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# Perioperative management of patient undergoing bariatric surgery

**An Essay**
Submitted for Partial Fulfillment of Master Degree in Anesthesia

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# Contents

# List of Abbreviations

| | | |
|---|---|---|
| AaDO2 | : | Alveolar-arterial oxygen partial pressure |
| AF | : | Atrial fibrillation |
| AHI | : | Apnea hypopnea index |
| AI | : | Apnea index |
| AV | : | Abdominal visceral fat |
| BMI | : | Body mass index |
| BP | : | Blood pressure |
| BV | : | Blood volume |
| CAD | : | Coronary artery disease |
| CHD | : | Coronary heart disease |
| CHF | : | Congestive heart failure |
| CO | : | Cardiac output |
| CPAP | : | Continuos positive airway pressure |
| DO2 | : | Oxygen delivery |
| ERV | : | Expiratory reserve volume |
| FEV1 | : | Forced expiratory volume in first second |
| FFA | : | Free fatty acid |
| FFM | : | Fat free mass |
| FRC | : | Functional residual capacity |
| HI | : | Hypopnea index |
| IBW | : | Ideal body weight |
| IL-6 | : | Interleukin 6 |
| IR | : | Insulin resistance |
| LAGB | : | Laparoscopic adjustable gastric banding |
| LBW | : | Lean body weight |
| LVEF | : | Left ventricular ejection fraction |
| LVH | : | Left ventricular hypertrophy |
| LVM | : | Left ventricular mass |
| MO | : | Morbid obese |

# List of Abbreviations (Cont.)

| | | |
|---|---|---|
| MVO2 | : | Myocardial oxygen consumption |
| NASH | : | Non alcoholic steato-hepatitis |
| NDNMBS | : | Non depolarizing neuromuscular blockers |
| NO | : | Nitric oxide |
| NREM | : | Non rapid eye movement |
| OHS | : | Obesity hypoventilation syndrome |
| OSA | : | Obstructive sleep apnea |
| OS-MRS | : | Obesity surgery mortality risk score |
| PACU | : | Post anaesthetic care unit |
| PE | : | Pulmonary embolism |
| PH | : | Pulmonary hypertension |
| PONV | : | Postoperative nausea and vomiting |
| PSG | : | Polysmnography |
| REM | : | Rapid eye movement |
| RYGB | : | Roux en Y gastric bypass |
| SaO2 | : | Oxygen saturation |
| SV | : | Stroke volume |
| T2D | : | Type 2 diabetes |
| TBV | : | Total blood volume |
| TBW | : | Total body weight |
| TLC | : | Total lung capacity |
| TNF | : | Tumor necrosis factor |
| TOF | : | Train of four |
| VO2 | : | Global oxygen consumption |
| VTE | : | Venous thromboembolism |

# List of Figures

# List of Tables

# Introduction

Obesity classified as a major health problem with a mild to severe health implications which include an increased risk for coronary artery disease, hypertension, dyslipidemia, diabetes mellitus, gall bladder disease, degenerative joint disease, sleep disorders, socioeconomic and psychosocial impairment **(Tank and Gohil, 2011).**

Implication for anaesthetic and perioperative care of severly obese patients are various, induction of anaesthesia is associated with potential risks as difficult airway, pulmonary aspiration of gastric contents, periods of hypoxia and hypercapnia, perioperatively may increase pulmonary vascular resistance and precipitate acute right sided heart failure especially in patients with pre-existing cardiac disease **(Brodsky *et al.*, 2001).**

The limited information available concerning pharmacodynamics and pharmacokienetics of peri-operatively administered medications in severe obesity make a challenge to proper drug dosing in those patients and to manage those patints intraoperative with their per-existing diseases and to control and avoid complications postoperatively especially with co-morbidities in those patients **(Ertatad, 2003).**

Bariatric surgery provides significant and sustained weight loss option for morbidly obese patients which improves the quality of the life, surgeries are classified into

many categories as example: gastric banding, gastric bypass, gastroplasty that can be done open or laparoscopic **(Buchwald and Buchwald, 2003).**

# Aim of The Essay

The aim of this essay is to highlighten the perioperative anaesthetic management of bariatric surgery patient and to anticipate and avoid postoperative complications.

# Pathophysiology

Obesity is described as an elevated level of fat storage in the form of hypertrophy and or hyperplasia of fat cells, known as adipocytes. Given the complexities of body composition analysis, the body mass index act as a surrogate for the amount of fat in the body.

Body mass index is defined as the body weight in kg divided by the square of the body height in meters, Obesity has been defined as BMI more than 30 $kg/m^2$, and more than 35 $kg/m^2$ obesity related comorbidity and more than 40 $kg/m^2$ is called morbid obesity.

The obesity is associated with cardiovascular deterioration, which include excess abdominal visceral fat (AVF), atherogenic, dyslipidemia, hypertension, insulin resistance, hyperglycemia, and a pro-thrombotic and pro-inflammatory state.Abdominal visceral fat should be considered a paracrine organ because it secretes hormones and inflammatory bio-active peptides, collectively known as adipokines (adipocytokines) **(Despres *et al.*, 2008).**

At ideal BMI adipokines have purely beneficial effects on metabolism, cardiac function and vascular endothelial well-being. However, as AVF increases in volume, excessive amounts of these hormones and inflammatory adipokines have detrimental effects on a variety of organ systems, including the heart and circulation Independent of hypertension, obstructive sleep apnea (OSA), coronary artery disease, and type 2

diabetes, excess AVF causes metabolic and structural changes of the heart and peripheral circulation, Hemodynamically this "cardio-metabolic" syndrome in its early stages is characterized by increased blood volume (BV), stroke volume (SV), cardiac output (CO), and preserved indices of global systolic pump performance, the most recognized being left ventricular ejection fraction (LVEF). The primary causes of obesity cardiomyopathy are myocardial fibrosis, fatty infiltration of the myocardium and the inappropriate accumulation of free fatty acids (FFA) and neutral lipids within the cardiomyocytes, Through the process of lipotoxicity, lipid overload causes cellular dysfunction, cell death, and ultimately clinically relevant cardiac dysfunction, If untreated, this eventually results in congestive heart failure and premature death **(Peeters *et al.*, 2003).**

## Adipokines:

Traditionally, adipose tissue has been perceived as an inert organ whose sole function is as a repository for storing excess energy in the form of triglycerides, It is now known that AVF secretes hormones beneficial to metabolic well-being, namely leptin, adiponectin, and the inflammatory adipokines **(Hajer *et al.*, 2008).**

## 1. Leptin:

In lean individuals, leptin is known to act on the satiety area of the hypothalamus to modulate and suppress appetite. Increased leptin levels have a direct effect on blood pressure (BP), cardiac remodeling, myocyte contraction, and may be a

cause of left ventricular hypertrophy (LVH), insulin resistance, hyperinsulinemia, hyperglycemia and diabetic dyslipidemia **(Perego *et al.*, 2005).**

## 2. Adiponectin:

Is an adipocyte-produced hormone thatis totally cardioprotective which improves insulin sensitivity, decreases non-esterified fatty acid uptake, and reduces gluconeogenesis. In skeletal muscle, adiponectin stimulates both glucose and fatty acid use and antiatherosclerotic effects and protects against endothelial dysfunction by increasing local nitric oxide (NO) production, protects cells from inflammation that result from high glucose levels or tumor necrosis , factor alpha (TNF-α), while it also inhibits plaque initiation, progression, rupture and thrombosis **(Harmancy *et al*., 2008).**

## 3. Inflammatory adipokines:

Inflammation, which is normally mediated by inflammatory adipokines, is a protective response of tissue that eliminates noxious agents and debris, and is closely tied to tissue repair, however, inflammatory adipokines, through enhanced synthesis of substances such as the potent vasoconstrictor endothelin-1, are responsible for decreased production or availability Of NO. This results in small vessel disease, including endothelial dysfunction with attendant dysregulation of vascular tone and vasomotor function **(Zhang, 2008).**

## 4. Angiotensinogen:

Angiotensinogen, a precursor of angiotensin II, is mainly produced in the liver, but with increasing levels of obesity, angiotensinogen is produced in excess by visceral adipocytes. Excessive production of angiotensin II provides one of several etiologies causing obesity-induced pressure-overload hypertension with attendant LVH, as well as direct toxicity to cardiac myocytes and vascular endothelium (**Zhang, 2008**).

## 5. Plasminogen activator inhibitor-1:

Reduces fibrinolytic activity and inducesa pro-thrombotic, hypercoagulable state, predisposes to platelet aggregation and adhesion, clot formation, venous thrombosis and pulmonary thromboembolism (**Hajer *et al.*, *2008***).

## 6. Resistin:

Alters cardiac contractility and induces cardiomyocyte hypertrophy and depresses cell contractility, as well as contractionand relaxation velocity.

## 7. Tumor necrosis factor-*α*:

Appear early in the metabolic syndrome and correlate highly with insulin resistance. Which decrease the produced NO, which leads to coronary artery and peripheral vascular endothelial dysfunction **(Hajer *et al.*, 2008**).

## 8. Interleukin-6:

Elevated levels of IL-6 predict the development of type 2 diabetes and myocardial infarction. Increased IL-6 levels are directly correlated with increased plasma FFAs and may also decrease insulin sensitivity by depressing adiponectin secretion.

## Pathophysiological changes in cardiovascular system:

## Global oxygen consumption[VO2]:

Progressive increases in AVF, and especially fat-free mass (FFM) elicit an increased demand for metabolic oxygen. At rest, total body oxygen consumption in the obese and MO exceeds that of individual of ideal BMI. Maintenance or augmentation of CO, hemoglobin concentration and arterial blood oxygen saturation (SaO2) in the peri-operative period is important in order to optimize oxygen delivery (DO2). Optimization of DO2 to meet VO2 prevents anaerobic metabolism, the consequences of which are metabolic acidosis and depression of myocardial contractility. If CO fails to increase with physiologic stress, such as in severe anemia and/or hypovolemia, oxygen transport becomes inadequate, leading to anerobic metabolism with lactic acidosis **(Seres *et al.*, 2003).**

## Blood volume:

Total blood volume (TBV) progressively increases over the full range of BMI, , The increase in plasma volume,