to be quite effective in providing support for patients with hypercapnic respiratory failure, with decreases in intubation (incidence and duration), complications, hospital stay and cost. **(Acton et al., 2002)**

Obesity can be defined as an excessive amount of body fat, which increases the risk of medical illness and premature death. **(Klein et al., 2002)** The difference between normality and obesity is arbitrary, but an individual must be considered obese when the amount of fat tissue is increased to such an extent that physical and mental health are affected, and life expectancy reduced. **(Hirsch et al., 1985)**

Accurate measurement of body fat content is difficult and requires sophisticated techniques such as computed tomography (CT) scanning or magnetic resonance imaging (MRI). Moreover, the determination of healthy and unhealthy amounts of fat mass is complicated. Usefull estimation, can be obtained by comparing the body weight with the ideal weight for a given height. **(Adams and Murphy, 2000)**

The ideal body weight (IBW) can be estimated by *Broca's Index* which is calculated as follows;

$$IBW \text{ (in kg)} = \text{height (in cm)} - x$$

Where x is 100 for adult males and 105 for adult females.

The body mass index (BMI) is a more robust measure of the relationship between height and weight, and is widely used in clinical and epidemiological studies. It is calculated as follows:

$$BMI = \text{body weight (in kg)} / \text{height}^2 \text{ (in meters)}.$$

(Adams and Murphy, 2000)

Although BMI is closely correlated with percent body fat mass (Gallgher et al. , 2000), some persons with an 'obese' BMI may have a normal amount of body fat and a large muscle mass, while others with 'normal' BMI may have excess adiposity and reduced muscle mass. (Klein et al., 2002)

Table 1; BMI-associated Disease Risk

	Obesity class	BMI (kg/m ²)	Risk
Underweight		<18.5	Increased
Normal		18.5-24.9	Normal
Overweight		25.0-29.9	Increased
Obesity	I	30.0-34.9	High
	II	35.0-39.9	Very High
Extreme obesity	III	>40.0	Extremely high

Pi-Sunyer et al., 1998

In general, the higher the BMI, the greater the risk of adiposity related diseases and premature mortality. However, other factors modify BMI-related risk. (Klein et al., 2002)

Fat distribution: There are 2 phenotypes of obesity according to fat distribution. In the central (android) type of distribution, which is

more common in males, fat is predominantly distributed in the upper body and is associated with increased deposition of intra-abdominal fat. In the peripheral (gynicoid) type, which is more common in females, fat is more typically distributed around the hips, buttocks or thighs. **(Ashwel et al., 1982)**

Central adipose tissue is metabolically more active than the peripheral one and is associated with more metabolic complication and greater risk from cardiovascular disease, the mechanism of this increased risk is unknown, but implicates delivery of the products of the breakdown of visceral fat directly into the portal circulation, inducing a significant secondary metabolic imbalance. **(Murphy, 2000)**

Practical assessment of fat distribution requires sophisticated imaging techniques, so the waist circumference or the ratio of waist to hip circumferences are useful clinical measures, as they correlate closely with abdominal fat mass **(Pouliot et al. , 1994)**. Men with a waist circumference greater than 102 cm or a waist:hip ratio greater than 1.0, and women with a waist circumference greater than 88 cm or a waist:hip ratio greater than 0.85 are at a higher risk. **(Pi-Sunyer et al., 1998)**

Weight gain: weight gain during adulthood is an additional risk factor for medical complications. Moreover, greater weight gain is associated with a greater risk of disease. **(Klein et al., 2002)**

Fitness: Aerobic fitness can modify the risk of developing diabetes or cardiovascular disease associated with obesity. It was found that across a range of body adiposity, those who were fit, defined by their maximal ability to consume oxygen during exercise, had a lower incidence of type 2 diabetes and cardiovascular mortality than those who were unfit. **(Klein et al., 2002)**

Ethnicity: For example, at the same BMI values, South-east Asian population has a higher risk of diabetes and cardiovascular disease than the white population. In contrast, Polynesians usually have more muscle mass and less body fat than white patients at the same BMI level. **(Inoue et al., 2000)**

Prevalence

The prevalence of obesity continues to rise in both developed and developing countries. On the Year 2000, According to a report from the World Health Organization, it was estimated that about 250 million adults worldwide — 7 % of the population — are obese and that at least 500 million adults are overweight. **(Bouchard, 2000)**

Pathogenesis

Energy imbalance:

Obesity is caused by more energy intake than is expended over a long period of time. The primary form in which potential chemical energy is stored in the body is fat (triglyceride). The amount of

triglyceride in adipose tissue is the cumulative sum over time of the differences between energy (food) intake and energy expenditure. Although homeostatic mechanisms keep this difference very low, very small imbalances over a long period can have a large cumulative effect. **(Rosenbaum et al., 1997)**

Daily total energy expenditure (TEE) consists of (1) resting energy expenditure (REE) (i.e., energy expended for normal cellular and organ function during postabsorptive resting conditions; approximately 70% of TEE); (2) thermic effect of food (i.e., the increase in energy expenditure associated with digestion, absorption, and increased sympathetic nervous system activity after eating a meal; approximately 10% of TEE); and (3) energy expended in physical activity. **(Klein et al., 2002)**

Contrary to popular belief, obese people have greater energy expenditure than thin people as it takes more energy to maintain their increased body size. Inactivity is usually the result, but not necessarily the cause of obesity. **(Adams & Murphy, 2000)**

Genetics and Environment

The genetic component of human obesity is complex and is likely to involve the interaction between multiple genes. **(Rankinen et al., 2002)** Some genetic defects lead to leptin deficiency and obesity; however, clinical studies suggest that only very rarely can such simple genetic defects in leptin production account for significant obesity.