POSTOPERATIVE PULMONARY COMPLICATIONS

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SUMMERY

The forthcoming years will, as before, pose several challenges for anaesthesiologists to improve perioperative care and to take part in the multidisciplinary collaboration. Thus, the anaesthesiologist will have a major role in the improvement of postoperative outcome. Patients undergoing elective surgery first need to be screened for operative risks by reviewing factors that relate to the patients and factors that relate to the procedure they are undergoing. Identification of high-risk patients undergoing high-risk procedures may be aided by reviewing the following factors e.g. the presence of symptomatic lung disease, smoking, obesity, abnormal blood gas values, spirometry, and presence of sleep apnea. Though many of these risk factors are not modifiable, however when high-risk patients are identified, preoperative therapy aimed at reducing overall postoperative morbidity and mortality may help decrease the risk to a minimum. Reducing PPCs at the patient level will require a greater understanding of the impact of modifying risk factors through interventional trials. Reducing hospital PPC rates will require future research into the process of care associated with PPCs through controlled observational and interventional trials.

Keywords : Postoperative, Pulmonary, Complications.

Anaesthesiologist involved in the management of patient undergoing surgery needs to be aware that postoperative pulmonary complications are a major cause of morbidity, mortality, prolonged hospital stay, and increased cost of care.¹ One broad definition of postoperative pulmonary complication (PPC) includes an identifiable disease or dysfunction that is clinically relevant and adversely affects the clinical course.²,³ A basic understanding of mechanism, such as atelectasis and pneumonia, seem to be related to disruption of the normal activity of the respiratory muscles, disruption that begins with the induction of anaesthesia and that may continue into the postoperative period. Breathing is a complex behavior requiring the coordinated activity of several muscle groups, both in the upper airway and in the chest wall. Anaesthetics and many other drugs used in the perioperative period affect the central regulation of breathing, changing the neural drive to respiratory muscles such as the diaphragm. At high doses, anaesthetics attenuate the activities of all respiratory muscles. However, at moderate depths of anesthesia, anaesthetics may produce respiratory depression by altering the distribution, and timing of neural drive to the respiratory muscles, rather than by producing a global depression of activity.² This definition would include several major problems such as pneumonia, bronchitis, lobar atelectasis, respiratory failure, and prolonged mechanical ventilation.

Therefore, it is important that anaesthesiologists working in the intensive care unit/postanaesthetic care unit are knowledgeable regarding the prevention, recognition, and management of pulmonary complications following surgery.

This article reviews (i) patient-related and procedure-related factors for the development of pulmonary complications, (ii) strategies for preventing the development of PPCs through perioperative management.

Risk factors predisposing postoperative pulmonary complications

Estimation of risk factors of PPC is a necessary part of the preoperative evaluation as it contributes to morbidity, mortality, and length of hospital stay. The risk factors may be grouped into patient-related and procedure-related (table 1).

I. Patient-related factors

To prevent or reduce the incidence of PPCs, there must be an understanding of patient conditions that increase the risk of developing PPCs and of effective interventions available to reduce the impact of pre-existing patient’s conditions on the subsequent development of PPCs. Patient-related risk factors for PPCs include patient’s general health, nutritional, neurological, fluid, immune status, obesity, underlying chronic respiratory disease, history of cigarette smoking, emergency surgery, and perhaps advanced age.⁴,⁵ They are outlined in table 1.

General health and nutritional status

Risk factors that are related to general health and nutritional status include increasing age, low albumin level, dependent functional status, weight loss,⁶,⁷ and obesity. Patients greater than 60 years of age are at increased risk.
for postoperative pneumonia, and respiratory failure.\textsuperscript{6,7} Low albumin level is associated with respiratory failure,\textsuperscript{6} and higher postoperative mortality and morbidity rates.\textsuperscript{5,9} Moreover, morbidity increases exponentially as albumin level falls below 4.0 gdl\textsuperscript{-1}.\textsuperscript{9} Dependent functional status, with respect to activities of daily living, is also associated with increased risk of PPCs.\textsuperscript{6,7} Patient with greater than 10\% weight loss in the 6 months prior to surgery are at increased risk for pneumonia and respiratory failure.\textsuperscript{5,7} The risk due to age alone, once corrected, for comorbidities seems small, although data are conflicting.\textsuperscript{10,11,12}

**Neurological status**

Neurological status associated with PPCs includes impaired sensorium,\textsuperscript{6,7,13} and previous stroke. These patients are less mobile postoperatively thus, leading to higher risk of atelectasis. They are also unable to protect their airway leading to higher risks of aspiration pneumonia and respiratory failure.

**Fluid status**

Risk factors associated with fluid status include congestive heart failure,\textsuperscript{6} acute renal failure,\textsuperscript{4,14,15} and blood transfusion.\textsuperscript{6,7,16} Patients with these conditions are at increased risk for pulmonary edema and pleural effusions that may lead to atelectasis, pneumonia, and even respiratory failure. High and low blood urea nitrogen is associated with pulmonary complications, implying that careful fluid management is needed in high-risk patients.

**Immune status**

Chronic steroid use is associated with an increased risk of postoperative pneumonia. Patients with alcohol use (greater than 2 pegs per day) within 2 weeks of surgery have 20\% increased odds of pneumonia and respiratory failure. Chronic alcohol use may be associated with diminished B-cell mediated immunity leading to an increased risk of pneumonia. Patients with insulin-dependent diabetes mellitus are at slightly increased risk for respiratory failure, but not pneumonia.\textsuperscript{5}

**Respiratory status**

Symptomatic chronic lung disease is the most important factor for PPCs. It has been reported that patients with abnormal findings on lung examination, including wheezing, rales, rhonchi, prolonged expiration, and decreased breath sounds were 6 times more likely to develop a pulmonary complication.\textsuperscript{17,18} However, the risk varies according to the severity of the underlying lung disease. Therefore, care must be taken to optimize respiratory function, so that the risk for major complications may be decreased.

Current cigarette smokers have an increased risk for PPCs, even in the absence of chronic lung disease. Warner et al\textsuperscript{19} found that patient who still were smoking or had stopped for less than 2 months before the operation had a complication rate almost four times that of patients who had quit smoking more than 8 weeks preoperatively. Therefore, smokers should be recommended for cessation of smoking for 2 months before surgery. If this duration is not feasible, the largest possible period of abstinence before surgery is recommended.\textsuperscript{20}

Bacterial lower respiratory tract infections, including bronchitis and pneumonia, do increase the risk of pulmonary complications. In this situation, it is recommended to defer elective surgery and treat the underlying infection.

Patients with asthma that is well controlled and with a peak flow measurement of greater than 80\% of that predicted value can proceed to surgery with an average risk.\textsuperscript{21}

Changes related to morbid obesity can accentuate and increase the risk of PPCs due to associated physiological changes,\textsuperscript{22} such as, (a) reduced total lung capacity, functional residual capacity, and vital capacity (b) increased work of breathing secondary to elastic load, chest wall resistance, upper airway resistance, and need to eliminate more carbon-dioxide (c) hypoxemia, widened alveolar-arterial oxygen gradient, and ventilation/perfusion mismatch.

**II. Procedure-related factors**

Procedure related factors are more important than patient related factors in predicting the risk of PPCs.

**Surgery related**

The site of surgery is most important factor in predicting the overall risk of PPCs. The rate of complication is inversely related to the distance of the surgical incision from the diaphragm. Patients undergoing upper abdominal and thoracic surgery have a decreased postoperative vital capacity, which leads to V\textsubscript{A}/Q mismatch and contributes to development of hypoxemia. Thus, the rate is substantially higher for thoracic and upper abdominal surgery than for lower abdominal surgery.\textsuperscript{5,23,24} This can be explained by diaphragmatic dysfunction.\textsuperscript{25,26,27} In patients undergoing laparotomy, FRC decreases to approximately 50\% of baseline, returning towards normal over 1 to 2 weeks.\textsuperscript{28}

Furthermore, surgical trauma may also increase airway reactivity.\textsuperscript{29} Exposure to airway irritants e.g. secretion, infection in this setting of increased airway reactivity could result in bronchospasm that could lead to atelectasis or pneumonia.
The perioperative pulmonary physiology after thoracic and upper abdominal surgery can be summarized as follows:

- Reduction in vital capacity by 50% to 60% and reduction in functional residual capacity by 30%
- Diaphragmatic dysfunction secondary to reflex inhibition after surgery when viscera are handled close to the diaphragm
- Pain and splinting
- Atelectasis and pneumonia
- Impaired gas exchange and pneumonia
- Impairment of cough and mucociliary clearance
- Microaspiration

**Anaesthesia related**

Surgery in supine posture under anaesthesia causes alteration in lung volumes, impairment in the functions of respiratory muscles, alterations in lung mechanics related to gas exchange, and impairment of mucociliary clearance mechanisms. Low lung volumes may contribute to areas of microatelectasis. Duration of anaesthesia also influences the postoperative outcome. Surgical procedure lasting longer then 3-4 hours are associated with an increased risk of pulmonary complications. General anaesthesia seems to be associated with a higher risk of clinically important pulmonary complications than epidural or spinal anaesthesia. Regional nerve block is associated with reduced risk, and should be considered, if possible, for patients at high risk.

After general anaesthesia, residual effect of intravenous or inhalational anaesthetics blunt the ventilatory responses to both hypercarbia and hypoxemia. Sedatives augment depression from opioids and anaesthetics and might directly depress ventilation. Residual neuromuscular blockade following use of pancuronium might also depress cholinergic portion of hypoxic drive neural arc when compared with shorter acting neuromuscular relaxants. Specially, the long acting neuromuscular blocking agents e.g. pancuronium, pipecuronium etc., may lead to incomplete reversal of neuromuscular blockade. Residual paralysis compromises cough, airway patency, ability to overcome airway resistance, and airway protection. These may lead to airway obstruction, microaspiration, and ultimately atelectasis, pneumonia, bronchitis. Therefore, long-acting neuromuscular blockers should be avoided in high-risk patients.

**Risk factors related to postoperative care**

Risk factors for PPCs related to postoperative care include usage of nasogastric tube and parenteral narcotics for pain control. The benefit of preventing large-volume aspiration through nasogastric tube placement may outweigh the risks of ineffective coughing and oropharyngeal aspiration in high-risk patients.

Pain control is particularly important for patients with incision close to the diaphragm. Though adequate pain control improves deep breathing, resulting in decreased atelectasis and pneumonia, narcotic medications may increase aspiration risk through slowing of GI motility and also increase the risk of PPCs by reducing the ventilatory response to hypoxia and hypercapnia. Patient receiving an epidural catheter for postoperative pain control have significantly fewer pulmonary and cardiac complications than those receiving standard parenteral opioid analgesics.

**Table 1: Patient-related and procedure-related risk factors.**

<table>
<thead>
<tr>
<th>Patient-related factors</th>
<th>Procedure-related factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>General health and nutritional status -</td>
<td>Surgical site:</td>
</tr>
<tr>
<td>Age &gt; 65 years</td>
<td>Thoracic surgery &gt; upper</td>
</tr>
<tr>
<td>Low albumin</td>
<td>abdominal surgery &gt;</td>
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<tr>
<td>Functional status</td>
<td>neurosurgery &gt; peripheral</td>
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<tr>
<td>Weight loss &gt; 10%</td>
<td>surgery)</td>
</tr>
<tr>
<td>Neurological status:</td>
<td>Surgery technique</td>
</tr>
<tr>
<td>Impaired sensorium</td>
<td>Open versus laparoscopic</td>
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<tr>
<td>History of CVA</td>
<td>Other type of surgery:</td>
</tr>
<tr>
<td>Fluid status:</td>
<td>Neck surgery</td>
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<tr>
<td>CHF history</td>
<td>Peripheral vascular surgery</td>
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<tr>
<td>Renal failure</td>
<td>Neurosurgery</td>
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<tr>
<td>Blood urea nitrogen</td>
<td>General anaesthesia</td>
</tr>
<tr>
<td>Blood transfusion</td>
<td>Duration of surgery &gt; 3 hours</td>
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<tr>
<td>Immune status:</td>
<td>Emergency surgery</td>
</tr>
<tr>
<td>Chronic steroid use</td>
<td>Type of neuroaxial blockade</td>
</tr>
<tr>
<td>Alcohol use</td>
<td>(use of Pancuronium)</td>
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<tr>
<td>Diabetes</td>
<td>Not using neuroaxial blockade</td>
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<tr>
<td>Chronic lung disease:</td>
<td>Pain control with parenteral</td>
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<tr>
<td>presence of productive cough</td>
<td>narcotics vs epidural analgesia</td>
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<tr>
<td>Cigarette smoking:</td>
<td>Nasogastric tube</td>
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<tr>
<td>current or within 8 wks</td>
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<tr>
<td>ASA class &gt; 2</td>
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<tr>
<td>Obesity:</td>
<td></td>
</tr>
<tr>
<td>body mass index &gt; 27.5 kg m²</td>
<td></td>
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<tr>
<td>Abnormal chest radiograph</td>
<td></td>
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</tbody>
</table>

**Preoperative assessment of risk factors**

The prevalence of PPCs depends on a variety of risk factors. Therefore, strategies aimed at preventing postoperative complications have the potential to decrease morbidity and mortality. Pulmonary function testing, pulmonary risk indices, cardiopulmonary exercise testing, and stair case climbing all have been used to assign preoperative risk in patients undergoing elective surgery. Although one can identify patients at
increased risk for the development of PPCs, it is not possible to quantitate with more precision their risk (excluding thoracic surgery patients for whom risk predictors are better accepted).39

Necessity of pulmonary function testing and arterial blood gases

For more than a decade studies have cast doubt on necessity of pulmonary function testing, spirometry, and arterial blood gases. Reduction of forced vital capacity in 1 second (FEV1), or other spirometric indices of abnormal lung function and abnormalities in arterial blood gases such as hypoxemia or hypercapnia suggest that the patient is at increased risk for developing PPCs. But their presence does not improve the ability to stratify risk if the patient is judged to have lung disease on clinical grounds. However, the concept that clinical identification of underlying lung disease is equivalent to laboratory testing is flawed. Several studies suggest that clinical identification of pre-existing chronic lung disease is inadequate for the purposes of risk assessment.37,38 Some asthmatic patients are unaware of significant changes in their lung function and in these patients symptoms are unreliable for assessing severity and optimization of function.39 The American College of Physicians (ACP) recommends preoperative PFTs in patients undergoing lung resection, coronary bypass surgery, or upper abdominal surgery with a history of tobacco use or dyspnea, patients undergoing lower abdominal surgery if there were unexplained pulmonary disease with anticipated prolonged or extensive surgery, or patient undergoing head and neck or orthopedic surgery with unexplained pulmonary disease.39 However, the total expense of ordering routine PFTs can be wasteful. One economic analysis estimates that roughly 40% of PFTs ordered do not meet ACP guidelines.40

Spirometry continues to have a role in preoperative risk assessment process. Spirometry is useful when there is uncertainty about the presence of lung impairment. An ambiguous clinical picture regarding the severity of bronchospasm, presence of COPD, response to bronchodilators, or unexplained shortness of breath can be clarified by spirometry. It should not be used indiscriminately nor should it be avoided; rather it should be used selectively when the information it provides will change management or improve risk stratification. A critical review concludes that preoperative spirometry is not useful in predicting pulmonary complications after abdominal surgery.

Baseline arterial blood gases (ABGs) do not improve risk assessment nor add to risk stratification. Since the need for postoperative supplemental oxygen is determined by level of oxygenation and hemoglobin after surgery, and because supplemental oxygen is titrated to effect, baseline ABGs are not necessary in most circumstances.41

Cigarette smoking

Cigarette smoking is a significant preoperative risk factor.42,43 This effect is primarily related to the resulting chronic lung disease. Cessation of cigarette smoking for 48hrs before surgery include an expected reduction in cough, reduction in lower airway pathogens, decreases carboxyhemoglobin levels to normal, abolishes the stimulant effect of nicotine on cardiovascular system, and improves respiratory ciliary beating. However, 1 to 2 weeks are required to decrease sputum volume and 4 to 6 weeks are required to improve symptoms and lung function.44 Patient who stop smoking more than 8 weeks before surgery have a reduced rate of PPCs compared with those who continue to smoke.45

The paradoxical increase in PPCs observed with short-term abstinence or reduced smoking may be caused by ineffective sputum removal. Reduced smoking may decrease bronchial irritation and the stimulus for coughing; at the same time, bronchial hyper secretion of mucus is still present or even transiently increased. This cascade may result in increased sputum retention.43

Obesity

In 1987, “Strandberg and colleagues46 found a weak correlation between obesity [calculated by Broca’s index: weight (kg)/(height in cm-100)] and the area of lung densities seen directly after induction of anaesthesia”. Desaturation and respiratory complications in the postoperative period are connected with postoperative pulmonary atelectasis in morbidly obese patients. Atelectasis persists for at least 24 hrs in morbidly obese patients whereas it disappeared in non-obese.45 Functional residual capacity is lower in morbidly obese patients, the alveolar arterial oxygenation gradient (A-aDo2) is increased and intra abdominal pressure is higher. When PEEP is applied, respiratory function improved in morbidly obese patients. Avoiding atelectasis formation may be particularly difficult in these patients but at the same time particularly important.

Chronic obstructive pulmonary disease

Patients with underlying chronic lung disease are at increased risk of development of PPCs. They may have chronically fatigued respiratory muscles. Impaired nutrition, electrolyte, and endocrine disorders contribute to respiratory muscle weakness and should be corrected before surgery.
Patients with COPD develop only a small shunt and almost no atelectasis during anaesthesia. However, they develop a more severe V/Q mismatch.\textsuperscript{48} Determination of exercise capacity may be a benefit in identifying patients at risk of PPCs.\textsuperscript{49}

Symptom-limited stair climbing
This test has the advantage of being simple and not requiring costly, sophisticated equipment. Pollock et al\textsuperscript{50} used in patient with varying degree of airflow obstruction a Douglas bag for sampling of expired gases, and confirmed a linear relationship between maximal oxygen consumption and flight of stairs climbed.

Subsequently, Olsen et al\textsuperscript{35} noted that patients unable to climb three flights had a high number of PPCs and longer hospital stay compared with patients able to climb three flights.

Maximal oxygen uptake
It is an indicator of cardiopulmonary reserve and the patient’s ability to tolerate cardiopulmonary stress. As the maximal oxygen uptake decreases to less than 15 mlkg\textsuperscript{-1}min\textsuperscript{-1}, the incidence of postoperative complications increases. Thus, the assessment of maximal oxygen consumption is an assessment of aerobic capacity and of the reserve a patient may have when dealing with the multiple physiologic abnormalities that accompany surgery.

Asthma
Risk factors for the development of PPCs in asthmatics include recent asthma symptoms, recent use of anti-asthma drugs or therapy in medical facility for asthma symptoms and history of tracheal intubation for asthma.\textsuperscript{21}

Obstructive sleep apnea
Obstructive sleep apnea (OSA) is a breathing disorder characterized by repeated collapse of the upper airway during sleep with cessation of breathing.\textsuperscript{51} The loss of upper airway muscle tone, particularly during rapid eye movement sleep, results in a narrow floppy airway becoming narrower. Airway obstruction results in arousal, interruption in sleep, restoration in muscle tone, and the airway becomes patent again. Almost all patients with OSA have a history of snoring. OSA is more common in men, obese individuals and elderly with hypertension, arrhythmias, congestive heart failure, coronary artery disease, and stroke. A preoperative snoring is a risk factor for postoperative apnea and lower postoperative mean oxygen saturation.\textsuperscript{52}

General anaesthesia alone results in transient and minimal alterations in sleep architecture although anaesthetic agents reduce upper airway muscle tone to a greater extent than diaphragmatic strength thus increasing the propensity for obstruction.

OSA patients may need expensive treatments like continuous positive airway pressure (CPAP) and bilevel positive airway pressure (BiPAP), may need special monitoring. Non-steroidal analgesics may reduce the need for narcotic based analgesia.\textsuperscript{41}

Preoperative and postoperative management of complications

Patient with severe COPD
Some patients with COPD have bronchospasm in addition to their fixed airway disease. Inhaled beta-2 adrenergic agonists, anticholinergic agents or a course of steroid may be useful. Patient with chronic hypoxemia benefit from short term oxygen administration, which usually results in lessening of pulmonary hypertension, reduction in signs and symptoms of heart failure, and improvement of mentation. A preoperative finding of hypoxemia should prompt further investigation. Even if hypoxemia is chronic, but the patient is not receiving oxygen at home, continuous oxygen administration should be started and elective surgery deferred to allow improvement in pulmonary hypertension and heart function.

Respiratory muscle training in patients of COPD has potential values. Pulmonary rehabilitation is helpful in reducing PPCs in high-risk patients.\textsuperscript{49}

Patient with asthma
The type of anaesthesia has not been demonstrated to be a risk factor for PPCs in asthmatics. In the study of Warner’s\textsuperscript{21} over 1500 patients with asthma, the complication rates for general and regional anaesthesia were similar, refuting the notion that regional anaesthesia was safer for patients with asthma. The risk of bronchospasm in perioperative period is low in stable asthmatic patients and when it occurs is usually not associated with serious morbidity.

Corticosteroids are effective in attenuating bronchospasm in the perioperative period starting 24 to 48 hrs before surgery in advance either orally (40 to 60 mg in adults), or intravenously (hydrocortisone 8 hrly) in those unable to take medication by mouth. Steroids can be discontinued after surgery without tapering doses in the absence of bronchospasm. Use of short course of systemic steroids in the perioperative period is not associated with increased wound infection or poor wound healing.\textsuperscript{53}
Patients who are wheezing before surgery should be free of wheezes and have a peak flow of at least 80% of the predicted value, or of personal best with inhaled beta-2 adrenergic agonists and corticosteroids. Elective surgery should be deferred if the patient does not improve. Airway hyperactivity persists for several weeks after an episode of asthma. Improvement of asthma symptoms does not preclude the development of bronchospasm in response to various stimuli. Volatile anaesthetic agents are bronchodilators and the differences between them with respect to their efficacy in treating bronchospasm are probably clinically significant. Propofol is useful in patients with bronchospasm and it is associated with reduction in wheezing during induction.

Regional anaesthesia may be used where appropriate; to avoid instrumentation of airway and it also does not lead to unopposed parasympathetic effects, or enhanced bronchoconstriction.

Laryngeal mask airway is associated with less airway reaction than endotracheal tubes suggesting they may be useful in patients with reactive airway disease.

Postoperative atelectasis

Atelectasis are found in 90% of all patients who are anaesthetised and occur in the basal part of lung. Development of basal atelectasis is neither dependent upon the technique of anaesthesia nor on the type of anaesthetic agents and whether patient is breathing spontaneously or is paralysed and ventilated mechanically. Most atelectasis occurs near diaphragm in supine posture and less towards the apex. After cardiac surgery with cardiopulmonary bypass atelectasis is more prominent than after other forms of surgery even thoracotomies.

Pulmonary atelectasis may be caused by a variety of factors, which have been classified into following mechanisms.

(A) Compression atelectasis occur when the transmural pressure distending the alveolus is reduced.

(B) Absorption atelectasis occur when less gas enters the alveolus than is removed by uptake by the blood.

(C) Loss-of-surfactant atelectasis occur when the surface tension of an alveolus increases because of reduced surfactant action.

Supplementation of high oxygen concentration has been often associated with atelectasis formation. Increasing FiO₂ at the end of surgery to 1.0 before extubation will favour atelectasis formation, persisting in postoperative period. Using 80% oxygen compared with 30% reduces the incidence of postoperative nausea and vomiting from 30% to 17%. More importantly use of 80% oxygen during colorectal resection halved the incidence of surgical wound infection compared with FiO₂ of 0.3. Such studies suggest that a FiO₂ of 0.8 may offer advantages during general anesthesia despite potential effect on atelectasis formation.

Atelectasis is not seen on conventional chest radiograph unless it becomes massive. This was first shown using computed tomography by Hedenstierna et al and provided a convincing explanation for the abnormality in gas exchange which occurred during anaesthesia in particular the extent of atelectasis.

Most of the atelectasis appearing during general anaesthesia resolves within 24hrs after surgery. But, some pulmonary complications occur during or immediately after anaesthesia, mainly hypoxemia and some will occur later, mainly pneumonia. A Vital Capacity Maneuver (VCM) can completely abolish atelectasis that develops after induction of general anaesthesia. Lung inflation to an airway pressure of 20 cm H₂O did not affect atelectasis; an airway pressure of 30 cm H₂O reduces atelectasis, only with a pressure of 40 cm H₂O maintained for 15 secs atelectatic lung tissue fully expand. This pressure is equivalent to inflation to vital capacity, and thus this maneuver has been called the VCM. It has been shown that this maneuver needs to be maintained for only 7 to 8 secs in order to re-expand all previously collapsed lung tissue.

Tusman and colleagues studied an alternative maneuver. They increased both PEEP to 15 cm H₂O and tidal volume to either 18 ml kg⁻¹ or pressure of 40 cm H₂O and maintained this for 10 breaths. PEEP was then decreased stepwise to 5 cm H₂O and tidal volume reduced to 9 ml kg⁻¹. The application of PEEP of 10cm of H₂O has been tested in several studies and will consistently reopened collapsed lung tissue, but PEEP may not be ideal.

Use of bilevel positive airway pressure (BiPAP) system with inspiratory and expiratory positive airway pressure set at 12 and 4 cm H₂O respectively to treat obese patients for the first 24 hrs after upper abdominal surgery significantly reduced pulmonary dysfunction.

Aspiration Pneumonitis

Regurgitation and aspiration of gastric contents during induction of anaesthesia leads to aspiration pneumonitis. Studies have found that 40 to 60% of ambulatory patients would be defined as "high risk" for aspiration pneumonitis by traditional criteria (gastric volume more than 25 ml with pH less than 2.5) despite an overnight fast. Although ambulatory patients were initially reported to have...
significantly higher residual gastric volumes than inpatients, this has not been confirmed in more recent studies. The H₂-receptor antagonists are effective in decreasing the number of patients at risk for pulmonary aspiration. Metoclopramide, a prokinetic agent, has been shown to reduce gastric volume without altering pH, hence, use of metoclopramide in combination with a H₂-antagonist has been advocated to decrease postoperative emesis and further reduction in the risk of aspiration pneumonitis.

0.3M sodium citrate, a non-particulate oral antacid, is less effective in raising pH than the H₂-antagonist and can increase gastric volume. However, sodium citrate should be used to increase the pH of gastric content in place of H₂-receptor antagonists when adequate time is not available before induction of anaesthesia.

Treatment should include intubation and ventilation if respiratory failure is severe. Bronchoscopy should be used at the earliest opportunity to facilitate suctioning and to remove particulate matter from bronchial tree. Broad-spectrum coverage is prudent if the initial sputum gram stain is positive for inflammatory cells and bacteria. Specific coverage with antibiotics is dictated by culture results and should be 7 to 10 days in duration if positive cultures are obtained.

Ventilation associated pneumonia

Pneumonia is associated with the placement of an endotracheal tube and mechanically assisted ventilation. The incidence of respiratory tract infection in patients requiring an endotracheal tube can be as high as 17 to 20 %. Approximately 60% of nosocomial infections are caused by aerobic gram-negative bacteria and 20% are caused by viral infection and may be an important source of morbidity and mortality in immunocompromised patients.

Ventilator alone is not considered an important source of bacterial spread, breathing circuits can become heavily contaminated with microorganisms from the patient’s oropharynx and trachea. Therefore, disposable circuits or disinfection of components used in the breathing system is equally effective in preventing cross infection between patients.

Pneumonia must be diagnosed and treated aggressively. Pulmonary toilet and culture-specific antibiotic strategies remain the basic treatment plan. Initially, coverage with broad-spectrum antibiotics may be needed until microbiology results are known, but then a tailored antibiotic regimen is strongly recommended to minimize the potential for the development of resistant organisms.

Deep vein thrombosis and pulmonary embolism

Deep Vein Thrombosis (DVT), with the associated risk of Pulmonary Embolism (PE), occurs in 45 to 75 % of patients who undergo orthopaedic procedures on lower extremities. Dyspnoea is the most important symptom of PE, and tachypnea is its most important sign. Other symptoms and signs like syncope, hypotension, cyanosis, pleuritic pain, cough or hemoptysis suggest PE in high-risk patients.

DVT is confirmed usually by ultrasonography of deep vein system, relies upon loss of vein compressibility as the primary criterion. In patients with PE chest roentgenography shows focal oligemia and peripheral wedge shaped density above the diaphragm. Selective pulmonary angiography is most specific, available for definite diagnosis.

Many measures have been employed to prevent this complication; none is fully effective. Early mobilisation, elevation of foot end of the bed, and pneumatic compression stockings seems safer. Many anticoagulant regimens employing varying doses of coumarin, heparin, low-molecular weight heparin, aspirin, or dextran have been investigated. These therapies typically reduces rate of DVT by 50 to 70 % compared with placebo, however, hematomas are more frequent in those, who had received anticoagulant regimens and the induced coagulopathy may discourage the use of spinal and epidural anaesthesia, for fear of producing an epidural haematoma.

Primary therapy of PE consists of clot dissolution with thrombolysis or removal of pulmonary thrombus by embolectomy. Anticoagulation with heparin and warfarin for 3 to 6 months to facilitate clot resorption and vascular recanalisation. Patients who have complications related to the anticoagulation, have recurrent PE on anticoagulation, or have contraindications to anticoagulation should receive an inferior vena cava filter. Fat embolism would often evolve into ARDS, whereas air embolism and amniotic fluid embolisms are usually lethal.

General postoperative consideration to prevent PPCs

Ventilation

Ventilatory support after recovery from anaesthesia may be brief or prolonged. Continuous Positive Airway Pressure (CPAP) or PEEP is usually helpful in improving oxygenation and CO₂ removal by decreasing atelectasis, improving Vₐ/Q, and reducing barotrauma to the lung.
Posture

Hypoxemia can be reduced and the ventilation of the lung bases improve by keeping the patient, especially if obese, in a sitting or semi recumbant position rather than in a supine position.

Pain control

Adequate pain control would decrease splinting and improve the ability to take deep breaths; they reduce pulmonary complication by these mechanisms. Hence, epidural analgesia with local anaesthetics and opioids would be ideal one to reduce PPC in patients undergoing abdominal and thoracic surgeries. Studies that show a reduction of PPCs with the use of regional anaesthesia after high risk patients (upper abdominal surgery, COPD) and use of epidural local anaesthetics. A recent meta-analysis suggests that postoperative epidural analgesia may reduce clinical PPCs.85

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