Preoperative assessment: pulmonary

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Postoperative pulmonary complications (PPCs) include atelectasis, pneumonia, bronchitis, bronchospasm, hypoxemia, respiratory failure, and prolonged mechanical ventilation. These conditions are a significant source of morbidity and mortality [1,2]. Postoperative changes include diaphragmatic dysfunction, V/Q mismatch, and reductions in functional residual capacity (FRC), which, while measurable, have an unclear relation to morbidity and mortality. The etiology of PPCs is complex and poorly understood. Because it is not practically or ethically possible to study the isolated effects of general anesthesia (GA) on the development of pulmonary complications after surgery, it is difficult to determine the dominant contribution (from anesthesia or from surgery). GA and surgery produce significant changes in the respiratory system, and the interaction of these changes is responsible, along with underlying respiratory conditions, for PPCs.

GA decreases the number and activity of alveolar macrophages, inhibits mucociliary clearance, increases alveolar-capillary permeability, inhibits surfactant release, increases the activity of pulmonary nitric oxide synthetase, and enhances the sensitivity of the pulmonary vasculature to neurohumoral mediators [3–22]. GA also results in mechanical and functional changes to the respiratory system that may contribute to the development of PPCs. After induction of GA, FRC decreases and atelectatic plaques form in dependent portions of the lungs [23]. GA also alters diaphragmatic movement with near uniform motion of the diaphragm along the ventral-dorsal axis [24]. This may result in more ventilation of the superior (poorly perfused) portion of the lung and less ventilation of the lung in the dependent (better perfused) portion. The resulting ventilation-perfusion (V/Q) inequality leads to shunt and dead-space ventilation. Regional diaphragmatic anatomic and neural differences result in abnormalities in diaphragmatic position and movement during GA [24]. These changes result in an

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increase in the alveolar-arterial oxygen gradient and explain why supplemental oxygen is necessary during surgery and GA.

Even small concentrations of intravenous or volatile anesthetics (0.1 MAC) blunt the ventilatory response to both hypoxemia and hypercarbia by depression of the peripheral chemoreceptors (which play an important role in the response to hypoxemia) and the central nervous system (CNS) regions that regulate $PaCO_2$ [25–30]. Respiratory drive is determined by the state of arousal, metabolic factors, including pHa, $PaCO_2$, and PaO_2, and the interaction between the response to hypoxia and hypercarbia [31]. Depending on the state of arousal and anesthetic agent, sedative concentrations of volatile anesthetics may result in either no change or a depressed response to hypoxemia and hypercarbia. Depression of the ventilatory response to hypoxemia or hypercarbia by subanesthetic concentrations of volatile anesthetics may be important in the pathogenesis and maintenance of postoperative hypoxia. In addition, neuromuscular blockers may reduce the hypoxic drive to ventilate [32]. Because a low level of volatile anesthetic persists for hours, patients are at risk for anesthetic-induced depression of the response to hypoxemia or hypercarbia after they leave the highly monitored operating room and postanesthesia care unit (PACU) environment.

Effects of surgery: postoperative changes

Patients who undergo abdominal (upper > lower) and thoracic surgery have a decreased postoperative vital capacity and functional residual capacity (FRC). This decrease in FRC results in V/Q mismatch and contributes to the development of hypoxemia. In patients undergoing laparotomy, FRC decreases to approximately 50% of baseline, returning toward normal over 1 to 2 weeks [33]. Diaphragmatic dysfunction occurs after upper abdominal or thoracic surgery and may help explain PPCs. The diaphragmatic "weakness" is not caused by residual neuromuscular blockade or inadequate pain relief. Phrenic nerve pacing restores diaphragm function suggesting that reflex inhibition of phrenic nerve output results in diaphragmatic dysfunction [34,35]. The hypothesis that phrenic nerve-induced diaphragmatic dysfunction may contribute to atelectasis has been tested and phrenic nerve stimulation during GA reduced atelectasis in human volunteers [36]. Surgical trauma may also increase airway tone and reactivity [37]. Exposure to airway irritants (secretions, infection) in this setting of increased airway reactivity could result in bronchospasm that could lead to atelectasis or pneumonia. Based on this evidence, bronchodilators have a potential role in the postoperative respiratory care of even those patients without asthma or chronic obstructive lung disease (COPD).

Because it is "minimally invasive," laparoscopic surgery might have advantages in patients with underlying lung disease. There is improved FEV_1 and FVC, better arterial oxygenation, and improved ventilation after laparoscopic, as compared with open, procedures [38]. But, upper abdominal laparoscopic surgery is associated with dysfunction of the diaphragm [39]. The site of surgery rather than

the surgical technique is critical in determining whether there will be diaphragmatic dysfunction. Importantly, no studies have demonstrated a reduction in clinical respiratory complications such as pneumonia, bronchitis, or respiratory failure after laparoscopic surgery.

Preoperative assessment

To prevent or reduce the incidence of PPC, 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 conditions on the subsequent development of PPCs. PPCs occur commonly in patients who undergo anesthesia and surgery, especially after upper abdominal or thoracic surgery. Risk factors for PPCs also include cigarette smoking, underlying chronic respiratory disease, emergency surgery, anesthetic time of 180 minutes or more, and perhaps advanced age [40]. Depending on the series, definition of pulmonary complications, and methods used to detect such complications, PPCs occur in approximately 10% to 30% (or even higher in some series) of patients undergoing major, nonthoracic surgery [1,2]. While perioperative cardiac complications are easy to define, a standard definition of exactly what constitutes a PPC does not exist. The lack of a standard definition for PPCs hinders comparisons between series.

Numerous studies have confirmed that the presence of underlying lung disease, specifically COPD, places the patient at risk for the development of PPCs. What is it about COPD that increases the risk of PPCs? In smokers undergoing abdominal surgery the presence of obstructive lung disease predicts bronchospasm but not prolonged endotracheal intubation or ICU admission [41]. Thus, the increased frequency of PPCs in patients with COPD may be caused by the combination of pre-existing pulmonary disease in addition to other co-morbidities (eg, cardiovascular disease) rather than by the isolated presence of airway obstruction. This finding suggests there are additional, currently unidentified risk factors that contribute to the development of PPCs. It also suggests that in the future one may be able to better predict which patients will develop PPCs, and the frequency with which an individual patient will develop PPC. Similarly, the care of patients undergoing surgery, and resource allocation decisions regarding the necessity for postoperative ICU care would be improved if there were an effective way to triage to the intensive care unit patients that are at high risk of developing PPCs. Patients with a longer history of smoking, lower preoperative arterial PaO_2, and greater intraoperative blood loss have been found to require mechanical ventilation for more than 24 hours after abdominal vascular surgery [42].

Perhaps surprisingly, risk factors for the development of PPCs are primarily clinical in nature. Although laboratory testing can predict patients at risk for PPCs, these tests have not been shown to have superior sensitivity or specificity compared with clinical observations. No studies have suggested a pulmonary function test (PFT) value that would contraindicate surgery. In fact, empiric ob-

servations suggest that even patients with significant obstructive lung disease can successfully undergo anesthesia and major surgery [43]. Lung volume reduction surgery (LVRS) is successfully performed on patients with severe lung impairment [44]. Consideration of the foregoing risk factors for the development of PPCs suggests patients undergoing LVRS are at high risk: they are older, have significant underlying lung disease, and are undergoing surgery associated with a significant risk for PPCs. The results of such surgery emphasize that assessment of patients with lung disease must take into account not only the risks of the proposed surgery but also the potential benefits.

Necessity for pulmonary function testing and arterial blood gases

Is preoperative laboratory evaluation of pulmonary function (PFTs, spirometry) required for proper assessment of patients with lung disease? For more than a decade studies have cast doubt on the necessity for such testing [45]. Clinical identification of lung disease is thought to be comparable to spirometry in terms of assessing a patient's risk for developing PPCs. Reduction in the forced vital capacity in 1 second (FEV_1), or other spirometric indices of abnormal lung function and abnormalities in arterial blood gases such as hypercapnia or hypoxemia 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 [46,47]. 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 [48]. PFTs (FEV_1, peak flow) can determine a change from baseline or response to therapy.

Thus, spirometry continues to have a role in the preoperative risk assessment process. Spirometry is useful when there is uncertainty about the presence of lung impairment. A proper determination of whether or not the patient has underlying lung disease can have a profound effect on subsequent management decisions. If a patient is inaccurately assessed as having COPD, their risk will be judged to be higher and the patient may be denied surgery or exposed to unnecessary treatment. Failure to diagnose underlying lung disease when it is present may suggest the patient is at lower risk and result in forgoing treatment that might prevent PPCs. 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. Spirometry can complement a clinician's ability to detect changes in asthmatic status or resolution of a COPD flare. Spirometry is non-invasive, easily performed and interpreted, inexpensive, and provides important information about lung disease. PFTs and exercise testing provide information that is useful in determining whether a patient is a candidate

for lung resection. Spirometry does not allow calibration of a patient's risk, but may enhance diagnosis of lung disease. 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.

Obtaining routine preoperative baseline arterial blood gases (ABGs) is usually not helpful as postoperative patients frequently have abnormalities in oxygenation and ventilation. Baseline ABGs do not improve risk assessment nor add to risk stratification. Since the need for postoperative supplemental oxygen is determined by the level of oxygenation and hemoglobin after surgery, and because supplemental oxygen is titrated to effect, baseline ABGs are not necessary in most circumstances. Pulse oximetry is non-invasive, cost-effective, and supplements the history and physical examination. Patients with hypoxemia will be detected by an abnormal arterial oxygenation saturation.

Cigarette smoking

Cigarette smoking is a significant preoperative risk factor [42,49]. This effect is primarily related to the resulting chronic lung disease although smoking has other effects on lung function. Cessation of cigarette smoking for 48 hours before surgery decreases carboxyhemoglobin levels to normal, abolishes the stimulant effect of nicotine on the 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 [50]. More than 20 pack-years of cigarette smoking is associated with an increased incidence of PPCs. Patients who stop smoking more than 8 weeks before surgery have a reduced rate of PPCs compared with those who continue to smoke [51,52].

A pulmonary risk index

Perioperative management of patients would be improved if one could predict with more assurance, not only which patients will develop PPCs but the magnitude of the risk faced by an individual patient (ie, perform risk stratification). Most of the data in the literature have suggested that 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 more well accepted) [45]. Efforts to use laboratory testing to aid in risk stratification have not been helpful but have been, as discussed, useful in identifying underlying lung disease when the clinical diagnosis of lung disease is not clear. Recently, there have been renewed efforts to develop a pulmonary risk index predictive of the development of postoperative respiratory failure or pneumonia [53,54]. In contrast to previous studies where attempts to develop a pulmonary risk index were limited to analysis of small numbers of patients, the risk factors in these studies were based on the analysis of thousands

of patients in the veteran affairs patient database. Points were assigned to clinical and other factors based on the significance of these factors as assessed by logistical regression. The major contributors to patients' risk remain clinical, and PFT does not seem to contribute to assessment of a patient's risk. The major factors that increase a patients risk of postoperative pneumonia or respiratory failure are: age (>70), type of surgery (vascular and thoracic), presence of underlying lung disease, renal failure, poor nutritional status, and the amount of blood loss during surgery (>4 units packed blood cells).

Decreasing postoperative complications

A variety of techniques have been reported to decrease PPCs, including incentive spirometry, pain relief, and agents such as theophylline, which in addition to its bronchodilating properties, has a stimulant effect on the respiratory system and is an inotrope to the diaphragm. None of these maneuvers is entirely effective in preventing PPCs. Incentive spirometry can decrease rates of PPCs and hospital length of stay [55]. There is conflicting evidence regarding the usefulness of regional analgesic techniques in preventing PPCs [56,57]. Studies that show a reduction of PPCs with the use of regional anesthesia after surgery examine high-risk patients (upper abdominal surgery, COPD) and use epidural local anesthetics. A recent meta-analysis suggests that postoperative epidural analgesia may reduce clinical PPCs [58]. However, the lack of a standard definition for PPCs and the age of the studies included in the meta analysis limit the applicability of the results of that study. A more recent double-masked randomized trial failed to show a benefit of regional anesthesia or analgesia with respect to reintubation, prolonged intubation, or pneumonia [59]. Other studies have also yielded contradictory results regarding the value of epidural analgesia in reducing the incidence of PPCs [60,61]. The conflicting data suggest that mechanisms underlying PPCs involve more than pain and effects of the stress response on the respiratory system. Additional well-performed randomized clinical trials are necessary to establish the role of epidural analgesia in the treatment or prevention of PPCs.

Episodic or sustained arterial desaturation occurring in the days after surgery may play a role in the development of other complications. If postoperative hypoxemia develops, tachycardia and hypertension will ensue at a time when postoperative anemia decreases arterial oxygen content. This may induce myocardial ischemia in patients with coronary artery stenoses. A recent study showed postoperative oxygen therapy decreased heart rate and increased arterial oxygen saturation several days after abdominal surgery [62]. Perioperative supplemental oxygen also has been shown to decrease both postoperative nausea and vomiting and surgical wound infections by approximately 50% [63,64]. Oxygen therapy may be beneficial even if administered for only several hours after surgery, rather than administered only in the PACU, especially in patients who have undergone major abdominal surgery.

Types of anesthesia

The results of many studies do not demonstrate a clear advantage of one anesthetic technique over another in lessening PPCs [56]. Regional anesthesia has the advantage of not requiring airway manipulation or affecting ventilatory control. Neuromuscular block is not necessary and there may be less impact on ventilatory control than with a GA. Neuraxial block with local anesthetics may result in respiratory muscle weakness and impaired cough. GA has the advantage of ensuring patient cooperation, control of the airway and the ability to suction secretions through an endotracheal tube. Disadvantages of GA include the common adjunct use of neuromuscular block, alteration of ventilatory control and airway manipulation, with its potential for secretions and bronchospasm.

Residual neuromuscular blockade occurs more commonly after pancuronium than after vecuronium or atracurium, and residual neuromuscular blockade from pancuronium is a risk factor for development of PPCs [65]. Use of shorter-acting neuromuscular blocking agents should be considered in patients at risk for PPCs. Further studies are required to define the role of residual neuromuscular blockade in the pathogenesis of PPCs and to determine the role of regional anesthesia in the patient with lung disease.

Patients with asthma

Bronchospasm is one of the most significant respiratory events that can occur during anesthesia [66]. Complication rates in asthmatic patients are lower than rates reported 30 years ago, suggesting that current management has reduced the risk of perioperative bronchospasm, pneumonia, respiratory failure, and death. Risk factors for the development of PPCs in asthmatics include recent asthma symptoms, recent use of anti-asthma drugs or therapy in a medical facility for asthma symptoms, and history of tracheal intubation for asthma [67]. The type of anesthesia has not been demonstrated to be a risk factor for PPCs in asthmatics. In Warner's study [67] of over 1500 patients with asthma, the complication rates for general and regional anesthesia were similar, refuting the notion that regional anesthesia was safer for patients with asthma. The risk for bronchospasm in the 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 [68–71]. Asthmatic patients at risk for PPCs may be treated with steroids in the perioperative period starting 24 to 48 hours before surgery, because the beneficial effect of steroids on airway reactivity occurs over a period of hours. An appropriate daily dose of prednisone in an adult would be 40 to 60 mg. Although there is limited evidence regarding the appropriate treatment regiment, if corticosteroids are administered preoperatively in asthmatic individuals, they may be given 1 to 2 days in advance of planned surgery either orally in the does suggested earlier or intravenously (hydrocortisone in a dose of 100 mg every 8 hours) in those unable to take

medications by mouth. Steroids can be discontinued after surgery without tapering doses in the absence of bronchospasm. Use of short courses of systemic steroids in the perioperative period is not associated with increased wound infections or poor wound healing [72]. Inhaled steroids might be a reasonable alternative to parenteral steroids for the purpose of reducing perioperative bronchospasm in patients with asthma.

Patients who are wheezing before surgery should receive treatment with inhaled beta-2 adrenergic agents and corticosteroids. Theophylline is no longer a front-line drug in the management of asthma and it has the potential for significant toxicity [73]. Those who improve with treatment may be allowed to proceed with surgery. Elective surgery should be deferred if the patient does not improve. Airway hyperreactivity persists for several weeks after an episode of asthma [74]. Improvement in asthma symptoms does not preclude the development of bronchospasm in response to various stimuli. Volatile anesthetic agents are bronchodilators and the differences between them with respect to their efficacy in treating bronchospasm are probably clinically insignificant [75]. Propofol is useful in patients with bronchospasm and it is associated with reductions in wheezing during induction [76,77]. Common sense suggests that regional anesthesia should be used, where appropriate, to avoid instrumentation of the airway. Regional anesthesia does not lead to unopposed parasympathetic effects or enhanced bronchoconstriction [67]. The combination of lidocaine and a beta-2 aerosol has a synergistic effect in attenuating the airway response to bronchoconstrictors. [78]

Laryngeal mask airways are associated with less airway reaction than endotracheal tubes suggesting they may be useful in patients with reactive airways disease [79,80]. Not all wheezes require treatment and not all wheezing is related to pre-existing reactive airways disease. Other causes of wheezing include pulmonary edema, pneumothorax, drug reactions, aspiration, carinal irritation, and endobronchial intubation. Assessment of the significance of wheezing requires measurement of tidal volume and associated airway pressures, arterial oxygenation, and vital signs. Oxygenation is more important than elimination of CO_2 in the patient who develops severe bronchospasm with increased airway pressures. In this situation permissive hypercapnia may be useful allowing $PaCO_2$ to rise above normal levels, using supplemental oxygen to insure adequate oxygenation and avoiding high airway pressures that can cause barotrauma [81–83]. "ICU-type" ventilators can also help improve gas exchange in patients with high airway pressures.

Patients with chronic obstructive pulmonary disease

Patients with underlying chronic lung disease are at risk for the development of PPCs. Respiratory infections should be treated with antibiotics. Some patients with COPD have bronchospasm, in addition to their fixed disease. Inhaled beta-2 adrenergic agonists, anticholinergic agents, or a course of steroids may be useful.

Patients with COPD 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 should be examined for unrecognized cor pulmonale; if present, it should be treated before surgery. One modality of potential value in patients with COPD is respiratory muscle training. Results of LVRS are consistent with the hypothesis that pulmonary rehabilitation is helpful in reducing PPCs in high-risk patients. Although this has not been tested with a randomized clinical trial, measurement of respiratory muscle strength may identify patients at risk for development of PPCs. Patients that demonstrate increased respiratory muscle strength after respiratory muscle training have fewer PPCs than those who do not increase their respiratory muscle strength [84]. Determination of exercise capacity may also be of benefit in identifying patients at risk for PPCs [85].

There is little published information to guide management of patients requiring chronic oxygen administration. Patients 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.

Patients with 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 [86]. The loss of upper airway muscle tone, particularly during rapid eye movement (REM) sleep, results in a narrow floppy airway becoming narrower. Airway obstruction results in arousal, sleep is interrupted, muscle tone is restored, 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 the elderly. OSA is associated with hypertension, arrhythmias, congestive heart failure, coronary artery disease, and stroke. A preoperative history of snoring or apneas during sleep strongly suggest OSA [87]. Preoperative snoring is a risk factor for postoperative apnea and lower postoperative mean oxygen saturation [88]. GA alone results in transient and minimal alterations in sleep architecture although anesthetic agents reduce upper airway muscle tone (which acts to oppose airway collapse) to a greater extent than diaphragmatic strength thus increasing the propensity for obstruction. In the initial nights after major surgery, sleep is fragmented with decreased REM sleep. In succeeding nights REM sleep is increased (REM rebound). Sleep disturbances are less after laparoscopic surgery suggesting that the magnitude of surgery or use of opioid analgesics may be important factors in the development of postoperative sleep disturbances [89–92].

Postoperative hypoxemia develops quickly after emergence from GA [93]. Diffusion hypoxemia may contribute to early postoperative hypoxemia. However, postoperative hypoxemia is usually related to V/Q inequality or hypoventilation. The elderly are prone to more severe hypoxemia. Multiple episodes of more acute and severe hypoxemia associated with airway obstruction have been observed days after surgery. There are interactions between natural "sleep," age, the postoperative period, and narcotic analgesia that produce ventilatory disturbances [94]. Preoperative sleep-related oxygenation disturbances need not be present for postoperative hypoxemia to occur [95].

Sleep disordered breathing may occur after major surgery even in patients that do not have OSA. Patients with sleep apnea are likely to have worsening of their disease process after anesthesia and surgery and may be at risk for development of more apneas and more severe episodes of hypoxemia postoperatively. Sedatives, anesthetic, and analgesic agents may worsen OSA by decreasing pharyngeal tone and attenuating ventilatory and arousal responses to hypoxia, hypercarbia, and obstruction. Postoperatively patients are often supine, which can aggravate OSA. OSA patients frequently have redundant pharyngeal tissue, which makes airway management difficult. OSA patients may need expensive treatments (eg, continuous positive airway pressure [CPAP] and bilevel positive airway pressure [BiPAP]), may need special monitoring, and may have cardiovascular disease. A recent retrospective study demonstrated a higher rate of adverse postoperative complications in patients with OSA undergoing hip and knee replacements compared with case-matched controls [96]. It seems prudent and cost-effective to diagnose OSA preoperatively so these measures are applied appropriately and other risk-reduction measures can be taken. Preoperative treatment of OSA can improve heart function, reduce pulmonary artery pressures, and allow blood pressure to normalize, all of which may reduce complications.

The limited evidence available regarding postoperative risks in patients with OSA suggests the type of surgery is more important than the anesthetic. Regional anesthesia may reduce or eliminate problems with airway maintenance, depression of ventilation and arousal responses but may not affect REM rebound. Outpatient surgery may be appropriate for extremity or "non-invasive" procedures, when rapid and complete restoration of postoperative consciousness is possible, if there is a limited need for narcotic analgesics, or if the patient's OSA does not require CPAP. A postoperative hospital stay is appropriate for abdominal surgery, when narcotic analgesia is required, if the patient is not awake enough to manage CPAP, snoring or airway obstruction occurs postoperatively, or episodic desaturations are evident in the PACU. Special monitoring or an ICU admission is warranted if, in addition to symptoms mentioned earlier, preoperative assessment of OSA reveals it to be severe, the patient cannot manage CPAP by themselves, airway management was difficult in the OR, severe obesity exists, the patient is very sedated postoperatively, or pain cannot be managed without opioids. There is no evidence to support risk stratification or the necessity for a period of observation in the ICU depending on whether the patient with OSA is having airway surgery [97,98].

Management of patients with OSA requires judicious use of sedatives. Difficult airway precautions are appropriate including availability of laryngeal mask airway, fiberoptic bronchoscope, and a tracheostomy kit. Short acting agents are preferred. Non-invasive ventilatory devices that can deliver positive pressure to the airway (eg, BiPAP) should be available postoperatively. Non-steroidal analgesics may reduce the need for narcotic-based analgesia.

Summary

Understanding the risk factors for the development of PPCs allows targeted interventions aimed at reducing the frequency and severity of PPCs. The broad categories of what increases the likelihood of developing a PPC are understood but specific understanding of how individual risk factors act to cause PPCs is lacking, and there is little information regarding the interaction or synergy between risk factors. Further research is needed to define the nature of risk factors and develop better predictive models of patients at risk for developing PPCs. It is clear that anesthetic agents produce significant changes in the respiratory system but further information is needed to define how such changes contribute, if at all, to the subsequent development of PPCs. The ongoing controversy regarding the value of regional analgesia or anesthetic techniques, especially epidural analgesia and anesthesia, in reducing or preventing PPCs requires well-done randomized clinical trials. Further research is also needed in the area of postoperative care such as interventions in patients with OSA or the use of inventive spirometric techniques.

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