Faculty of Medicine

Management of Postoperative Pulmonary Complications

An Essay
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in Anesthesia

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List of abbreviations

ABG	:	Arterial blood gases
ACS	:	Abdominal compartment syndrome
AERD	:	Aspirin exacerbated respiratory disease
ALI	:	Acute lung injury
APP	:	Abdominal perfusion pressure
ARDS	:	Acute respiratory distress syndrome
ASA	:	American society of anesthiologists
BAL	:	Broncho alveolar lavage
Bi-PAP	:	Bilevel-positive airway pressure
BMI	:	Body mass index
CABG	:	Coronary artery bypass graft
CHF	:	Congestive heart failure
COPD	:	Chronic obstructive pulmonary disease
CPAP	:	Continuous positive airway pressure
CPB	:	Cardio-pulmonary bypass
CPET	:	Cardio-pulmonary exercise testing
CRP	:	C-Reactive protein
DVT	:	Deep venous thrombosis
ECG	:	Electrocardiography
FET	:	Forced expiratory time
FEV1	:	Forced expiratory volume1
F_IO_2	:	Fractional inspired oxygen
FRC	:	Functional residual capacity
FVC	:	Forced vital capacity
HAP	:	Hospital acquired pneumonia
IAH	:	Intraabdominal hypertension

List of abbreviations (Cont.)

IAP	:	Intraabdominal pressure
ICP	:	Intracranial pressure
IPPB	:	Intermittent positive pressure breathing
MDI	:	Metered dose inhaler
MDR	:	Multi- drug resistant
MRSA	:	Methicillin resistant staph.aureus
MSSA	:	Methicillin sensitive staph.aureus
NGT	:	Nasogastric tube
NMBA	:	Neuromuscular blocking agents
NPPE	:	Negative pressure pulmonary edema
$NSAID_S$	:	Non-steroidal anti-inflammatory drugs
OSA	:	Obstructive sleep apnea
PEEP	:	Positive end expiratory pressure
PEFR	:	Peak expiratory flow rate
PFT_S	:	Pulmonary function tests
PPCS	:	Post pericardiotomy syndrome
PPCS	:	Postoperative pulmonary complications
PCT	:	Procalcitonin
REM	:	Rapid eye movement
UAO	:	Upper airway obstruction
VA/Q	:	Ventilation/perfusion
VAP	:	Ventilator associated pneumonia
VCM	:	Vital capacity maneuver

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Introduction

Postoperative pulmonary complications contribute significantly to overall perioperative morbidity and mortality rates. Such complications account for about 25% of deaths occurring within 6 days of surgery. The frequency rate of these complications varies from 5-70% ***(McAlister et al., 2005).***

Postoperative pulmonary complications include atelectasis, bronchospasm, pneumonia, and exacerbation of chronic lung disease. However, the list can be expanded to include acute upper airway obstruction, complications from obstructive sleep apnea, pleural effusions, chemical pneumonitis, pulmonary edema, hypoxemia due to abdominal compartment syndrome and tracheal laceration ***(Fisher et al., 2002).***

The risk of postoperative pulmonary complications varies with the type of surgery being performed. Pulmonary complications occur much more in patients undergoing elective surgery to the thorax and upper abdomen. Operations at sites farther from the diaphragm are associated with a much lower incidence of postoperative pulmonary complications. Postoperative pulmonary complications are also more common in patients with preexisting lung and cardiac diseases, poor nutritional status, overall poor health, obesity and in those who smoke ***(Arozullah et al., 2003).***

Treatment to reduce the risk of postoperative pulmonary complications begins prior to surgery. Potential preoperative strategies include cigarette cessation, optimization of underlying chronic lung disease, and patient education. Antibiotics may be indicated for patients with lower respiratory tract infection ***(Wong et al., 2012).***

The selection of the type of anesthesia and neuromuscular blockade both affect the incidence of postoperative pulmonary complications. Briefer, lower risk procedures should be used whenever possible in high risk patients ***(Hausman et al., 2015).***

Aim of the Essay

The aim of this work is to review different postoperative pulmonary complications & risk factors predisposing of them and to discuss various management &prevention strategies for such complications.

Chapter 1

Overview of postoperative pulmonary complications

Pulmonary complications are major causes of morbidity and mortality during the postoperative period ***(Lawrence et al., 2006).***

Postoperative pulmonary complications include atelectasis, bronchospasm, pneumonia and exacerbation of chronic lung disease. However, the list can be expanded to include acute upper airway obstruction, complications from obstructive sleep apnea, pleural effusions, chemical pneumonitis, pulmonary edema, hypoxemia due to abdominal compartment syndrome and tracheal laceration ***(Fisher et al., 2002)***.

1-Postoperative Atelectasis:-

Atelectasis is defined as complete or partial collapse of a lung or lobe of a lung when the alveoli within the lung become deflated ***(Reinius et al., 2009).***

Atelectasis occur in the most dependent parts of the lungs and are found in almost 90% of all patients who are anesthetized. They develop with both i.v. and inhalational anesthesia and whether the patient is breathing spontaneously or is paralysed and ventilated mechanically. On the contrary, epidural anesthesia caused no or little atelectasis and no change in shunting, ventilation/perfusion (VA/Q) matching or oxygenation ***(Magnusson and Spahn, 2003).***

Most atelectasis occurs near the diaphragm in the supine patient and less towards the apex **(Figure 1)** ***(Magnusson and Spahn, 2003).***

Fig.1: Two-dimensional representation of a volume image from an anesthetized subject. The surface of the lung is shown in shades of grey and atelectasis is shown in white. The anteroposterior view (top) shows the right and left lungs, with a visible cardiac shadow (arrow). The lateral view (bottom) shows the atelectatic regions in the most dependent lung ***(Magnusson and Spahn, 2003).***

Atelectasis can persist for two days after major surgery but disappears within 24 hrs after laparoscopy in non-obese patients ***(Eichenberger et al., 2002).***

Causes of atelectasis formation during general anaesthesia:-

1-Compression atelectasis:-

Occurs when the transmural pressure distending the alveolus is reduced. Compression atelectasis occurs during general anesthesia and is caused by chest geometry and diaphragm position and motion. General anesthesia induces a cephalad displacement of the most dorsal part of the diaphragm. In the supine position during spontaneous ventilation, the dependent part of the diaphragm had the greatest displacement. However, after neuromuscular block and positive pressure ventilation, exactly the opposite was seen: the non-dependent part had the greatest displacement **(Figure 2)** ***(Magnusson and Spahn, 2003).***

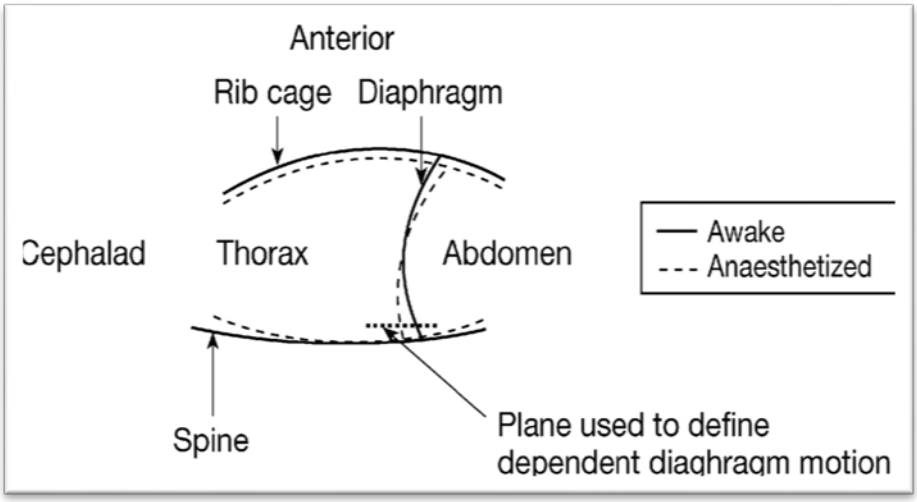

Fig.2: Diagram of a midsagittal section of the thorax while awake (solid lines) and while anaesthetized (dashed lines). Note the alteration in the position of the diaphragm ***(Magnusson and Spahn, 2003).***

2-Absorption atelectasis:-

Absorption atelectasis can occur by two mechanisms :-

The first mechanism is complete airway occlusion, which creates a pocket of trapped gas in the distal lung unit. The pressure in the pocket initially is close to atmospheric pressure. Mixed venous blood continues to perfuse the pocket, and since the sum of the gas partial pressures in the mixed venous blood is subatmospheric, gas uptake from the pocket by the blood continues and the pocket collapses ***(Edmark et al., 2001).***

The second mechanism is when the inspired VA/Q ratio is less than a critical value. If the inspired VA/Q ratio of a lung unit is reduced, a point is reached where the rate at which inspired gas entering the alveolus is exactly balanced by gas uptake from the alveolus into the blood. This point is known as the critical VA/Q ratio. If the inspired VA/Q ratio is less than this, the lung unit will collapse. This is likely when F_IO_2 is high and the gas uptake is large. Conversely, a reduction in the amount of atelectasis is seen when lower concentrations of oxygen are used at induction, during maintenance of general anesthesia or just before extubation ***(Edmark et al., 2001).***

3-Loss of surfactant atelectasis:-

Occurs when the surface tension of an alveolus increases because of reduced surfactant action. Recurrence of atelectasis within 5 min after a vital capacity manoeuvre (VCM) at F_IO_2 =1.0 or immediately after removal of PEEP at F_IO_2 =0.4 suggests an instability in the alveoli that have

been collapsed. It is possible that atelectasis, once formed, impedes surfactant function so that such a region is prone to collapse again after having been reopened. A VCM may promote surfactant production or release, and distribution of surfactant over the alveolar surface may cause a longer lasting protection against new collapse ***(*Magnusson *et al., 2000).***

Clinical presentation:-

Postoperative atelectasis can be asymptomatic or it may manifest as increased work of breathing and hypoxemia. The onset of hypoxemia due to postoperative atelectasis tends to occur after the patient has left the post-anesthesia care unit. It typically becomes most severe during the second postoperative night and continues through the fourth or fifth postoperative night ***(Magnusson and Spahn, 2003).***

Hypoxemia that develops earlier (ie, in the post-anesthesia care unit) should prompt the consideration of postoperative complications other than atelectasis such as hypoventilation due to residual anesthetic effects and upper airway obstruction due to airway tissue edema ***(Magnusson and Spahn, 2003).***

Diagnosis:-

Atelectasis is recognized by the finding of persistent postoperative hypoxemia in the absence of other plausible diagnoses. The patient demonstrates dyspnea or tachypnea, and physical findings can include basilar rales and