Introduction

The use of volume-controlled ventilation (VCV) is common, as this has been the only available mode on ventilators for a long time. This mode utilizes a constant flow to deliver a target tidal volume (Vt) and thus insures a satisfactory minute ventilation (MV), despite frequently seen high-pressure levels in obese patients ***(Sprung et al., 2002).***

The traditional approach of using large tidal volumes in volume-controlled ventilation (VCV) during laparoscopic surgery in obese patients causes cardiovascular embarrassment, rise in peak inspiratory pressure and plateau pressure without significant improvement in arterial oxygenation. Moreover, high tidal volume promotes alveolar rupture, leading to volutrauma ***(Marino et al., 2007).***

On the contrary, the decelerating inspiratory flow used during pressure-controlled ventilation (PCV) generates high initial flow rate, causing more rapid alveolar inflation. This mechanical effect of PCV allows a homogeneous distribution of ventilation leading to better ventilation–perfusion matching. At the same time, pressure limits and

uniform distribution of forces within the lung reduce the risk of volu- and barotraumas. These characteristics of PCV tend to compensate for any potential reduction in ventilation caused by pressure limitation ***(Nichols and Haranath, 2007).***

Furthermore, the limitation of pressure levels has a positive effect on the patient's haemodynamics and might even reduce the risk of barotrauma. Owing to these theoretical advantages, and our clinical experience, we suggested that, in obese patients undergoing laparoscopy, PCV could provide sufficient MV, ensuring adequate CO2 removal and improved oxygenation, while using a lower plateau pressure than VCV ***(Prella et al., 2002).***

Aim of the Work

The aim of this study is to compare the efficacy and safety of pressure controlled ventilation versus volume controlled ventilation in laparoscopic gastric sleeve surgery.

Pathophysiology of Obesity

Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health, leading to reduced life expectancy and/or increased health problems ***(Mullen et al., 2008).***

The body mass index (BMI) has become the most widely applied classification tool used to assess individual weight status. BMI is defined as the patient's weight in kilograms, divided by the square of the patient's height in meters. Using this system, patients are classified according to BMI, and the associated risk of health problems developing is shown in (Table 1). Patients are considered to be overweight with a BMI between 25 and 29.9 kg/m^2 and obese with a BMI between 30 and 49.9 kg/m^2. The obese classification is further subdivided into class 1 (BMI range of 30 to 34.9 kg/m^2), class 2 (35 to 39.9 kg/m^22), and class 3 (40 to 49.9 kg/m^2). Patients with a BMI of 50 kg/m^2 or greater are considered superobese ***(Sinha and Eckmann, 2010).***

Some authors propose substituting the term morbid obesity for clinically significant obesity, defined by a BMI > 40 or a BMI > 35 with significant comorbidities ***(Lorentz et al., 2007).***

Table (1): Classification of Obesity and Levels of Risk Associated with Increasing Body Mass Index ***(Sinha and Eckmann, 2010).***

Classification	BMI (kg/m²)	Risk of Health Problems Developing
Underweight	<18.5	Increased
Normal weight	18.5-24.9	Least
Overweight	25.0-29.9	Increased
Obese		
Class 1	30.0-34.9	High
Class 2	35.0-39.9	Very high
Class 3	40.0-49.9	Extremely high
Superobese	≥50	Exceedingly high

Co-existing diseases:-

Morbid obesity is associated with progressive, serious co-morbidties such as type Π diabetes, hypertension, hyperlipidemia, accelerated atherosclerosis, arthritis of weight-bearing joints, breathing difficulties during sleep, gastro-esophegeal reflux disease, infertility, certain types of cancer, immobility and psychological problems. Virtually all organ systems can be included in the extended list of health risks associated with having an increased BMI. A listing of the most common specific disease associated with obesity is detailed in (Table 2) ***(Grant and Newcombe, 2004).***

Table (2): Systemic consequences of obesity ***(Grant and Newcombe, 2004).***

Cardiovascular system	Hypertension — systematic and pulmonary Atherosclerosis and hyperlipidemia Congestive cardiac failure Sudden cardiac death Coronary artery disease Peripheral vascular disease Chronic venous insufficiency
Respiratory system	Dyspnea and fatigue Altered physiology Sleep apnea Obesity hypoventilation syndrome Venous and pulmonary embolism
Gastrointestinal system	Hepatic steatosis Cholelithiasis Hernias
Obstetrics and gynecology	Female infertility Disrupted menstruation and ovulation Early menstruation Urinary incontinence Abnormal labour and increased progression to Caesarean section Increased fetal size Pre-eclampsia and eclampsia Gestational diabetes
Endocrine system	Diabetes mellitus Disorders or plasma cortisol and growth hormone Decreased levels of testosterone and increased levels of estradiol and estrogen in men
Oncology	Males: Increased risk of developing colorectal and prostate cancer Females: Higher mortality from gallbladder, cervical, endometrial, ovarian and breast cancer
Musculoskeletal system	Osteoarthritis Hyperuricaemia and gout
Psychiatry	Impaired body image depression
Metabolic syndrome	30% of middle-aged people in developed countries have features of metabolic syndrome

1-Obesity and Respiratory System:

A-Ventilation:

Lung volume:

Morbid obesity is associated with reduction in functional residual capacity (FRC), expiratory reserve volume and total lung capacity. These changes have been attributed to mass loading and splinting of the diaphragm. The FRC decreases with increasing BMI and may get tothe point that small airway closure occurs during normal tidal ventilation, generating a ventilation-to-perfusion mismatch, right-to-left shunting, and arterial hypoxemia. Assumption of the supine position further decreases the FRC. These factors impair the ability of obese patients to withstand long periods of apnea such as during laryngoscopy ***(Tantawy, 2008).***

Lung compliance and resistance:

Increasing BMI is associated with exponential decline in respiratory compliance. Although accumulation of fat tissue in and around the chest wall leads to modest reduction in chest wall compliance, recent work suggests that the decrease in total compliance is principally due to a decrease in lung compliance, this in turn is due to increased

pulmonary blood volume. Reduced compliance is associated with a decrease in FRC, encroachment on the closing volume and impairment of gas exchange ***(Sin et al., 2002).***

Morbid obesity is also associated with an increase in total respiratory resistance, mainly due to increase in lung resistance. This derangement of lung compliance and resistance results in a shallow and rapid pattern of breathing, increases the work of breathing and limits the maximum ventilatory capacity ***(Chlif et al., 2005).***

Work of breathing:

Obese patients have higher oxygen consumption and carbon dioxide production. In order to maintain normocapnia, they need higher minute ventilation.

The combination of increased mechanical pressure from the abdomen, reduction in pulmonary compliance and increase in the metabolic demands of the respiratory musculature result in respiratory muscle inefficiency and increase in the work of breathing ***(Tantawy, 2008).***

B-Obstructive sleep apnea (OSA):

Pathogenesis:

Apnea occurs when the pharyngeal airway collapses during sleep. Pharyngeal patency depends on the action of dilator muscles which prevent upper airway closure. This muscle tone is lost during sleep, and in many individuals, this leads to significant narrowing of the airway, causing turbulent airflow and snoring. Increased inspiratory effort and the response to hypoxia and hypercapnia lead to arousal which in turn restores upper airway tone. The patient then gasps, takes a few breaths and falls asleep again, the cycle then restarts ***(Parish and Somers, 2004).***

Approximately 5% of obese patients will have obstructive sleep apnea which is characterized by the following features:

i- Frequent episodes of apnea or hypopnea during sleep: an obstructive apneic episode is defined as 10 seconds or more of total cessation of airflow despite continuous respiratory effort against a closed airway, hypopnea is defined as 50% reduction in airflow. The number of episodes thought to be clinically significant is often quoted as five or more per hour or >30 per night.

ii- Snoring: which gets louder as the airway obstructs followed by silence as the airflow ceases then gasping as the person rouses and airway patency is restored.

iii- Daytime Symptoms: repeated episodes of fragmented sleep throughout the night causes daytime sleepiness, which is associated with impaired concentration and memory problems.

iv- Physiolgical Changes: recurrent apnea leads to hypoxemia, hypercapnia and pulmonary and systemic vasoconstriction. Recurrent hypoxemia leads to secondary polycythemia and is associated with increased risk of ischemic heart disease and cerebrovascular disease, while hypoxic pulmonary vasoconstriction leads to right ventricular failure.

(Resta et al., 2001)

Evaluation of obstructive sleep apnea in obesity:

Nocturnal polysomnography is the "gold standard" diagnostic test for sleep apnea. This test allows the identification of complete cessation of airflow (apnea) and reduction of airflow associated with a decrease in oxygen saturation and arousal (hypopnea) or both. Diagnosis of obstructive sleep apnea is made when symptomatic patients have an apnea–hypopnea index (the number of episodes of

apnea and hypopnea per hour of total sleep time) greater than five (***Kheterpal et al., 2006).***

However, nocturnal polysomnography is time consuming, costly and of limited availability (***Ross et al., 2000).***Therefore, other diagnostic strategies such as nocturnal oximetry or cardiorespiratory monitoring (which are considered first-line tests for patients with high pretest propability of sleep apnea).

Sequelae of OSA:

Possible complications include arrhythmias, myocardial ischemia, cerebrovascular insufficiency, mental dysfunction and poor wound healing. Chronically, if the sleep apnea is severe enough, respiratory and right-side heart failure may develop as the result of persistant severe hypoxemia and hypercapnia ***(Yaggi et al., 2005).***

C-Obesity Hypoventilation Syndrome:

Obesity hypoventilation syndrome was classically described as "Pickwickian syndrome" in 1956, case report by Burwell.

Symptoms suggestive of the diagnosis may include features of sleep apnea (witnessed apnea, snoring, chronic mouth breathing, daytime sleepiness, morning headaches,

decreased exercise tolerance, poor school performance, poor memory and poor concentration).

The condition has been poorly defined in the past and often confused with obstructive sleep apnea. Differentiation has been made between people suffering from sleep apnea and Pickwickian syndrome in which hypoventilation is also evident whilst they are awake. Most patients with the syndrome also have sleep apnea but some patients do not, suggesting that it is the obesity *per se* which is causing chronic hypoventilation ***(Loadsman and Hillman, 2001).***

Diagnosis:

Pickwickian syndrome cannot be diagnosed on history and examination alone but requires the demonstration of daytime hypercapnea.

Diagnostic criteria for Pickwickian syndrome:

- Body Mass Index $\geq$30 kg/m^2.
- Daytime $PaCO_2$>45 mmHg.
- Associated sleep-related breathing disorder (sleep apnea-hypopnea syndrome or sleep-hypoventilation or both).
- Absence of other known causes of hypoventilation.

(Kral, 2001)

2-Obesity and Cardiovascular Disease:

Cardiovascular disease dominates the morbidity and mortality in obesity and manifest itself in the form of ischemic heart disease, hypertension, arrhythmias and heart failure.

A-Hypertension:

Mild to moderate hypertension is seen in 50-60% of obese patients and severe hypertension in 5-10%. An expansion of extra cellular volume resulting in hypervolemia and an increase in cardiac output are characteristic of obesity-induced hypertension ***(Tantawy, 2008)***.

The exact mechanism for hypertension in obese is unknown, and probably represents an interaction between genetic, hormonal, renal and hemodynamic factors. Hyper-insulinemia which is characteristic of obesity, can contribute by activating sympathetic nervous system and by causing sodium retention. In addition, insulin resistance may be responsible for enhancement in pressor activity of norepinephrine and angiotensin II ***(Tantawy, 2008).***

Hypertension per se leads to concentric left ventricular hypertrophy and progressively non-compliant left ventricle

which, when added to the increase blood volume, increase risk of cardiac failure ***(Poirier and Eckel, 2002)*.**

B-Ischemic Heart Disease:

It is now generally accepted that obesity is an independent risk factor for ischemic heart disease and is more common in those obese individuals with a central distribution of fat. Other factors such as hypertension, diabetes mellitus hypercholesterolemia and reduced high density lipoprotein, which are all common in obese, will compound the problem. Interestingly, 40% of obese patients with angina do not have demonstrable coronary heart disease in other words, angina may be a direct symptom of obesity ***(Poirier et al., 2009).***

C-Congestive Heart Failure:

The morbidly obese individual is at risk of a specific form of obesity induced cardiac dysfunction, the belief that this is secondary to fatty infiltration of the heart is no longer valid.

Obesity is associated with an increased blood volume and cardiac output, the increase in cardiac output is a result of ventricular dilation and an increase in stroke volume. The ventricular dilation results in increased left ventricular wall stress, leading to hypertrophy. Such eccentric left

ventricular hypertrophy results in reduced compliance and left ventricular diastolic dysfunction, i.e: impairment of ventricular filling, leading to elevated left ventricular end diastolic pressure and pulmonary edema ***(Wilborn et al., 2005).***

The problem is often compounded by superimposed hypertension and ischemic heart disease. Ventricular hypertrophy and dysfunction worsen with increased duration of obesity and improve to some extent with weight loss.

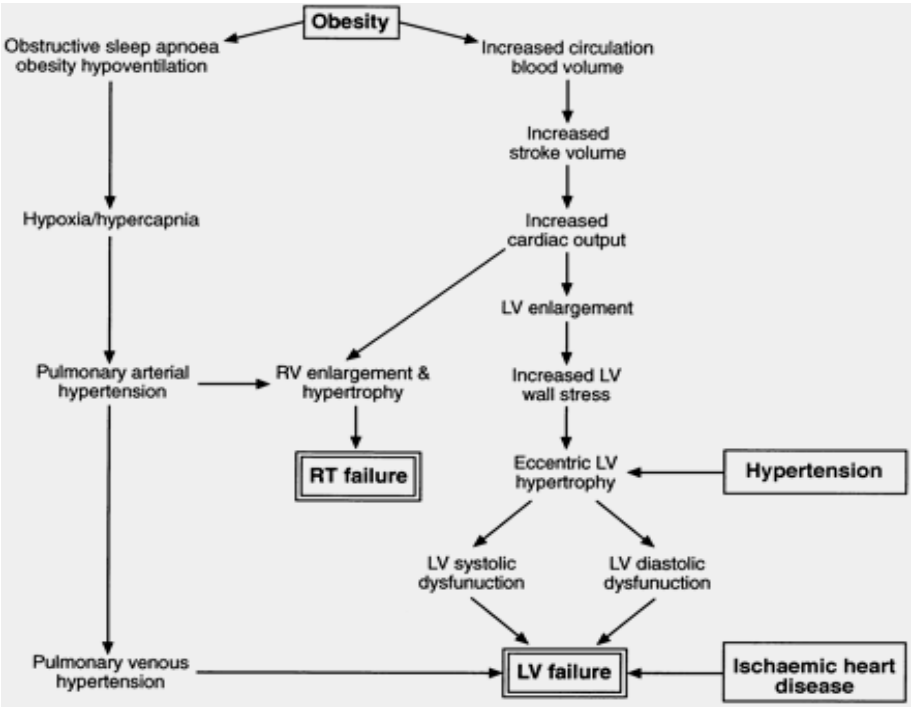

Figure (1): Obesity induced cardiomyopathy (RV right ventricle, RT right, LV left ventricle) ***(Tantawy, 2008).***