PREOPERATIVE PULMONARY EVALUATION

UNIVERSITY OF TEXAS SOUTHWESTERN MEDICAL CENTER DEPARTMENT OF INTERNAL MEDICINE GRAND ROUNDS

December 2, 1993

Juliette Wait, MD

Or is the death of a despairing queen Not worth preventing Tho' too well foreseen?

The Aeneid
-Virgil

"In acute diseases it is not quite safe to prognosticate either death or recovery...."

-Hippocrates

Patients subjected to the rigors of a surgical procedure must endure the necessary pain and stress that are involved in the postoperative healing process. Fortunately, most patients tolerate most surgical procedures quite well. Some, however, experience complications which may prolong hospitalization, impair quality or length of life, or result in death. This review will address those factors that contribute to and are predictive of significant postoperative pulmonary morbidity and mortality.

The population of patients that is most likely to experience respiratory difficulties post operatively must be recognized before a study can identify the specific risk factors within that population ¹. This includes patients with underlying pulmonary disorders, those exposed to risks of pulmonary disorders (such as smoking), or those undergoing a type of surgical procedure that is more likely to produce a respiratory complication. Many reports in the literature also intermix true postoperative pulmonary complications with postoperative pulmonary events that may not directly cause morbidity, mortality or even prolong hospitalization. Therefore, one must carefully review the literature regarding postoperative pulmonary complications.

PULMONARY EFFECTS OF ANESTHESIA:

In order to perform a surgical procedure, the patient must be provided with adequate analgesia, muscle relaxation and usually amnesia. This may be accomplished in a variety of ways and with combinations of agents. General anesthesia may be administered as inhalational agents or intravenous agents. The following is a brief list of commonly used anesthetic agent types ²:

<u>Nitrous Oxide</u>: Excellent analgesic properties, some amnesia, no muscle relaxation-low potency - used with other agents

<u>Volatile agents</u>: (halothane, enflurane, isoflurane) excellent, potent, and can be used as sole agent

<u>Narcotics</u>: (morphine, fentanyl, sufentanil, etc.) often used to supplement other agents during induction, do not provide amnesia, little muscle relaxation

Barbiturates: (short acting agents) good amnesia, no analgesia or muscle relaxation

Benzodiazepines: Used as an adjunct. No analgesic or muscle relaxation

<u>Neuromuscular blocking agents</u>: (depolarizing agents) depolarization of the muscle, used for induction of anesthesia and intubation (non-depolarizing agents): fewer side effects

General Anesthesia:

(Impairment of pulmonary gas exchange)

Ventilatory response:

Both inhaled and intravenous anesthetic agents blunt the ventilatory response to hypoxemia. Even very low levels of anesthetic including nitrous oxide can significantly blunt the central response to hypoxemia, while intravenous opiates can affect the peripheral chemoreceptor response³. These effects are profound and can occur at the subanesthetic levels which often persist into the recovery room 3. Thus, a patient with COPD who relies on the hypoxic drive is at increased risk of respiratory failure while receiving oxygen in the recovery room. The ventilatory response to CO₂ is also blunted by inhaled anesthetics in a dose-dependent fashion and is worse in COPD patients 4,5. This may be countered by the ventilatory stimulation that occurs during surgical manipulation. With longer anesthesia time, the inhaled agents demonstrate less blunting of the CO₂ response ⁶. Nitrous oxide is the exception that is known for its sparing effect on the PaCO2, and it is often used with other agents in spontaneously breathing patients 7. The anesthetized, spontaneous breathing patient takes rapid, shallow breaths and no sighs. Narcotics produce slow, deep respirations but can cause apnea in higher doses. These problems are overcome by intubation and mechanical ventilation.

Shunt:

During anesthesia many areas of the lung develop low ventilation/perfusion ratios. This is related to the decrease in the functional residual capacity, FRC, relative to the closing volume, the volume of air remaining in the lung at end exhalation beyond which the small airways begin to close ⁸. The baseline closing volumes may be lower than the FRC in patients with COPD, the elderly and obese. Thus, with the fall in FRC due to anesthesia small regions develop that are not well ventilated or not ventilated at all. These regions may still be well perfused, creating shunts ⁹. Interestingly, although PEEP decreases the shunt, it does not improve oxygenation in this situation perhaps due to a simultaneous decrease in cardiac output ¹⁰. The amount of shunt that develops in those with increased closing capacities may be as much as 11% ⁸.

Some inhalation anesthetic agents, opiates, nitroglycerine, nitroprusside and

other vasodilator drugs used in the operating room on patients with cardiovascular diseases can reverse the normal hypoxic vasoconstrictive response to alveolar hypoxia ^{11,12}. Theoretically, this would increase shunting through the unventilated areas of the lung. In this situation the addition of PEEP has been found to reverse the increased hypoxemia associated with infusions of nitroglycerine ¹².

Ventilation-Perfusion Mis-Matching:

The induction of anesthesia causes a redistribution of ventilation and an increase in alveolar dead space due to more ventilation to the nondependant regions of the lungs ^{5,13}. Intubation and mechanical ventilation does not correct this problem since most anesthesia machines contribute additional dead space, thus adding back the dead space that may have been corrected by intubation. Further, administration of anesthesia to patients with COPD increases dead space to as much as 70% of the tidal volume ⁵.

The reduction in FRC during anesthesia ranges from 11% to as much as 17% ^{5,8,14}. It is caused by many factors: the relaxed diaphragm shifts cranially, the relaxation of the chest wall also decreases the thoracic volume while some blood volume shifts from the thoracic compartment into the abdominal ¹⁴. As a secondary event, areas of atelectasis develop within minutes after induction of anesthesia ¹⁰. These can be prevented by the addition of PEEP.

The inhalation agents can actually cause bronchodilatation by inhibition of airway reflexes, causing dilation of smooth muscles and possibly by increasing beta adrenergic tone ^{11,15}. The inhibition of airway reflexes is also seen with deep thiobarbiturate anesthesia and is due to blocking of acetylcholine release from the nerve terminals of the parasympathetic system ¹⁵. Finally, mucociliary clearance is decreased during and for several days following general anesthesia ^{11,16}. This is often a significant problem for patients with increased mucus production preoperatively such as those with obstructive airways disease.

REGIONAL ANESTHESIA

Spinal anesthesia is sometimes chosen for surgical procedures when general anesthesia is not mandatory. The physiological effects of this type of anesthesia are quite different. Since the diaphragm is innervated by the cervical roots, spinal anesthesia does not impair its function. In addition, the accessory muscles are also innervated by the cervical roots, and are preserved. Nevertheless, the FRC may be reduced with spinal anesthesia. Loss of the abdominal muscle tone decreases forced expiration, hence, there is a reduced ability to cough and clear secretions. This can be detrimental to patients with obstructive airways disease who make use of active expiration and have increased secretions that must be expectorated. Several studies have found that there is no difference between the use of spinal vs general anesthesia in the incidence of postoperative pulmonary complications ¹⁷⁻¹⁹.

Respiratory arrest can occur during high spinal anesthesia and has been attributed to medullary ischemia secondary to systemic hypotension and a decreased cardiac output ². Most studies indicate there is little change in gas exchange during spinal anesthesia ^{18,20}. Regional blocks of extremities have little effect on the respiratory system. One exception is the risk of pneumothorax with a supraclavicular or infraclavicular block of the brachial plexus. A slight risk of also blocking the phrenic nerve exists with an interscalene block.

Although the respiratory risks of the anesthesia appear to be less, the use of spinal anesthesia is not without cardiovascular risks, however. Spinal anesthesia produces vasodilation below the level of the sympathetic block affecting both the arterioles and veins ²¹. Thus, both cardiac preload and afterload are reduced. Cardiovascular effects vary depending on the level of anesthesia in relation to the origin of cardiac sympathetic fibers(T1-T4), absorption of drugs from the peridural space, and whether anesthesia is subarachnoid, or epidural ²¹⁻²³.

CLINICAL PULMONARY COMPLICATIONS:

Evaluation of patients prior to non-cardiac surgery for the risks of developing postoperative cardiac complications such as episodes of heart failure, perioperative myocardial infarction or death has become well established 24,25. On the other hand, the ability to accurately predict postoperative pulmonary complications has remained Part of the difficulty lies in defining what constitutes a relatively imprecise. postoperative PULMONARY complication. Early studies used endpoints such as subsegmental atelectasis, increased sputum production, dyspnea or cough which may not have contributed significantly to the length of the patient's hospital stay or to morbidity. However, complications such as respiratory failure requiring prolonged mechanical ventilation, severe pneumonia that prolong hospital stay or complications that result in death are regarded as having greater importance to the patient and physician, and are obviously those that are to be most avoided. The combined effects of both cardiac and pulmonary diseases also must be considered. A patient who is otherwise considered to be at moderate risk of developing a cardiac event may increase that risk in the presence of post operative respiratory failure and hypoxemia. Finally, many early studies on determining the risk of developing postoperative pulmonary complications did not address the risk of pulmonary thromboembolism.

Modern therapies directed toward reducing postoperative complications in general have changed the approach to the patient who is being considered for a surgical procedure. Improved surgical techniques, early mobilization, and shorter surgical procedures have broadened the ability to offer surgery to patients who may have been rejected as acceptable candidates a decade ago. While prolonged hospital stay, prolonged mechanical ventilation (beyond 24 hours), and death are easily understood complications, comments about other pulmonary complications are in

order.

Atelectasis:

Atelectasis is defined as alveolar collapse. According to Laplace's theory a distensible sphere has an increased surface tension as the radius decreases. Surfactant is produced by the type II alveolar cells and serves to counteract the increases surface tension in the small alveoli. Hence, damage to the type II alveolar cells can cause less surfactant production and atelectasis at a microscopic level. Prolonged breathing at small tidal volumes due to splinting from pain, inadequate respiratory muscle function, compression of the lung from pleural effusions, or pneumothorax and sometimes absorption of high levels of oxygen from poorly ventilated lung units can lead to micro-atelectasis. This is important in that it is a cause of post operative hypoxemia from shunting of blood through some of these atelectatic units with a relatively clear chest x-ray 9,26. Macro-atelectasis, on the other hand is a term applied to atelectasis of larger lung units that are visible on the chest radiograph as segmental or lobar collapse. Sometimes an entire lung is affected. Lobar collapse is usually due to retained secretions, or mucus plugging with or without associated bronchospasm. Additional contributing factors are damage to a phrenic nerve during cardiothoracic surgery, or impairment of the diaphragm following abdominal surgery.

Pneumonia:

The presence of fever, purulent secretions, leukocytosis and a pulmonary infiltrate on chest radiograph is most commonly used as a definition of a pneumonia. However, this definition has recently been questioned when applied to patients in intensive care units on mechanical ventilators. Using bronchoscopic cultures of lungs with a telescoped protected brush, Fagon, et al., determined that as few as 35% patients on mechanical ventilators actually had bacterial pneumonia ²⁷. Most importantly, those who did not have positive cultures and were not treated with antibiotics had favorable outcomes. Since only half of the patients in Fagon's study were postoperative, it has not been determined if this definition could be applied to all postoperative surgical patients on mechanical ventilators. It does raise question regarding the clinical definition of bacterial pneumonia in mechanically ventilated patients. In spontaneously breathing patients, however, retained secretions, atelectasis and poor cough can promote bacterial colonization of the lower airway and development of pneumonia.

Respiratory Failure:

An acute rise in the arterial pCO₂ with associated hypoxemia and acidosis is recognized as a definition of acute respiratory failure. Postoperative causes are multiple: ventilatory insufficiency in resectional surgery, over sedation from prolonged anesthesia effect or pain medications, exacerbations of obstructive airways disease, dysfunction of the respiratory muscles, volume overload and/or myocardial pump failure.

ARDS may develop in the post operative patient for a variety of reasons. Commonly it is related to the original need for surgery, such as trauma, massive transfusions, head injuries or sepsis syndrome in a patient with an abdominal abscess, infarcted bowel, etc. Thorough discussion is beyond the scope of this topic. However, patients with ARDS postoperatively are usually considered to have suffered a postoperative pulmonary complication.

Pulmonary Thromboembolism:

This is a well recognized postoperative complication which has been stratified into those patients and surgical procedures with increased risk factors. The incidence of postoperative thrombosis in general surgery patients can be lowered by use of several different effective measures ²⁸.

Classification of the Risk of Postoperative Venous Thromboembolism 29

	Risk of Venous Thromboembolism, %				
Risk Category	Calf Vein Thrombosis	Proximal Vein Thrombosis	Fatal Pulmonary Embolism		
High risk General surgery in patients >40 yr with recent history of DVT or PE Extensive pelvic or abdominal surgery for malignant disease Major orthopedic surgery of lower limbs	40-80	10-20	1-5		
Moderate risk* General surgery in patients >40 yr lasting 30 min or more	10-40	2-10	0.1-0.7		
Low risk Uncomplicated surgery in patients <40 yr with no additional risk factors Minor surgery (ie, <30 min) in patients >40 yr with no additional risk factors	<10	<1	<0.01		

^{*}Risk is increased by advancing age, malignancy, prolonged immobility, varicose veins, and cardiac failure.

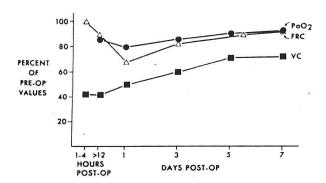
EFFECTS OF TYPES OF SURGERY ON THE RESPIRATORY SYSTEM:

Early studies demonstrated that the type of surgery was a major factor in determining whether or not the patient would suffer from a postoperative pulmonary complication. Consequently the different types of surgery will be addressed separately:

Upper Abdominal Surgery:

When specifically sought, the pulmonary complications following surgery to the upper abdomen is often greater than 20% ³⁰. The most common reported complication is atelectasis which often is of little clinical significance ³¹. The separation of upper abdominal surgery from lower abdominal surgery is warranted here since it is known that the postoperative complication rate is higher in the former ³⁰. One of the mechanisms contributing to this difference is the effect of upper abdominal surgery on the respiratory muscles.

Although the minute ventilation, the volume of air moved per minute, remains the same following abdominal surgery the tidal volume falls and the respiratory rate increases. This particular breathing pattern persists for several days up to one week and is associated with a fall in the vital capacity (see figure) 32,33.



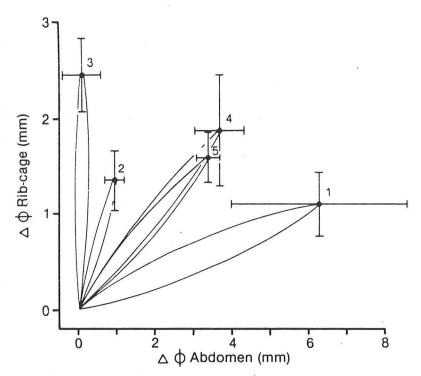
Post operative VC, FRC, and PaO, (on room air) as percent of preoperative values.

As early as 1914 Pasteur noted that patents developed a paradoxical breathing pattern following upper abdominal surgery which he attributed to diaphragmatic paralysis or weakness ³⁴. During normal breathing there is a tight link between both the inspiratory accessory neck muscles and intercostal muscles which stabilize and elevate the rib cage, to the diaphragm which descends during contraction thus further expanding the thoracic volume. Patients with weak diaphragms may contract the diaphragm and abdominal muscles during expiration pushing air out of the lung so that

the following inspiration begins at a lung volume below the normal FRC. At the beginning of inspiration the relaxation of the expiratory muscles is associated with a fall in the gastric pressure which is transmitted to the pleural space and expansion of the lung occurs. Also, as noted by Pasteur, the weakened diaphragm may not contract completely and the negative pressure formed by contraction of the intercostal and accessory muscles in the neck serve to 'suck' the diaphragm into the chest. Hence, the inspired volume is less, the vital capacity falls and there may be an increased tendency to collapse alveoli.

Using magnetometers and measurements of gastric and pleural pressures the breathing patterns of patients following upper abdominal surgery were characterized ^{32,35}. (see diagram):

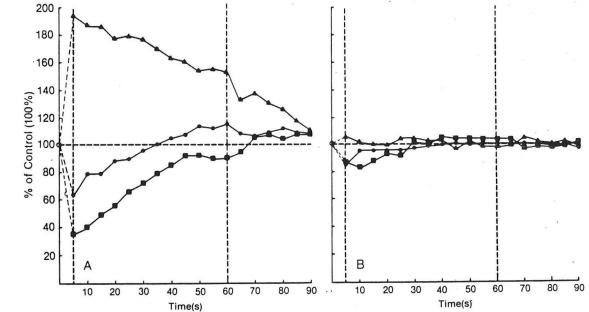
Konno-Mead diagram of chest wall configuration in patients undergoing cholecystectomy. Change in ribcage diameter (Δ φRC) was plotted against change in abdominal diameter (A φAbd) for each of the designated time periods. Each loop represented a tidal breath. These data showed a shift from predominantly abdominal breathing to ribcage breathing and back again. Bars indicate ± SEM. 1 = Control; 2 = 2 - 4hours postoperative; 3 = 6 - 8hours postoperative; 4 = 24 hours postoperative; 5 = 48hours postoperative.



Change in rib cage diameter was plotted against change in abdominal diameter for each of the designated time periods. Each loop represented a tidal breath. These data showed a shift from predominantly abdominal breathing to rib cage breathing and back again. from reference ³⁵

All patients shifted to a pattern of breathing that increased the rib cage excursions. Other studies have demonstrated that patients may also adopt a pattern with increased use of the abdominal expiratory muscles as described above ^{36,37}. Furthermore, other factors may decrease the function of the diaphragm post operatively such as hypophosphatemia and hypocalcemia ^{38,39}.

Studies in anaesthetized animals (dogs and cats) demonstrated that there is a reflex inhibition of neuronal stimulation to the diaphragm with manipulation or stimulation of abdominal viscera, and in humans this inhibition can be reversed by administration of epidural anesthesia ^{37,40}. This effect can also be found with distention of the esophagus in animals and humans ^{41,42}. General anesthesia itself has not been found to produce the same findings. Also, laparotomy causes an inhibition of the expiratory abdominal muscles which are needed for coughing ⁴³. Whether or not these physiological responses to abdominal surgery are linked as a cause and effect to the decrease in vital capacity and formation of atelectasis is speculative. These changes appear to be due to reflexes rather than pain alone. However, this breathing pattern is likely to promote atelectasis which may in turn lead to retained secretions and possibly pneumonia in the postoperative patient.



Intercostal muscle activity, phrenic nerve activity, and tidal volume during dilation of the small intestine. Closed triangles show intercostal muscle activity and closed squares show the decrease in phrenic nerve activity with a slight decrease in tidal volume (closed circles). A. Before and B. after mesenteric denervation 40

Laparoscopic cholecystectomy:

The newer technique of laparoscopic cholecystectomy has been reported to be associated with fewer postoperative respiratory complications ⁴⁴. Comparisons to open surgical techniques may be difficult since patient selection and follow up of

patients in different studies may not be similar. Nevertheless, studies have revealed improved postoperative lung function after laparoscopic surgery ^{45,46}. These results seem logical since this technique involves smaller incisions in the abdominal muscles and less manipulation of the abdominal organs which helps minimize the adverse effects of the surgical procedure on the patients. Finally, early ambulation and discharge from the hospital (mean hospital stay of 1.2 days) probably contribute significantly to a decreased incidence of postoperative complications of laparoscopic procedures⁴⁴.

Non-pulmonary Risk Factors:

"Hence a small disturbance will speedily cause death in old age."

On Youth and Old Age
-Aristotle

Several studies ⁴⁷⁻⁴⁹ have examined the non-pulmonary factors that contribute to postoperative pulmonary complications in the patient undergoing upper abdominal surgery: While these factors may have a relatively good positive predictive value, they do not have a good negative predictive value ^{47,48}. The non pulmonary risk factors are listed below:

Duration of surgery (greater than 2 hours)
Age (greater than 60 years)
Presence of significant medical disease (ASA classification > 1)*
Gender (male)
Smoking

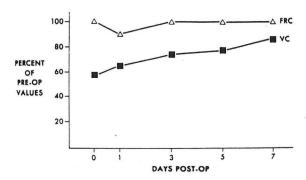
The ASA (American Society of Anesthesiology) score is used by anesthesiologists to determine preoperative risks based on simple clinical criteria. Although this scoring system has been criticized for its subjectivity, most would agree that any score above 1 is relatively easy to identify ⁵⁰.

*ASA score: class 1 = healthy, class 2 = mild to moderate systemic disease, class 3 = severe systemic disease, class 4 = severe systemic disorders that are already life threatening, and class 5 = moribund

Not listed in this scoring system is the nutritional state of the patient, which may be linked to underlying illnesses. Nevertheless, it is relevant to the development of postoperative complications ⁵¹.

Lower Abdominal Surgery:

A very low risk of pulmonary complications have been found in studies on patients with surgery to the lower abdomen (appendix, inguinal hernia, female and male genital tracts) ^{49,52}. The effects of this type of surgery on the postoperative ventilatory pattern are similar to but much less than those involving the upper abdomen ⁵². These patients may still be at an increased risk for postoperative deep venous thrombosis and pulmonary embolism.



Postoperative VC and FRC (as percent of preoperative values) following lower abdominal surgery. from ref 52

Extremity Surgery:

In general, the incidence of significant pulmonary complications such as pneumonia or respiratory failure are not significantly increased, even in those patients with underlying pulmonary disease. Cardiovascular diseases often are the primary risk determinants in medical patients needing extremity surgery. Surgery on the hip or lower extremity carries an increased risk for post operative DVT and pulmonary thromboembolism. The incidence is highest in those receiving orthopedic surgery to the lower extremity where the incidence of lower extremity clot approaches 50-60% ⁵³. The incidence of severe or fatal pulmonary thromboembolism in these groups may approach 5% if routine preventive measures are not used ^{29,53}.

Head and Neck:

Surgical procedures on the head and neck for non-malignant conditions are not associated with increased pulmonary morbidity or mortality. Complications are more likely to be related to the type of and duration of anesthesia. Cancer surgery, on the

other hand, is often extensive and performed on older patients. Carcinomas of the head and neck region are usually found in heavy smokers who also have a higher probability of having significant obstructive airways disease, although these patents usually tolerate radical surgical procedures without significant respiratory difficulties. The exception to this would be the anticipated alteration of the upper airway which predisposes these patients to aspiration of oral contents or food. The presence of increased mucus production preoperatively may cause increased problems with removal of secretions post operatively. However, there are few studies that indicate there is an increased incidence of postoperative respiratory complications in these patients. Neurosurgical patients also may have decreased ability to cough, and due to altered mental status, may have lower tidal volumes and an increased risk of aspiration of oral contents.

THORACIC SURGERY:

Thoracotomy:

With simple thoracotomy, the chest wall sustains the majority of the injury, resulting in a decrease in chest wall compliance ⁵⁴. There is a reduction in vital capacity and alveolar ventilation with subsequent hypoxemia. These changes are most marked at 2 to 3 days postoperatively, but may persist longer. The decrease in chest wall compliance and vital capacity is often aggravated by pain. Attempts to control the pain with narcotics may further depress respiration and promote atelectasis, however.

Cardiac Surgery:

Left lower lobe atelectasis is found up to 90% of the time and is such a common finding in postoperative cardiac patients that some don't even consider this to be a complication unless it interferes with the postoperative recovery and discharge of the patient 55. The causes are multifactorial, but damage to the phrenic nerve during surgery by using iced slushes to decrease myocardial oxygen demands may contribute significantly 55. Damage to the nerve may last from 30 days to as long as 2 years, and is associated with an elevated left hemidiaphragm ⁵⁶⁻⁵⁸. The incidence ranges from 26% to 73%, but is decreased if pericardial insulation is used 59,60. Other operative factors associated with damage to the phrenic nerve are longer bypass times, entrance into the pleural cavity, or direct damage from mobilization of the internal mammary artery ⁵⁹. The internal mammary artery lies close to the phrenic nerve at the thoracic inlet where manipulation can result in inadvertent damage to the phrenic nerve. Bilateral phrenic nerve damage is much less common, but is associated with greater morbidity, often requiring prolonged mechanical ventilation 60. A recent study of patients with COPD found spirometry was not an independent predictor of post operative pulmonary complications in cardiac surgery 61. The use of

bronchodilators and any abnormality (including hyperinflation) on the chest x-ray predicted serious complications or death in 33% ⁶¹.

Esophageal Surgery:

Pulmonary complications defined as respiratory infections, respiratory failure or death from respiratory causes are high in patents receiving thoracotomy for resection of esophageal carcinomas. Age greater than 65, poor nutritional status and hypoxemia were the best predictors of these complications ⁶². These risks are usually greater because these surgical procedures are often very long, may involve procedures below as well as above the diaphragm and these cancers often occur in older patients who have significant smoking histories which predisposes them to obstructive lung diseases and cardiovascular diseases ⁶².

Video Assisted Thoracoscopic Surgery/Thoracoscopy:

Recently there has been a resurgence of a previously abandoned technique, the use of the thoracoscope to perform surgical procedures in the chest. This procedure was used frequently in the 40's and 50's in treatment of the tuberculous pleural space, but abandoned in favor of larger incisions to allow the surgeon better access and hemostasis. However, newer technologies such as the videoscope, the staple gun and the laser knife now allow thoracic surgeons to operate through significantly smaller incisions. Not only are the size of the incisions smaller, the recovery and hospitalization time shorter, but the complication rate also appears to be reduced 63. Examination of the pleural space, biopsies of the pleura and lung, surgical staging of the mediastinum, some esophageal surgeries, surgical resection of solitary masses, pericardial windows and lobectomies are now performed using this technique 63-66. Certainly, the hospitalization time is significantly reduced to 3 to 5 days as compared to 8 to 10 days for larger procedures 67,68. Again, the lower complication rate is partly due to the earlier mobilization and discharge of the patients from the hospital. The smaller incisions, performed without spreading of the ribs, and limited manipulation of internal organs may also contribute to less postoperative pain and quicker recovery. It is likely that this technique will allow many patients with otherwise prohibitive lung function to receive surgery to the thorax, including some resections of peripheral lung cancers 66,69.

Lung resection surgery:

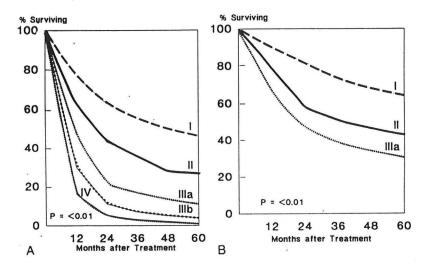
Today resection of the lung is primarily done for bronchogenic cancer. In the 1940's and 1950's lung resection surgery was performed for infectious processes such as abscesses, bronchiectasis or tuberculosis. Thoracoplasty, chest wall collapse of 5 to 7 ribs, was often performed in tuberculosis patients. The intent was to prevent problems with residual air spaces in the thorax and to prevent over distention

of the remaining lung. Unfortunately this procedure sometimes resulted in progressive scoliosis to the operated side and further respiratory embarrassment, so this procedure has been largely abandoned 70. In 1955 Gaensler reported a 40% mortality following thoracoplasty or resectional surgery for tuberculosis in patients with decreased lung function 71. Over the years several attempts have been made to predict which patients would best tolerate resectional surgery of the lung. Unfortunately, some of the earlier studies did not effectively separate cardiac events from pulmonary in their assessment of post operative complications in thoracic surgery patients 72-74. A recent multicenter study of more than 12 thousand thoracotomies performed between January 1983 and December 1986 examined the incidence of in-hospital mortality for lung resectional surgery regardless of the criteria used for selection of patients. They found in-hospital mortality of 3.8% after wedge resection, 3.7% after segmental resection, 4.2% after lobectomy and 11.6% after pneumonectomy 75. multivariate regression analysis, the significant predictors of in-hospital mortality were age greater than 60 years, male gender, extended resection, chronic lung or heart diseases, diabetes and size of the hospital 75. These figures are higher than those in some reported series 76-78, but they are not far from others 79,80.

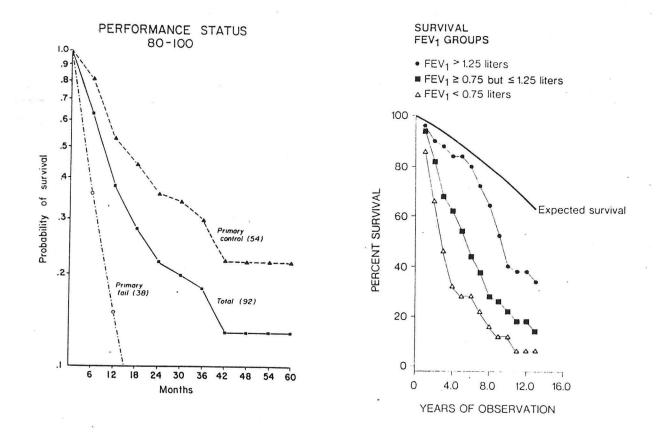
In addition to the above association between extent of pulmonary resection and morbidity, there is a significant early loss of lung function with lobectomy that improves with time ^{76,81,82}. Hence, patients that have marginal lung function may have greater difficulties immediately following lobectomy than anticipated based on the predicted postoperative lung function. This does not occur with predictions for pneumonectomy patients, however.

Long term survival:

Complete resection of a surgical Stage I or II non-small cell lung cancer results in a predicted five year survival of 70 to 50% respectively 83,84 . However, one may question the validity of the procedure if that same curative surgery causes severe postoperative morbidity, mortality or leaves the patient incapacitated from loss of lung function. The mortality of severe obstructive lung disease with a forced expired volume in one second, FEV₁, less than 0.8L/sec (in an adult male) with associated cor pulmonale is relatively high, with a five year survival of only 20-30% 85,86 , which in some series approaches that of unresectable lung cancer in relatively healthy individuals treated with radiation 87 .



Cumulative proportion of patients with non-small cell lung cancer expected to survive 5 years or more according to clinical (A) and surgical (B)(pathological) stage, excluding operative deaths. from ref ⁸⁴



A. Effect of local control with radiation therapy on survival of patients with inoperable lung cancer with high performance status (80-100) from ref ⁸⁷, and B. survival of COPD patients grouped on the basis of FEV1 at time of enrollment in the study. from ref ⁸⁵.

Predicting Post Operative Lung Funtion:

Thus, whenever lung tissue is to be removed, serious consideration needs to be made as to how much lung function will remain. Over the past several years improved techniques for making this prediction have been described. Earlier studies used bronchospirometry or the lateral position tests to determine the function of each lung so that a predicted value after pneumonectomy could be determined 88,89. Difficulties in performing the tests and lack of reproducibility of the lateral position tests 90 resulted in them falling out of favor. However, in an attempt to predict the postoperative lung function, quantitative lung scanning has been used to determine the function of a lung or a portion of a lung. Radioactive xenon ventilation scanning has been used to predict the volume of lung that was to be resected 81,91. A good correlation between the predicted post operative lung function was found in both studies. These correlations were pertinent only for pneumonectomy or lobectomy, and not so for smaller resections 81. In 1980 Wernly, et al examined both ventilation and perfusion scans to predict postoperative lung function in lobectomy and pneumonectomy patients and found no difference in predictive value with either method ⁹². These good correlations have been found in other studies ^{80,93-95}. Although this method was good for predicting postoperative lung function, it was less satisfactory as a predictor of complications. The greatest variability among these studies was the use by some of a predicted postoperative FEV₁ value less than 0.8L/sec as a lower limit to deny surgery. This may have been unfortunate for the female patients in these studies because an FEV₁ of 0.8 L/sec could be nearly normal for a small woman of 55 years. It is now suggested that the absolute postoperative predicted FEV₁ be determined, then examined as a percent of the normal predicted value for that patient.

One method of using the quantitative lung scan to calculate the postoperative FEV_1 is as follows:

The expected loss of function = $Preop FEV_1 X$ (% of function of the affected lung from the lung scan)

= (Number of segments in lobe to be resected)
(Total number of segments in the whole lung)

Thus, quantitative lung scanning predicts the postoperative lung function, but it does not accurately predict postoperative morbidity or mortality ^{80,81,96}. One study noted the agreement of the perfusion lung scans in estimating changes not only in spirometric indices such as FEV₁, MVV, FVC, but also the diffusing capacity of the lung and the exercise capacity ⁹⁵.

EVALUATION OF PATIENTS FOR SURGERY:

CARDIOVASCULAR ASSESSMENT:

The most important factors in postoperative morbidity and mortality are those due to postoperative cardiovascular events. The risks for these complications can be adequately assessed using easily determined criteria ^{24,25}. Discussion of this topic is beyond the scope of this review, but these factors must also be considered when evaluating patients for surgery.

PULMONARY FUNCTION STUDIES:

Low Risk Surgery:

Patients who are about to undergo surgical procedures that are known to be very low risk for pulmonary complications do not need preoperative pulmonary function studies. Exceptions may be made to this generalization for those patients with advanced age, known pulmonary disease and anticipated prolonged anesthesia time or anticipation of significant postoperative pain or prolonged bed rest. The established preoperative lung function may be used as a guide for the immediate pre and post operative care.

Upper Abdominal Surgery and Thoracotomy:

These procedures as stated above are associated with a significant rate of postoperative pulmonary complications. Knowledge of the extent of the pulmonary dysfunction before surgery will lead to increased post operative vigilance, although they may not predict postoperative complications ⁹⁷.

Lung Resection Surgery:

Lung tissue is also removed in addition to the alterations of chest wall and respiratory mechanics from the thoracotomy. Many studies have used pulmonary function studies both to determine how much lung can safely be removed and as predictors of postoperative morbidity. Boushy et al found that an FEV₁ less than 2L and an FEV/FVC less than 50% in patients over 60 years old was associated with both a higher postoperative mortality following surgery and worsening dyspnea in 40% of these patients ⁹⁸. Similar findings were obtained by others ⁷⁴. In 1973 Lockwood extensively analyzed pulmonary function studies to identify those at high risk for resectional surgery ⁹⁹. Lockwood's results, however, were less than satisfactory and Keagy et al found no predictive value from lung function studies ¹⁰⁰. Nevertheless, some predictive value appears to exist for obtaining pulmonary function studies for those patients about to undergo resection surgery of the lung. Age less than 60 with normal lung function and less extent of surgery are expected to have a

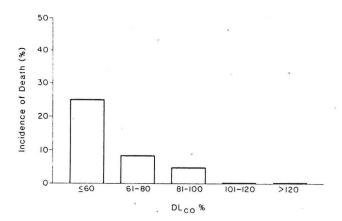
significantly lower mortality. Very low complication rates were reported in one study that stratified the patients according to their pulmonary function studies and anticipated extent of surgery ⁷⁸. Unfortunately, these rates were not reproducible with other studies, although the values of PFTs proposed in this study were often used as criteria for resectability.

Routine spirometry is usually performed on most adults considered for resection of the lung. Based on the predictive factors for postoperative morbidity and mortality, all patients with known or suspected underlying pulmonary disease, cardiovascular disease, diabetes, need for extensive surgery or age greater than 60 years should have pulmonary function studies performed. These should include the forced expired volume in one second (FEV₁), the forced vital capacity (FVC), and the single breath diffusion capacity of the lung. Arterial blood gas analysis has not been shown to be of predictive value for post operative complications, but is often obtained to assist with post operative management ⁴⁹. The exception is a stable PaCO₂ > 45 which has been noted to be associated with respiratory complications ⁹⁷.

Resection of a lung or portion of a lung may cause respiratory embarrassment if insufficient lung parenchyma remains to accommodate the demands of the postoperative course or of daily life. Fortunately, the normal pulmonary circulation has the capacity to vastly expand to meet the demands of pneumonectomy where the remaining lung must suddenly receive the entire cardiac output. If underlying pulmonary vascular disease exists, such as from recurrent pulmonary emboli or destruction from emphysema causing loss of the pulmonary capillary bed, an increase in pulmonary vascular resistance can occur after resectional surgery. This can precipitate acute right heart failure and increased morbidity or mortality beyond hospital discharge. It was this understanding that precipitated several studies that measured the pulmonary vascular resistance or pulmonary artery pressures via right heart catheterization in order to predict the postoperative morbidity and mortality in patients undergoing resectional surgery of the lung 101-106. An increase in the PVR or mean pulmonary artery pressure either with balloon occlusion of the pulmonary artery on the side to be resected or an increase during exercise was found to be associated with a poor prognosis 102,105,106. One study noted pulmonary artery hypertension developed over time after pneumonectomy although immediate post operative values were not obtained 104.

Since balloon occlusion of the pulmonary artery is necessarily invasive and carries a certain morbidity and mortality, it is not routinely used. The first studies of this nature were described in 1951. Since then, recognition of the importance of the pulmonary circulation has increased and other methods to indirectly examine the pulmonary vascular bed have proven more useful. Ferguson, et al found this to be measurement of the diffusing capacity of the lung for carbon monoxide (DLCO) which served as a good predictor of mortality following resectional surgery ⁷⁹.

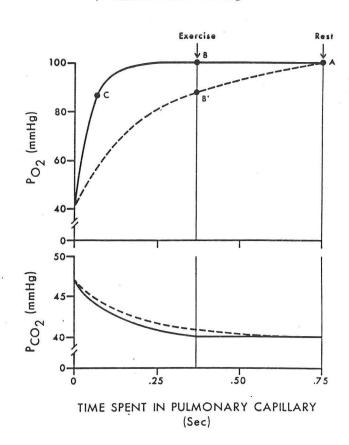
A retrospective review of 237 patients who underwent lobectomy, bilobectomy, or pneumonectomy in a large volume Chicago hospital was conducted with logistic regression analysis to determine the most important predictor of morbidity and mortality. Of all the parameters examined, the DLCO corrected for lung volume and hemoglobin was the best predictor of mortality and the only parameter that predicted postoperative pulmonary complications ⁷⁹. (This has been further substantiated by examination of a larger population of patients by the same investigator). Other studies have not found this correlation, or found that a decreased DLCO only predicted postoperative morbidity, not mortality ^{76,107-109}. These may be due to differences in reporting the DLCO as an absolute value vs corrected for Hb and volume. Rejection of patients for surgery with low DLCO would result in a decrease of post operative mortality by 33% and postoperative complications of 14%.



Prevalence of mortality after major pulmonary resection versus DLCO% for 165 patients. From ref 79.

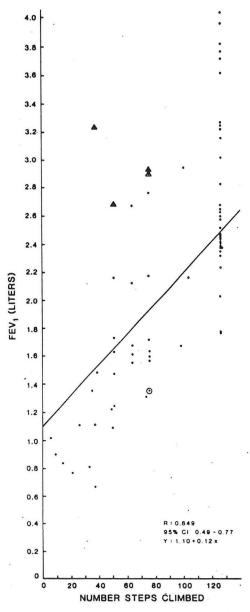
A low DLCO also correlates with a fall in the arterial pO₂ during exercise which is demonstrated in the diagram below from Murray, The Normal Lung ¹¹⁰:

Point A represents equilibration of oxygen in the blood and alveoli at rest, Point B represents increased blood flow through the capillaries during exercise in normal individuals and demonstrates that equilibration of oxygen still occurs. Point B also represents the increased blood flow in the remaining vessels under resting conditions in individuals with loss of the pulmonary capillary bed. When these individuals exercise, the blood flow increases further, no equilibration occurs and oxygen levels fall, Point C. Point B' represents the fall in oxygen level during exercise in individuals with a decreased diffusing capacity due to a thickened airblood barrier.



EXERCISE TESTING:

Whether postoperative morbidity or mortality from resectional surgery could be best predicted by exercise testing preoperatively was addressed by Reichel in 1972 ¹⁰⁷. They found increased postoperative pulmonary complications in many of the patients who failed to complete a graded incremental exercise protocol (37). Many subsequent studies have been reported with varying degrees of predictability which range from no predictive value 111,112, to those that were able to stratify the likelihood of postoperative complications and morbidity on the level of exercise achieved 108,109,113-115. Unfortunately, the studies which did not find predictive value either included surgical complications (wound infection) or did not well define the postoperative pulmonary complications. Hence, it is likely that there may be some value in performing some form of evaluation of the patient's preoperative exercise capacity. This may be particularly true for those patients whose static lung function studies are borderline, allowing some patients to undergo surgery who may have been rejected on the basis of the static lung function studies 116. Although not used to predict postoperative outcome, one study found the number of stairs climbed correlated well with preoperative spirometry, which is useful to assess those patients who cannot perform simple spirometry, e.g. those with laryngectomies ¹¹⁷.



Forced expiratory volume in one second compared to number of stairs climbed.

Postoperative exercise testing revealed that five of eight younger patients recovered from extensive resectional surgery for tuberculosis were found to have their maximal exercise capacity limited by cardiac output, but not by impaired ventilation ¹¹⁸. This suggests that careful attention needs to be placed on the preoperative condition of the cardiovascular system, and of other pre-existing diseases which may cause postoperative morbidity, mortality or long term functional impairment ¹⁰⁷. A right ventricle that is working against an elevated pulmonary vascular resistance will limit the cardiac output during exercise ¹¹⁹. The normal pulmonary vascular bed dilates during exercise to accommodate the increased blood flow. If this capacity is limited, there will be a failure of the cardiac output to also increase further during exercise. Blood cannot get from the right heart to the left heart, and pulmonary artery

pressure rises. Since the DLCO is directly related to the volume of the pulmonary capillary bed, it is logical that a low preoperative value of the DLCO may herald difficulties with the pulmonary circulation postoperatively. Furthermore, it is also logical to note that the general pre operative medical condition of the patient may also indicate that some difficulties with the cardiovascular or ventilatory system will occur. A low level of attained VO₂ with exercise may reflect a poor cardiac output, deconditioning, or poor motivation of the patient ¹¹⁹. In summary, a VO₂ greater than 20 ml/kg/min. demonstrates good cardiovascular reserve and a low risk for resectional surgery. Similarly, a very low VO₂ also correlates with a greater incidence of postoperative complications.

The following is a chart of selected studies using exercise protocols to assess post operative pulmonary complications.

Reference	# Patients	Mean Age (yrs)	Type of Resection	Complications ¹	No Complications	Death	Predictive Parameters [VO ₂ ml/kg/min]	Type of Exercise
(116)	54 .	60	W,L,P	²2.3 .7	N/A N/A	1/7 0/47	<3 flights stairs >3 flights stairs	Stairs Stairs
(96)	28	64	W,L,P	N/A N/A	N/A N/A	5/8 1/20	<9 >9	Ergometer 2 work levels
(114)	22	56	W,L,P,T	*6/6 *1/10	0/6 9/10	2/6 0/10	<15 >20	Ergometer to max attained
(113)	50	64	W,L,P	*5/7 *3/29 *0/14	2/7 . 26/29 14/14	2/7 0/29 0/14	<10 >10 <20 >20	Ergometer to max attained
(120)	42	63	W,L,P	*8/14 *6/14	10/28 18/28	N/A N/A	<15 >15	Ergometer to max attained

¹Atelectasis, respiratory failure, pneumonia

N/A = not available

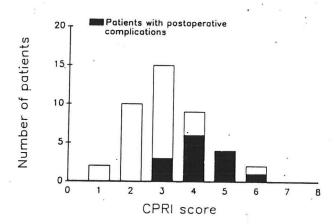
²Complication rate

W = wedge, L = lobectomy, P = pneumonectomy, T = thoracotomy

^{*}Including cardiac complications & PE

Most studies searching for a method to predict postoperative morbidity or mortality examine a large array of pulmonary function studies in hopes of finding the best test. Two recent studies attempted to predict both cardiac and pulmonary complications by combining exercise testing and assessment of cardiovascular risk factors which were modified from the Goldman criteria 120,121. One study, Gerson, et al, examined older patients, mean age 73, undergoing primarily abdominal surgery 121. The other, by Epstein et al, studied slightly younger patients, mean age 63, undergoing pulmonary resection ¹²⁰. The Gerson study found the inability to exercise 2 min to a heart rate >99 with one Goldman risk factor, was the best predictor of cardiopulmonary complications 121. The Epstein study found a decreased exercise capacity was also associated with greater morbidity, but this was primarily reflective of the underlying medical status of the patient. It was not an independent predictor of morbidity and added nothing to their other criteria for predicting morbidity 120. Further examination of Gerson's study demonstrates that the inability to exercise was not determined by dyspnea, but by other predictable factors that limit exercise, such as amputation, inability to comprehend the study, etc. The Epstein study utilized an index based on the Goldman criteria and another pulmonary index, which they determined to be the CardioPulmonary Risk Index, the CPRI. The CPRI > 4 indicated those patients who were 22 times more likely to develop postoperative complications and prolonged hospital courses 120. The level of VO2 attained during exercise was lower in those with higher CPRI. Mortality could not be well assessed since only one death occurred.

Number of patients with significant postoperative pulmonary complications increased with increasing CPRI score.



CPRI variables:

Variable:	Points	Variable	Points	
1)Congestive Heart Failure ¹				
(S3, jugular venous distension, LVEF < 40%)	11	1) obesity (body mass index > 27kg/N	1 ² 1	
2) Myocardial infarction during previous	10	2) cigarette smoking within eight week	s	
six months		of surgery	1	
	3) productive cough within fiv		1	
		of surgery		
3) Greater than 5 PVCs/min	7			
(noted at anytime preop)				
Rhythm other than NSR or PACs (on preop ECG)	7	4) diffuse wheezing or rhonchi	1	
5)Age > 70 years	5	5)FEV1/FVC < 70%	1	
6) Important valvular aortic stenosis ¹	3	6) PaCO2 > 45 mmHg	1	
7) Poor general medical condition ¹	3	_		
		*		
Cardiac risk points = 3-47 [each patient				
assigned 3 points for t	Pulmonary Risk Index Score (PRI) = 0-6			

CARDIOPULMONARY RISK INDEX SCORE (CPRI) = CRI + PRI = 1-10 points

CRI score = 1 (3-5 points), 2 (6-12 points), 3 (12-25 points)

RECOMMENDATIONS FOR PREOPERATIVE EVALUATION:

Smoking cessation:

Cardiac Risk Index Score (CRI) = 1-4

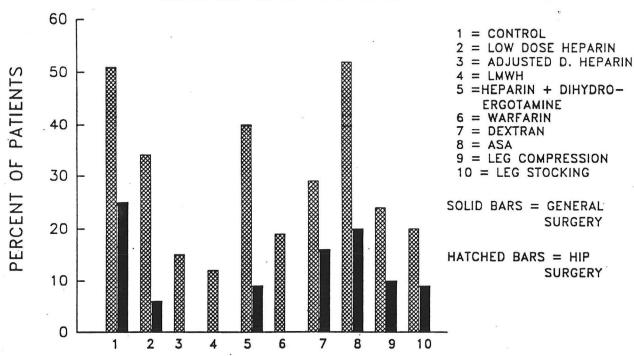
4 (> 25 points) 1 = reference 24

Studies on postoperative pulmonary complications in patents undergoing cardiovascular surgery found a positive correlation with smoking prior to surgery. Cessation of smoking was found to decrease the incidence of postoperative complications if it took place more than 8 weeks prior to surgery 122. This is likely related to the normal mechanism of pulmonary clearance. In fact, complications were noted to increase when the patents had sustained from smoking for approximately 2 weeks 34. Of note, the definition of complications included relatively minor events, and there was no evidence that hospitalization was increased. Thus, it is ideally recommended that patients who will undergo purely elective surgery sustain from smoking for 8 or more weeks. It is also recognized that patients in need of semi urgent surgery need not have their surgery delayed, but they should nevertheless quit smoking for as long as possible. This is to decrease the cardiac effects of nicotine and increased carbon monoxide levels 123. Cessation of smoking for at least 24 hours is recommended.

DVT prophylaxis:

Prevention of deep venous thrombosis is recommended for all patients undergoing major surgical procedures, particularly those noted to be associated with a high risk of formation of deep venous clots. Many different measures can be utilized with varying degrees of success, sometimes depending on the type of surgical procedure performed. Subcutaneous heparin, pneumatic compression boots are among the best and most commonly used methods. Below is a bar graph compiled from multiple studies, on the prevention of postoperative venous thrombosis ^{28,53}:



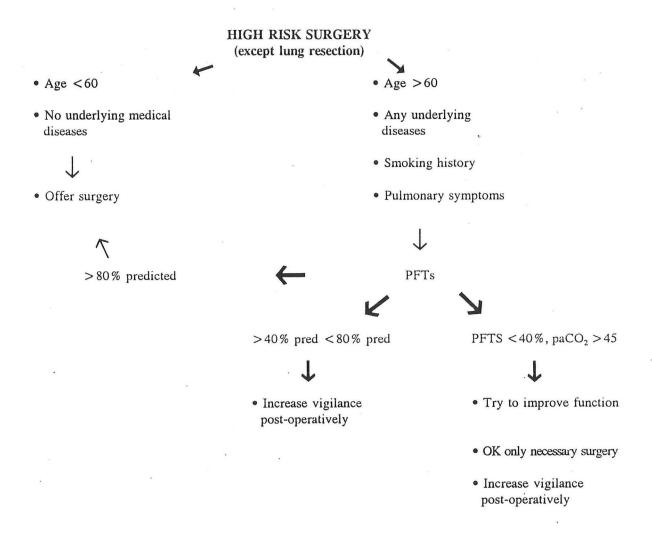


LOW RISK SURGERY

age < 60, no underlying medical diseases, and/or very low risk surgery = OK for surgery

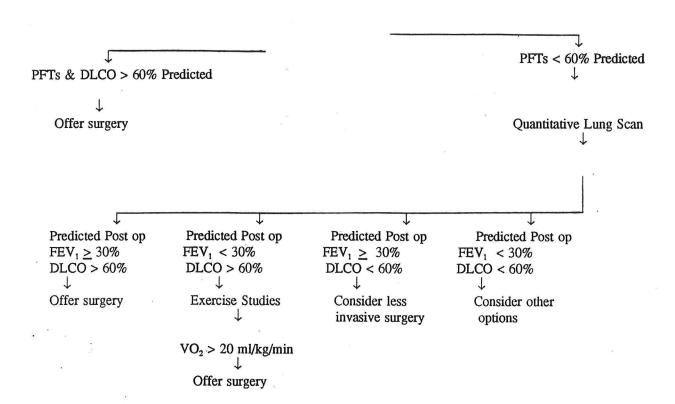
age > 60 + pulmonary symptoms (cough, wheezing, dyspnea) = may do PFTs, cardiovascular risk evaluation and monitor post op

The following are flow diagrams for assessment for high risk surgery and lung resection surgery:



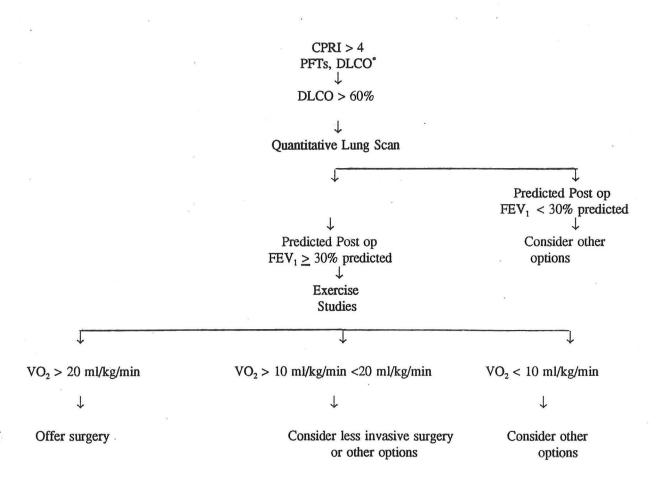
LUNG RESECTION

CPRI ≤ 3 PFTs, DLCO*



^{*} Corrected for Hb, lung volume

LUNG RESECTION



^{*} Corrected for Hb, lung volume

POSTOPERATIVE MANAGEMENT:

Postoperative management actually begins with the initial consideration for surgery and the type of surgery to be performed. Management is directed toward reduction in the anticipated postoperative complications and the mechanisms that are known to promote them. These measures are useful in decreasing postoperative pulmonary complications in patients with respiratory disease ¹²⁴. Most of these recommendations are based on common sense and a little knowledge of the expected complications.

Preoperative:

Upper abdominal surgery with increased risk factors for pulmonary complications; or any surgery with anesthesia time greater than 2 hours in a patient with known lung disease or pulmonary symptoms.

- 1. Incentive Spirometry or training on deep breathing exercises
- 2. Use of bronchodilators if clinically indicated (wheezing, productive cough) use beta agonists for asthmatics, and atropine analogues for elderly patients with COPD.
- 3. Delay surgery if cough is productive until antibiotics have had an effect on the bronchitis (1-2 weeks)
- 4. If patient is asthmatic with PFTs < 80% predicted, consider administration of iv solumedrol (40-80 mg iv) the morning of surgery
- 5. If patient has obstructive lung disease and is on theophylline, it may be administered iv just prior to surgery to maintain adequate blood level (0.4-0.5 mg/kg/hr).
- 6. Begin prophylaxis for DVT on day of operation if major procedure is to be performed.

Postoperative:

- 1. Continuation of steps initiated preoperatively such as bronchodilators, DVT prophylaxis, and steroids, or theophylline (if needed)
- 2. Routine Efforts to improve postoperative FVC:
- a. Coughing from a deep inspiration has been demonstrated to improve lung volume after abdominal surgery and is still recommended in postoperative patients ¹²⁵. A protective cover (pillow) is often held over the operative site to minimize pain and

decrease tension on the wound.

- b. IPPB is an attempt to inflate the lungs passively with a positive pressure assist. It is still used by some surgeons in spite of several controlled studies demonstrating its lack of effectiveness in this patient population ^{31,126}. It also may be detrimental to patients by causing significant abdominal distention ¹²⁵. Currently, it is reserved for those patients who have significant neuromuscular disorders and who cannot cooperate with other respiratory maneuvers.
- c. Incentive Spirometry (IS) uses a device with visual feedback to the patient to encourage deep breathing. Controlled studies do not demonstrate a significant difference between the use of incentive spirometry and deep breathing exercises in the effectiveness of decreasing postoperative pulmonary complications^{125,127,128}. However, the devices continue to be used since they can be used with minimal input from hospital staff. Furthermore, when IS is started preoperatively the patient has the knowledge of the preoperative function and hence has a goal to be achieved.
- d. CPAP and PEEP Positive end expiratory pressure or continuous positive airway pressure applied intermittently via a mask to a spontaneously breathing patient has been shown to improve post operative FVC, and $(A-a)O_2$ difference and incidence of atelectasis at three days post operatively compared to IS. However, there was no difference among the groups of patients in the first 24 hours ¹²⁹. It is likely that the cost is higher, and CPAP or PEEP may be better reserved for treatment of hypoxemia postoperatively rather than used as a protective postoperative therapy administered to all patients. If this criteria were used, then 60% of the patients treated with either CPAP or PEEP in the above study would not have needed treatment.
- 3. Pain control The patient obviously experiences pain following major surgery. The use of systemic opiate analgesia may actually increase the pulmonary complication rate due to the sedative effects, decreased response to arterial PaCO₂, and deceased cough ¹³⁰. Also, this adverse effect is not avoided by use of intrathecal or epidural administration of opiod analgesics, particularly in the elderly ^{131,132}. The respiratory suppressive effects may persist for as long as 24 hours after injection, so these patients need continued monitoring ¹³². The use of a local anesthetic agent for spinal analgesia has been shown to reverse the suppression of diaphragmagic activity found after upper abdominal surgery, improve the FVC as well as give adequate pain control ³⁷. Furthermore, one study noted a decreased incidence of respiratory complications by using postoperative epidural anesthesia ¹³³. Other forms of pain control have been utilized, such as patient controlled analgesia (PCA) which has resulted in infrequent episodes of respiratory depression ¹³⁴.

The postero-lateral thoracotomy incision causes one of the greatest iatrogenic pains. The pain is exacerbated by movement, particularly that of breathing and coughing. Hence, with such pain the patient is unwilling and unable to participate in the prescribed routine that is intended to reduce postoperative pulmonary complications. Administration of opiates via the intrathecal route has been used with good pain control, but with the added complication of urinary retention, and delayed

respiratory suppression ¹³¹. As with upper abdominal surgery, the epidural route has become favored for administration of analgesia ^{135,136}. Opiates have yielded to less respiratory suppressive agents such as fentanyl ^{137,138}. Good results are also reported with the local anesthetic bupivacaine ¹³⁹⁻¹⁴¹ infused intra pleurally¹⁴², intercostally ¹⁴³, or via an extrapleural tunnel ¹⁴⁴. Good control of pain without increased complications has been reported with PCA via the epidural route ¹³⁶.

Other systemic agents can be used to improve pain control. Due to the manipulation of the pleura, periosteum of the ribs, and chest wall muscles, the non steroidal anti-inflammatory agents can provide significant relief with less opiates required ^{145,146}. Their use is often limited by gastrointestinal or renal side effects and patient intolerance. Pain relief is usually tailored to each patient and will vary depending on the patient's age, prior exposure to and tolerance of opiates. It is nevertheless recognized as a necessary adjunct in the post operative care of the patient.

4. Atelectasis: Once atelectasis of a lobe or segment occurs acutely, initial treatment with vigorous chest physical therapy has been shown effective ¹⁴⁷. Bronchoscopy was not more effective than chest physiotherapy with a bronchodilator ¹⁴⁷. As above, positive expiratory pressure via a face mask may be effective in improving postoperative atelectasis ¹²⁹.

SUMMARY:

Pulmonary complications following surgery are those undesirable postoperative effects which cause morbidity to the patient such that additional medical therapy is needed, hospitalization is increased, or death occurs.

Pulmonary complications are more likely to occur in those patients who undergo upper abdominal or thoracic surgery. The complications in upper abdominal surgery are related to reflex inhibition of diaphragm function which in turn decreases FRC. While pain is contributory, it is not the primary cause.

Patients who are most likely to develop postoperative pulmonary complications are those with poor underlying medical health, particularly those with active symptoms of pulmonary disease, and advanced age. There is no evidence that this can be accurately predicted with preoperative lung function studies.

There is no indication that necessary non-thoracic surgery should be denied to a patient on the basis of pulmonary function studies alone.

Predictions of postoperative lung function following resectional surgery of the lung are reasonably accurate, but has less predictive value of morbidity and mortality from the surgery. The best predictor of postoperative mortality following resectional surgery of the lung is measurement of the DLCO corrected for Hb and lung volume.

Postoperative pulmonary complications that cause significant morbidity following resectional surgery can be predicted with a cardiopulmonary risk index which can be determined with routine clinical studies. The addition of quantitative exercise testing to the evaluation process remains to be explicitly defined. It may have some use in identifying those patients with poor cardiovascular reserve, however.

Postoperative care is based on increasing mobilization of the patient, pain control, DVT prophylaxis, and early treatment of atelectasis and bronchospasm.

REFERENCES

- 1. Diamond GA, Forrester JS: Analysis of probability as an aid in the clinical diagnosis of coronary-artery disease. N Engl J Med 300:1358, 1979.
- 2. Sykes LA, Bowe EA: Cardiorespiratory effects of anesthesia. Clin Chest Med 14:211, 1993.
- 3. Knill RL: Control of breathing: Effects of analgesic, anaesthetic and neuromuscular blocking drugs. Can Anaesth Soc J 35:S4, 1988.
- 4. Knill RL, Kieraszewicz HT, Dodgson BG: Chemical regulation of ventilation during isoflurane sedation and anaesthesia in humans. <u>Can Anaesth Soc J</u> 30:607, 1983.
- Pietak S, Weenig CS, Hickey RF, et al: Anesthetic effects on ventilation in patients with chronic obstructive pulmonary disease. <u>Anesthesiology</u> 42:160, 1975.
- 6. Fourcade HE, Larson CP, Hickey RF, et al: Effects of time on ventilation during halothane and cyclopropane anesthesia. <u>Anesthesiology</u> 36:83, 1972.
- 7. Murat I, Saint-Maurice JP, Beydon L, et al: Respiratory effects of nitrous oxide during isoflurane anaesthesia in children. <u>Br J Anaesth</u> 58:1122, 1986.
- 8. Dueck R, Prutow RJ, Davies NJH, et al: The lung volume at which shunting occurs with inhalation anesthesia. Anesthesiology 69:854, 1988.
- 9. Hedenstierna G, Tokics L, Strandberg A, et al: Correlation of gas exchange impairment to development of atelectasis during anaesthesia and muscle paralysis. Acta Anaesthesiol Scand 30:183, 1986.
- Hedenstierna G: Gas exchange during anaesthesia. <u>Br J Anaesth</u> 64:507, 1990.
- 11. Pavlin EG and Su JY: Cardiopulmonary pharmacology. In: *Anesthesia*, edited by Miller, R.D. New York: Churchill Livingstone, 1990, p. 105-134.
- 12. Berthelsen P, Haxholdt OS, Husum B, et al: PEEP reverses nitroglycerin-induced hypoxemia following coronary artery bypass surgery. Acta Anaesthesiol Scand 30:243, 1986.
- 13. Krayer S, Rehder K, Vettermann J, et al: Position and motion of the human diaphragm during anesthesia-paralysis. Anesthesiology 70:891, 1989.

- 14. Hedenstierna G, Strandberg A, Brismar B, et al: Functional residual capacity, thoracoabdominal dimensions, and central blood volume during general anesthesia with muscle paralysis and mechanical ventilation. <u>Anesthesiology</u> 62:247, 1985.
- 15. Hirshman CA, Bergman NA: Factors influencing intrapulmonary airway calibre during anaesthesia. <u>Br J Anaesth</u> 65:30, 1990.
- 16. Pizov R, Takahashi M, Hirshman CA, et al: Halothane inhibition of ion transport of the tracheal epithelium. <u>Anesthesiology</u> 76:985, 1992.
- 17. Baron J-F, Bertrand M, Barre E, et al: Combined epidural and general anesthesia versus general anesthesia for abdominal aortic surgery. Anesthesiology 75:611 618, 1991.
- 18. Ravin MB: Comparison of spinal and general anesthesia for lower abdominal surgery in patients with chronic obstructive pulmonary disease. <u>Anesthesiology</u> 35:319, 1971.
- 19. Tarhan S, Moffitt EA, Sessler AD, et al: Risk of anesthesia and surgery in patients with chronic bronchitis and chronic obstructive pulmonary disease. Surgery 74:720, 1973.
- 20. Muravchick S, Johnson R: Oxygenation of peripheral tissues in young and elderly patients during spinal anesthesia. Reg Anesth 11:7, 1986.
- 21. Shimosato S, Etsten BE: The role of the venous system in cardiocirculatory dynamics during spinal and epidural anesthesia in man. <u>Anesthesiology</u> 30:619, 1969.
- 22. Bonica JJ, Berges PU, Morikawa K: Circulatory effects of peridural block: I. Effects of level of analgesia and dose of lidocaine. <u>Anesthesiology</u> 33:619, 1970.
- 23. Ward RJ, Bonica JJ, Freund FG, et al: Epidural and subarachnoid anesthesia. JAMA 191:275, 1965.
- 24. Goldman L, Caldera DL, Nussbaum SR, et al: Multifactorial index of cardiac risk in noncardiac surgical procedures. N Engl J Med 297:845, 1977.
- 25. Detsky AS, Abrams HB, Forbath N, et al: Cardiac assessment for patients undergoing noncardiac surgery. <u>Arch Intern Med</u> 146:2131, 1986.

- 26. Latimer RG, Dickman M, Day WC, et al: Ventilatory patterns and pulmonary complications after upper abdominal surgery determined by preoperative and postoperative computerized spirometry and blood gas analysis. <u>Am J Surg</u> 122:622, 1971.
- 27. Fagon JY, Chastre J, Hance AJ, et al: Detection of nosocomial lung infection in ventilated patients. Use of a protected specimen brush and quantitative culture techniques in 147 patients. <u>Am Rev Respir Dis</u> 138:110, 1988.
- 28. Clagett GP, Reisch JS: Prevention of venous thromboembolism in general surgical patients. Ann Surg 208:227, 1988.
- 29. Hull RD, Raskob GE, Hirsh J: Prophylaxis of venous thromboembolism. An overview. Chest 89:374S, 1986.
- 30. Wightman JAK: A prospective survey of the incidence of postoperative pulmonary complications. <u>Br J Surg</u> 55:85, 1968.
- 31. Becker A, Barak S, Braun E, et al: The treatment of postoperative pulmonary atelectasis with intermittent positive pressure breathing. <u>Surg Gynecol Obstet</u> 111:517, 1960.
- 32. Simonneau G, Vivien A, Sartene R, et al: Diaphragm dysfunction induced by upper abdominal surgery. Am Rev Respir Dis 128:899, 1983.
- 33. Easton PA, Fitting JW, Arnoux R, et al: Recovery of diaphragm function after laparotomy and chronic sonomicrometer implantation. <u>J Appl Physiol</u> 66:613, 1989.
- 34. Pasteur W: Active lobar collapse of the lung after abdominal operations. <u>Lancet</u> 2:1080, 1910.
- 35. Ford GT, Whitelaw WA, Rosenal TW, et al: Diaphragm function after upper abdominal surgery in humans. Am Rev Respir Dis 127:431, 1983.
- 36. Duggan JE, Drummond GB: Abdominal muscle activity and intraabdominal pressure after upper abdominal surgery. <u>Anesth Analg</u> 69:598, 1989.
- 37. Mankikiari B, Cantineau JP, Bertrand M, et al: Improvement of diaphragmatic function by a thoracic extradural block after upper abdominal surgery. Anesthesiology 68:379, 1988.
- 38. Aubier M, Murciano D, Lecocguic Y, et al: Effect of hypophosphatemia on diaphragmatic contractility in patients with acute respiratory failure. N Engl J Med 313:420, 1985.

- 39. Aubier M, Viires N, Piquet J, et al: Effects of hypocalcemia on diaphragmatic strength generation. <u>J Appl Physiol</u> 58:2054, 1985.
- 40. Prabhakar NR, Marek W, Loeschcke HH: Altered breathing pattern elicited by stimulation of abdominal visceral afferents. <u>J Appl Physiol</u> 58:1755, 1985.
- 41. DeTroyer A, Rosso J: Reflex inhibition of the diaphragm by esophageal afferents. Neurosc Lett 30:43, 1982.
- 42. Oliven A, Haxhiu M, Kelsen SG: Reflex effect of esophageal distension on respiratory muscle activity and pressure. <u>J Appl Physiol</u> 66:536, 1989.
- 43. Farkas GA, De Troyer A: Effects of midline laparotomy on expiratory muscle activation in anesthetized dogs. <u>J Appl Physiol</u> 66:599, 1989.
- 44. Meyers WC, Southern Surgeons Club: A prospective analysis of 1518 laparoscopic cholecystectomies. N Engl J Med 324:1073, 1991.
- 45. Frazee RC, Roberts JW, Okeson GC, et al: Open versus laparoscopic cholecystectomy. Ann Surg 213:651, 1991.
- 46. Katagiri M, Young RN, Rothwell BC, et al: Lung and diaphragm function in open (OC) versus laparoscopic cholecystectomy (LAPC) in humans. <u>FASEB J</u> 6:A2027, 1992.
- 47. Hall JC, Tarala RA, Hall JL, et al: A multivariate analysis of the risk of pulmonary complications after laparotomy. Chest 99:923, 1991.
- 48. Torrington KG, Henderson CJ: Perioperative Respiratory Therapy (PORT). A program of preoperative risk assessment and individualized postoperative care. Chest 93:946, 1988.
- 49. Tisi GM: Preoperative evaluation of pulmonary function validity, indications and benefits. Am Rev Respir Dis 119:293, 1979.
- 50. Owens WD, Felts JA, Spitznagel EL: ASA physical status classifications: A study of consistency of ratings. <u>Anesthesiology</u> 49:239, 1978.
- 51. Windsor JA, Hill GL: Risk factors for postoperative pneumonia. <u>Ann Surg</u> 208:209, 1988.
- 52. Craig DB: Postoperative recovery of pulmonary function. <u>Anesth Analg</u> 60:46, 1981.
- 53. Hirsh J: Prevention of venous thrombosis in patients undergoing major orthopaedic surgical procedures. <u>Acta Chir Scand</u> 556:30, 1990.

- 54. Ellison LT, Duke JF, III., Ellison RG: Pulmonary compliance following open-heart surgery and its relationship to ventilation and gas exchange. Circulation 35:I-217, 1967.
- 55. Wilcox P, Baile EM, Hards J, et al: Phrenic nerve function and its relationship to atelectasis after coronary artery bypass surgery. Chest 93:693, 1988.
- 56. Weiman DS, Ferdinand FD, Bolton JWR, et al: Perioperative respiratory management in cardiac surgery. <u>Clin Chest Med</u> 14:283, 1993.
- 57. Wilcox PG, Pare PD, Pardy RL: Recovery after unilateral phrenic injury associated with coronary artery revascularization. Chest 98:661, 1990.
- 58. Efthimiou J, Butler J, Woodham C, et al: Diaphragm paralysis following cardiac surgery: Role of phrenic nerve cold injury. <u>Ann Thorac Surg</u> 52:1005, 1991.
- 59. Curtis JJ, Nawarawong W, Walls JT, et al: Elevated hemidiaphragm after cardiac operations: Incidence, prognosis, and relationship to the use of topical ice slush. <u>Ann Thorac Surg</u> 48:764, 1989.
- 60. Esposito RA, Spencer FC: The effect of pericardial insulation on hypothermic phrenic nerve injury during open-heart surgery. <u>Ann Thorac Surg</u> 43:303, 1987.
- 61. Kroenke K, Lawrence VA, Theroux JF, et al: Postoperative complications after thoracic and major abdominal surgery in patients with and without obstructive lung disease. Chest 104:1445, 1993.
- 62. Fan ST, Lau WY, Yip WC, et al: Prediction of postoperative pulmonary complications in oesophagostric cancer surgery. <u>Br J Surg</u> 74:408, 1987.
- 63. Mack MJ, Aronoff RJ, Acuff TE, et al: Present role of thoracoscopy in the diagnosis and treatment of diseases of the chest. <u>Ann Thorac Surg</u> 54:403, 1992.
- 64. Miller DL, Allen MS, Trastek VF, et al: Videothoracoscopid wedge excision of the lung. <u>Ann Thorac Surg</u> 54:410, 1992.
- 65. Landreneau RJ, Hazelrigg SR, Ferson PF, et al: Thoracoscopic resection of 85 pulmonary lesions. <u>Ann Thorac Surg</u> 54:415, 1992.
- 66. Roviaro GC, Rebuffat C, Varoli F, et al: Videoendoscopic thoracic surgery. Int Surg 78:4, 1993.
- 67. Bensard DD, McIntyre RC, Jr., Waring BJ, et al: Comparison of video thoracoscopic lung biopsy to open lung biopsy in the diagnosis of interstitial lung disease. Chest 103:765, 1993.

- 68. Daniel TM, Kern JA, Tribble CG, et al: Thoracoscopic surgery for diseases of the lung and pleura. Ann Surg 217:566, 1993.
- 69. Donnelly RJ, Page RD, Cowen ME: Endoscopy assisted microthoracotomy: Initial experience. Thorax 47:490, 1992.
- 70. Cournand A, Berry FB: The effect of pneumonectomy upon cardiopulmonary function in adult patients. Ann Surg 116:532, 1942.
- 71. Gaensler EA, Cugell DW, Lindgren I, et al: The role of pulmonary insufficiency in mortality and invalidism following surgery for pulmonary tuberculosis. <u>J Thorac Cardiovasc Surg</u> 29:163, 1955.
- 72. Miller JI, Mansour KA, Hatcher CR, Jr.: Carcinoma of the lung: Five-year experience in a university hospital. Am Surg 46:147, 1980.
- 73. Mittman C: Assessment of operative risk in thoracic surgery. <u>Am Rev Respir Dis</u> 84:197, 1961.
- 74. Boysen PG, Block AJ, Moulder PV: Relationship between preoperative pulmonary function tests and complications after thoracotomy. <u>Surg Gynecol Obstet</u> 152:813, 1981.
- 75. Romano PS, Mark DH: Patient and hospital characteristics related to in-hospital mortality after lung cancer resection. Chest 101:1332, 1992.
- 76. Markos J, Mullan BP, Hillman DR, et al: Preoperative assessment as a predictor of mortality and morbidity after lung resection. Am Rev Respir Dis 139:902, 1989.
- 77. Gass GD, Olsen GN: Preoperative pulmonary function testing to predict postoperative morbidity and mortality. Chest 89:127, 1986.
- 78. Miller JI, Grossman GD, Hatcher CR: Pulmonary function test criteria for operability and pulmonary resection. <u>Surg Gynecol Obstet</u> 153:893, 1981.
- 79. Ferguson MK, Little L, Rizzo L, et al: Diffusing capacity predicts morbidity and mortality after pulmonary resection. <u>J Thorac Cardiovasc Surg</u> 96:894, 1988.
- 80. Boysen PG, Block AJ, Olsen GN, et al: Prospective evaluation for pneumonectomy using the 99mtechnetium quantitative perfusion lung scan. Chest 72:422, 1977.

- 81. Ali MK, Mountain CF, Ewer MS, et al: Predicting loss of pulmonary function after pulmonary resection for bronchogenic carcinoma. Chest 77:337, 1980.
- 82. Khalil M, Ewer MS: Preoperative cardiopulmonary evalutaion of patients undergoing surgery for lung cancer. <u>Cancer Bull</u> 32:100, 1980.
- 83. Ishida T, Yano T, Maeda K, et al: Strategy for lymphadenectomy in lung cancer three centimeters or less in diameter. <u>Ann Thorac Surg</u> 50:708, 1990.
- 84. Mountain CF: Lung cancer staging classification. Clin Chest Med 14:43, 1993.
- 85. Diener CF, Burrows B: Further observations on the course and prognosis of chronic obstructive lung disease. Am Rev Respir Dis 111:719, 1975.
- 86. Traver GA, Cline MG, Burrows B: Predictors of mortality in chronic obstructive pulmonary disease. Am Rev Respir Dis 119:895, 1979.
- 87. Eisert DR, Cox JD, Komaki R: Irradiation for bronchial carcinoma: Reasons for failure. <u>Cancer</u> 37:2665, 1976.
- 88. Neuhaus H, Cherniack NS: A bronchospirometric method of estimating the effect of pneumonectomy on the maximum breathing capacity. <u>J Thorac Cardiovasc Surg</u> 55:144, 1968.
- 89. Walkup RH, Vossel LF, Griffin JP, et al: Prediction of postoperative pulmonary function with the lateral position test. <u>Chest</u> 77:24, 1980.
- 90. Schoonover GA, Olsen GN, McLain WC, III., et al: Lateral position test and quantitative lung scan in the preoperative evaluation for lung resection. Chest 86:854, 1984.
- 91. Kristersson S, Lindell S-E, Svanberg L: Prediction of pulmonary function loss due to pneumonectomy using 133Xe-radiospirometry. Chest 62:694, 1972.
- 92. Wernly JA, DeMeester TR, Kirchner PT, et al: Clinical value of quantitative ventilation-perfusion lung scans in the surgical management of bronchogenic carcinoma. <u>J Thorac Cardiovasc Surg</u> 80:535, 1980.
- 93. Olsen GN, Block AJ, Tobias JA: Prediction of postpneumonectomy pulmonary function using quantitative macroaggregate lung scanning. <u>Chest</u> 66:13, 1974.
- 94. Boysen PG, Harris JO, Block AJ, et al: Prospective evaluation for pneumonectomy using perfusion scanning. <u>Chest</u> 80:163, 1981.

- 95. Corris PA, Ellis DA, Hawkins T, et al: Use of radionuclide scanning in the preoperative estimation of pulmonary function after pneumonectomy. <u>Thorax</u> 42:285, 1987.
- 96. Olsen GN, Weiman DS, Bolton JWR, et al: Submaximal invasive exercise testing and quantitative lung scanning in the evaluation for tolerance of lung resection. Chest 95:267, 1989.
- 97. Cain HD, Stevens PM, Adaniya R: Preoperative pulmonary function and complications after cardiovascular surgery. Chest 76:130, 1979.
- 98. Boushy SF, Billig DM, North LB, et al: Clinical course related to preoperative and postoperative pulmonary function in patients with bronchogenic carcinoma. Chest 59:383, 1971.
- 99. Lockwood P: Lung function test results and the risk of post-thoracotomy complications. Respiration 30:529, 1973.
- 100. Keagy BA, Schorlemmer GR, Murray GF, et al: Correlation of preoperative pulmonary function testing with clinical course in patients after pneumonectomy. <u>Ann Thorac Surg</u> 36:253, 1983.
- Van Nostrand D, Kjelsberg MO, Humphrey EW: Preresectional evaluation of risk from pneumonectomy. <u>Surg Gynecol Obstet</u> 127:306312, 1968.
- 102. Suzuki C, Arakaki Y, Nagashima Y, et al: Resection tolerance of the patients with lung cancer associated with chronic obstructive pulmonary disease. <u>Sci Rep Res Inst Tokohu Univ</u> 17:60, 1970.
- 103. Carlens E, Hanson HE, Nordenstrom B: Temporary unilateral occlusion of the pulmonary artery. <u>J Thorac Surg</u> 22:527, 1951.
- 104. Harrison RW, Adams WE, Long ET, et al: The clinical significance of cor pulmonale in the reduction of cardiopulmonary reserve following extensive pulmonary resection. <u>J Thorac Surg</u> 36:352, 1958.
- 105. Laros CD, Swierenga J: Temporary unilateral pulmonary artery occlusion in the preoperative evaluation of patients with bronchial carcinoma. <u>Med Thor</u> 24:269, 1967.
- 106. Fee HJ, Holmes EC, Gewirtz HS, et al: Role of pulmonary vascular resistance measurements in preoperative evaluation of candidates for pulmonary resection. <u>J Thorac Cardiovasc Surg</u> 75:519, 1978.
- 107. Reichel J: Assessment of operative risk of pneumonectomy. Chest 62:570, 1972.

- 108. Nakagawa K, Nakahrar K, Miyoshi S, et al: Oxygen transport during incremental exercise load as a predictor of operative risk in lung cancer patients. Chest 101:1369, 1992.
- 109. Miyoshi S, Nakahara K, Ohno K, et al: Exercise tolerance test in lung cancer patients: The relationship between exercise capacity and postthoracotomy hospital mortality. <u>Ann Thorac Surg</u> 44:487, 1987.
- 110. Murray, J.F. *The Normal Lung*, Philadelphia:W.B. Saunders Company, 1986. Ed. 2.
- 111. Colman NC, Schraufnagel DE, Rivington RN, et al: Exercise testing in evaluation of patients for lung resection. Am Rev Respir Dis 125:604, 1982.
- 112. Bagg LR: The 12-min walking distance; its use in the pre-operative assessment of patients with bronchial carcinoma before lung resection. Respiration 46:342, 1984.
- 113. Bechard D, Wetstein L: Assessment of exercise oxygen consumption as preoperative criterion for lung resection. <u>Ann Thorac Surg</u> 44:344, 1987.
- 114. Smith TP, Kinasewitz GT, Tucker WY, et al: Exercise capacity as a predictor of post-thoracotomy morbidity. Am Rev Respir Dis 129:730, 1984.
- 115. Olsen GN, Bolton JWR, Weiman DS, et al: Stair climbing as an exercise test to predict the postoperative complications of lung resection. <u>Chest</u> 99:587, 1991.
- 116. Morice RC, Peters EJ, Ryan MB, et al: Exercise testing in the evaluation of patients at high risk for complications from lung resection. Chest 101:356, 1992.
- 117. Bolton JWR, Weiman DS, Haynes JL, et al: Stair climbing as an indicator of pulmonary function. Chest 92:783, 1987.
- 118. DeGraff AC, Jr., Taylor HF, Ord JW, et al: Exercise limitation following extensive pulmonary resection. <u>J Clin Invest</u> 44:1514, 1965.
- 119. Wasserman K, Whipp BJ: Exercise physiology in health and disease. Am Rev Respir Dis 112:219, 1975.
- 120. Epstein SK, Falig LJ, Daly BDT, et al: Predicting complications after pulmonary resection. Preoperative exercise testing vs a multifactorial cardiopulmonary risk index. Chest 104:694, 1993.

- 121. Gerson MC, Hurst JM, Hertzberg VS, et al: Prediction of cardiac and pulmonary complications related to elective abdominal and noncardiac thoracic surgery in geriatric patients. <u>Am J Med</u> 88:101, 1990.
- 122. Warner MA, Divertie MB, Tinker JH: Preoperative cessation of smoking and pulmonary complications in coronary artery bypass patients. <u>Anesthesiology</u> 60:380, 1984.
- 123. Burns DM: Cigarettes and cigarette smoking. Clin Chest Med 12:631, 1991.
- 124. Gracey DR, Divertie MB, Didier EP: Preoperative pulmonary preparation of patients with chronic obstructive pulmonary disease. <u>Chest</u> 76:123, 1979.
- 125. Celli BR, Rodriguez KS, Snider GL: A controlled trial of intermittent positive pressure breathing, incentive spirometry, and deep breathing exercises in preventing pulmonary complications after abdominal surgery. <u>Am Rev Respir Dis</u> 130:12, 1984.
- 126. Baxter WD, Levine RS: An evaluation of intermittent positive pressure breathing in the prevention of postoperative pulmonary complications. <u>Arch Surg</u> 98:795, 1969.
- 127. Stock CM, Downs JB, Gauer PK, et al: Prevention of postoperative pulmonary complications with CPAP, incentive spirometry, and conservative therapy. Chest 87:151, 1985.
- 128. Hall JC, Tarala R, Harris J, et al: Incentive spirometry versus routine chest physiotherapy for prevention of pulmonary complications after abdominal surgery. Lancet 337:953, 1991.
- Ricksten S-E, Bengtsson A, Soderberg C, et al: Effects of periodic positive airway pressure by mask on postoperative pulmonary function. <u>Chest</u> 89:774, 1986.
- 130. Egbert LD, Bendixen HH: Effect of morphine on breathing pattern. <u>JAMA</u> 188:485, 1964.
- 131. Cousins MJ, Mather LE: Intrathecal and epidural administration of opiods. Anesthesiology 61:276, 1984.
- 132. Coleman DL: Control of postoperative pain. Nonnarcotic and narcotic alternatives and their effect on pulmonary function. Chest 92:520, 1987.
- 133. Yeager MP, Glass DD, Neff RK, et al: Epidural anesthesia and analgesia in high-risk surgical patients. <u>Anesthesiology</u> 66:729, 1987.

- 134. McKenzie R: Patient-controlled Analgesia (PCA). <u>Anesthesiology</u> 69:1027, 1988.
- 135. Baxter AD, Samson B, Penning J, et al: Prevention of epidural morphine-induced respiratory depression with intravenous nalbuphine infusion in post-thoracotomy patients. Can J Anaesth 36:503, 1989.
- 136. Grant RP, Dolman JF, Harper JA, et al: Patient-controlled lumbar epidural fentanyl compared with patient-controlled intravenous fentanyl for post-thoracotomy pain. Can J Anaesth 39:214, 1992.
- 137. Melendez JA, Cirella VN, Delphin ES: Lumbar epidural fentanyl analgesia after thoracic surgery. <u>J Cardiothorac Anesth</u> 1993.
- 138. Badner NH, Sandler AN, Koren G, et al: Lumbar epidural fentanyl infusions for post-thoracotomy patients: Analgesic, respiratory, and pharmacokinetic effects. <u>J Cardiothorac Anesth</u> 4:543, 1990.
- 139. Mann LJ, Young GR, Wiliams JK, et al: Intrapleural bupivacaine in the control of postthoracotomy pain. <u>Ann Thorac Surg</u> 53:449, 1992.
- 140. Eng J, Sabanathan S: Site of action of continuous extrapleural intercostal nerve block. <u>Ann Thorac Surg</u> 51:387, 1991.
- 141. Delilkan AE, Lee CK, Yong NK, et al: Post-operative local analgesia for thoracotomy with direct bupivacaine intercostal blocks. <u>Anaesthesia</u> 28:561, 1973.
- 142. Ferrante FM, Chan VWS, Arthur R, et al: Interpleural analgesia after thoracotomy. Anesth Analg 72:105, 1991.
- 143. Berrisford RG, Sabanathan SS, Mearns AJ, et al: Pulmonary complications after lung resection: The effect of continuous extrapleural intercostal nerve block. <u>Eur J Cardio-thorac Surg</u> 4:407, 1990.
- 144. Majid AA, Hamzah H: Pain control after thoracotomy: An extrapleural tunnel to provide a continuous bupivacaine infusion for intercostal nerve blockade. Chest 101:981, 1992.
- 145. Kalso E, Perttunen K, Kaasinen S: Pain after thoracic surgery. <u>Acta Anaesthesiol Scand</u> 36:96, 1992.
- 146. Conacher ID: Pain relief after thoracotomy. Br J Anaesth 65:806, 1990.
- 147. Kirilloff LH, Owens GR, Rogers RM, et al: Does chest physical therapy work? Chest 88:436, 1985.