

Airway Management In Morbidly Obese Patient

Essay

**Submitted for partial fulfillment
of the Master Degree in Anaesthesia**

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2013

كيفية التعامل مع المجرى الهوائي للمرضى مفرطي السممه

رسالة مقدمة من

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**بكالوريوس الطب و الجراحة
توطئة للحصول على درجة الماجستير فى التخدير**

تحت إشراف

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2013

CONTENTS

	Page
Introduction	1
Chapter 1: Anatomical and physiological considerations of airway in morbidly obese patient.....	4
Chapter 2: Airway management of morbidly obese patient	14
Preanaesthetic evaluation	16
Positioning of the patient	16
Preoxygenation	18
Mask ventilation, tracheal intubation and intubating device	20
Post tracheal intubation management	32
Effect of different positions on patient ventilation	35
Extubation	38
Chapter 3: Airway management for morbidly obese patient undergoing ambulatory surgery.....	41
Preoperative assessment and preparation	47

CONTENTS (Cont.,)

Page

Intraoperative management	48
Chapter 4: Post-operative care.....	51
Summary.....	55
References.....	58
Arabic Summary	

List of Figures

Figure No.	Title	Page
1	Effect of obesity on lung volumes	8
2	A morbidly obese patient placed in the ‘sniff’ position .	18
3	A morbidly obese patient placed in ‘ramped’ position	18
4	The ASA difficult airway algorithm	22
5	Flexible fiber-optic bronchoscope	26
6	GlideScope	26
7	Optical stylet	29
8	Optical stylet.	29
9	The laryngeal mask airway	30
10	The esophageal tracheal combitube.	31

List of Abbreviations

Abb.	Full name
ASA	American Society of Anesthesiologists.
BiPAP	Bilevel Positive Airway Pressure.
BMI	Body Mass Index.
CPAP	Continuous Positive Airway Pressure.
CT	Computerized Tomography.
DMV	Difficult Mask Ventilation .
ERV	Expiratory Reserve Volume.
ETC	Esophageal Tracheal Combitube.
FEV1	Forced Expiratory Volume in One Second .
FRC	Functional Residual Capacity.
IC	Inspiratory Capacity .
ILMA	Intubating Laryngeal Airway Mask.
LMA	Laryngeal Mask Airway.
MLSD	Measurement of Liver and Spleen Displacement.
MRI	Magnetic Resonance Imaging.

List of Abbreviations_(cont.,)

Abb.	Full name
MVV	Maximum Voluntary Ventilation .
NMDA	N_Methyl D Aspartate.
OSA	Obstructive Sleep Apnea.
PaCO₂	Arterial Pressure of Carbon Dioxide.
RSI	Rapid Sequence Induction.
RV	Residual Volume .
SpaO₂	Arterial Oxygen Saturation.
TLC	Total Lung Capacity.
VC	Vital Capacity.
V/Q	Ventilation/Perfusion Ratio.



ACKNOWLEDGEMENT

First of all, I want to thank our **HollyGod** almighty for giving me the strength and motivation to pursue this degree.

I am heartily thankful to my great supervisor, **Professor Hoda Omar**, whose encouragement, guidance and support enabled me to finish this subject.

I offer my regards to **Doctor Mohamad El Shafie** who provided me with the support and valuable information I needed to complete this subject.

I give many great thanks to **Doctor Walid Nofal**, who provided me with the new papers and latest available information about my work, and for the meticulous revision of this work.

I also want to thank my wife who gave me help and advice during the completion of the subject.

Lastly, I offer many thanks for my family, God bless them all.

INTRODUCTION

Obesity is defined as the degree of excess weight associated with adverse health consequences. It is estimated that more than 30% of the adult population is obese, and this number seems to be increasing. The World Health Organization defines overweight as a body mass index (BMI) of 25 kg/m² or higher, obesity as a BMI of 30 kg/m² or higher, and extreme (morbid) obesity as a BMI of 40 kg/m² or higher (*Bessesen, 2008*).

Any patient can have a difficult airway, but obese patients have anatomic and physiologic features that can make airway management particularly challenging. Changes in respiratory mechanics, such as a decreased functional residual volume, may result in rapid onset of hypoxemia in the obese patient. Excess soft tissue around the neck and in the oropharynx can make mask ventilation difficult or impossible. Increased intra-abdominal pressure from abdominal adiposity increases the risk of

regurgitation and aspiration. To effectively manage airways in obese patients, anaesthesiologists need to be skilled in mask ventilation, direct laryngoscopy and need to know how to safely manage a difficult airway situation, and should be familiar with the American Society of Anesthesiology's Difficult Airway Algorithm (*Langeron et al, 2006*).

One of the major co-morbidities associated with obesity includes obstructive sleep apnea (OSA), which is associated with hypertension, stroke, depression & diabetes. It is estimated that 60%-80% of people with OSA are obese. Of note, the prevalence of OSA is higher in the surgical population than that reported in the general population. With approximately 60%-70% of all surgical procedures performed on an outpatient basis, it is inevitable that anesthesiologists will encounter morbidly obese patients with OSA in an ambulatory setting (*Eastwood et al, 2010*).

Obese patients are more in danger of becoming hypoxic or developing airway obstruction when left

undisturbed in the recovery area. It has been suggested that most significant postoperative complications in obese patients usually occur within 2 hours after surgery. Therefore, it may be worthwhile to observe these patients in the recovery room for at least 2 hours (*De Freitas carvalho et al, 2006*).

Finally, knowing how to quickly evaluate an airway and when to call for help, either before or after the difficulty is encountered, are critical skills and judgments that must be acquired by anyone dealing with the airway (*William, 2010*).

As Obesity is the most common metabolic disease in the world and its prevalence has risen worldwide, all anaesthiologists should have enough informations about anatomic and physiologic features of obese patients airway. Obese patients compared to none obese ones have anatomic and physiologic features that can make airway management particularly challenging. Excess soft tissue around the neck and in the oropharynx can make mask ventilation difficult or impossible. Increased intra-abdominal pressure from abdominal adiposity increases the risk of regurgitation and aspiration. (*Langeron, 2006*).

They can be attributed in part to the effect of obesity on the respiratory system. Obesity affects various resting respiratory physiologic parameters such as lung volumes, spirometric measures, compliance, neuromuscular strength, and work of breathing & respiratory resistance. Changes in respiratory mechanics, such as a decreased functional residual volume, may result in rapid onset of hypoxemia in the obese patient. Diminished total lung capacity and vital capacity in obesity result from decreased chest wall

compliance and increased abdominal cavity contents (*Kuchta, 2005*).

The most common and consistent indicator of obesity is a reduction in expiratory reserve volume (ERV). This occurs because of displacement of the diaphragm into the thorax by the obese abdomen and increased chest wall mass. Although this association is seen even with modest obesity, ERV decreases rapidly in an exponential relationship with increase in body mass index (BMI). On the other hand, obesity has fairly modest effects on the extremes of lung volumes at residual volume (RV) and total lung capacity (TLC) but a relatively larger effect in reducing functional residual capacity (FRC). This reduction is often so marked that FRC approaches RV. When the reduced FRC is equal to or lower than the closing volume, regional thoracic gas trapping may take place in obese subjects, as suggested by an elevated RV/TLC ratio. Further, in order to compensate for the reduced FRC, inspiratory capacity (IC) may be increased in severe obesity. As mentioned previously, TLC is usually preserved

in most obese subjects, other than those with morbid obesity (weight-to-height ratio of ≥ 0.9 kg/cm), with excessive central adiposity (waist-to-hip ratio of ≥ 0.95), or with obesity hypoventilation syndrome (*Jones, 2006*).

Sequential studies after weight loss, usually in the context of bariatric surgery, usually shows a marked improvement in ERV intermediate improvement in RV and FRC with a more modest improvement in TLC (*Sutherland, 2008*).

Obesity may be associated with a reduction in vital capacity (VC) and forced expiratory volume in one second (FEV1), depending upon the age, type of body fat distribution (with central fat distribution having a relatively greater effect) , and severity of obesity. Possible causes of reduced VC in obese subjects may be mechanical and inflammatory (**Fig 1**). Mechanical causes include decreased respiratory compliance (with consequently decreased lung volumes) and increased gas trapping from premature small airway closure (particularly at the lung bases). In addition, obesity is associated with both increased levels of

proinflammatory adipokines (such as leptin, interleukin-6, and tumor necrosis factor-alpha) and decreased levels of anti-inflammatory adipokines (such as adiponectin). The secretion of these adipokines by adipose tissue in chronic respiratory diseases may be regulated by chronic or intermittent hypoxia. These adipokines in turn regulate systemic inflammation which is associated with impaired lung function. Either directly or via systemic inflammation, adipokines may also affect inflammation of small airways, resulting in premature closure of the inflamed and edematous small airway (*Leone, 2009*).

Interestingly, while increased fat mass may be negatively associated with spirometric lung function, increased lean mass (*i.e.* primarily muscle mass) may be positively associated with FEV1 and to a lesser extent with VC, particularly among men. This protective effect of lean mass on lung function may be associated with stronger respiratory musculature or larger overall thoracic size, although the mechanism remains uncertain. In addition to the above spirometric changes, severe obesity is associated